

**Short term Impact on lung function from Traffic Related Air Pollution  
(TRAP) from undertaking bicycling in different TRAP exposure routes**

by

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### List of Acronyms

AURN	Automatic urban and rural network
BC	Black carbon
CO	Carbon monoxide
CP	Coarse particle
CPC	Condensed particle counter
DF	Deposition factor
EBC	Exhaled breath condensate
FP	Fine particle
FENO	Fractional exhaled Nitric Oxide
FEV <sub>1</sub>	Forced expiratory volume in one second
FEV <sub>6</sub>	Forced expiratory volume in six second
FEF	Forced expiratory flow
NO	Nitric oxide
NO <sub>2</sub>	Nitrogen dioxide
NO <sub>x</sub>	Oxides of nitrogen
O <sub>3</sub>	Ozone
PA	Physical Activity
PAH	Polycyclic aromatic hydrocarbon
PM	Particulate Matter
SO <sub>2</sub>	Sulphur dioxide
PNC	Particle number concentration
TRAP	Traffic related air pollution
UFP	Ultrafine particle
VE	Ventilation rate

Word count: 14938

## Executive Summary

### Introduction

Air quality in cities today is not only affecting the health of the residents but also hampering efforts undertaken to decarbonize transport as a result of global issues like climate change and promoting use of active travel like bicycling more attractive (Jones, 2014). Air pollution is the 5<sup>th</sup> leading mortality risk factor worldwide, In 2016, total reduced average life expectancy by 1 year 8 months worldwide (HEI, 2019). Particulate matter (PM) and Ozone are two key pollutants and a significant proportion is contributed by transport sector (HEI, 2019). While attempts to decarbonize transport by use of active travel modes like bicycling are critical it is of vital importance that we also understand health impacts from undertaking bicycling in currently polluted environment of cities for better policy inputs on active travel.

There are variety of epidemiological studies undertaken to understand health impacts on lung function from exposure to air pollution. For example, understanding impact on lung function by comparing demographics based on exposure hours and air pollution events with hospital admissions (Spix et al., 1998), difference in dosage of air pollutants based on choice of mode (Namdeo Anil et al., 2016), chamber studies and real world studies with controlled exposure and corresponding volumetric and inflammatory changes in lung function (Daigle et al., 2003). Majority of the epidemiological evidence is for long term studies which in itself is a result of cumulative impact from daily short-term impacts (WHO, 2006). Also, evidence on health impact is primarily premised upon exposure-response function which is not constant across the spatial scale, but concentration profile of pollutants in urban realm has instances of high exceedances at street levels due to lack of dispersion in comparison to areas outside the city boundary or less built up environment (Tate, 2018). Thus, air quality is measured in terms of background and kerbside conditions and using the same lens to understand health impacts is a crucial piece for evidence-based policy inputs.

### Scope

The scope of the research is limited to assess the volumetric impact using spirometry parameters of FEV<sub>1</sub>(fractional exhaled breath volume in one second), FEV<sub>6</sub> (fractional exhaled breath volume in six seconds) and FEF (Forced expiratory flow) (NHLBI, 2019) and inflammatory changes in airways with fractional exhaled nitric oxide (FeNO) in breath as a biomarker for inflammation (Annesi-Maesano and Dinh-Xuan, 2016) from exposure to ultra

fine particles (UFP) (with primary source from transport emissions). Impact in lung function was studied for four male participants undertaking bicycling on a dedicated polluted and controlled route for duration of 7 days.

### Methodology and Analysis

The experiment designed to understand if there was a statistically significant short term volumetric and inflammatory impact in lung function from undertaking bicycling on polluted and controlled route during AM peak. In order to avoid contamination prior to experiment a taxi was used to transfer all the participants to controlled route. The polluted route round trip had origin and destination as controlled route. Four participants bicycled for seven days on controlled and polluted route based on routes allocated for day. The start time and end time for all the participants irrespective of the route were same thus duration of exposure remained constant for a given day. The chosen polluted route with PM concentration levels representative of kerbside conditions while controlled route was representative of urban background conditions as per DEFRA, (2019d) guidelines.

Continuous measurements of particle concentration were measured with handheld condensed particulate counter (CPC) while ventilation rate were measured with sweetzpot device tied to chest via harness for all participants. GPS enabled mobile phones with STRAVA application recorded details of journey time and route. Before and after cycling FeNO and spirometry readings for all participants were recorded.

For analysis a common indicator of inhaled dosage adopted from Namdeo Anil et al., (2016) which is function particle exposure was developed which was used to understand the impact on lung function. Lung inflammation were interpreted based on FeNO guidelines from Dweik et al., (2011) inline to ATS/ERS. While spirometry parameters the average of three repeated measurements were analysed for degradation post bicycling. Statistical t-test was used to identify if the mean particle concentration on polluted route was higher in comparison to controlled route. Similarly, t-test was used to check if the change in FeNO and spirometry parameters was statistically significant for cohort.

## Results

The mean concentration on polluted route was always higher in comparison to controlled route and was statistically significant at 98% confidence interval for the duration of study. FeNO results post cycling for participants grouped as cohort by route were not statistically significant at 95% confidence interval. Similar was the case for spirometry parameters. Table-A shows the change in FeNO and spirometry parameters for each run. There is no evidence of simplistic particle dosage from exposure and corresponding spirometry or inflammatory impact. No evidence of simultaneous impact between spirometry and lung inflammation.

*Table-A Spirometry and FeNO results for participants by route*

Day	Participant	Route	Journey time (mins)	Inhaled dose (count)	Std dev Inhaled Dose (count)	FEV1 (L/s)		FEV6 (L/s)		FEV1/FEV6		FEF (L)		FeNO (ppb)	
						Before	After	Before	After	Before	After	Before	After	Before	After
day-1	A	controlled	53	0.0221	0.0086	3.727	3.953	5.42	5.557	0.6867	0.71	2.553	2.81	3	6
	B	controlled		0.0431	0.0220	3.14	3.127	3.943	3.947	0.7967	0.7933	2.633	2.63	31	36
	X	polluted		0.0155	0.0099	3.06	3.885	3.495	4.27	0.87	0.88	3.515	4.695	13	13
	Y	polluted		0.0286	0.0095	3.11	3.303	4.007	4.16	0.78	0.7933	2.383	2.677	14	16
day-2	A	polluted	58	0.0951	0.0811	3.733	3.873	5.203	5.207	0.7167	0.7467	2.597	2.827	6	4
	B	polluted		0.0951	0.0811	3.177	3.287	3.947	4.137	0.8067	0.7967	2.693	2.8	32	24
	X	controlled		0.0114	0.0035	3.79	3.827	4.43	4.363	0.8567	0.88	4.117	4.507	16	17
	Y	controlled		0.0444	0.0185	3.21	3.23	4.16	4.12	0.77	0.7867	2.403	2.5	26	18
day-3	A	controlled	56	0.0193	0.0075	3.853	3.76	5.45	5.1	0.7067	0.7367	2.73	2.74	6	2
	B	polluted		0.0698	0.0203	3.157	3.05	3.977	3.94	0.7933	0.7733	2.617	2.467	30	20
	X	controlled		0.0039	0.0013	3.76	3.783	4.353	4.323	0.8633	0.8767	4.21	4.48	11	18
	Y	polluted		0.0257	0.0086	3.19	3.157	4.127	3.997	0.7733	0.79	2.467	2.517	23	19
day-4	A	polluted	73	0.1209	0.0721	3.903	3.82	5.35	5.237	0.73	0.73	2.833	2.757	19	12
	B	controlled		0.0781	0.0399	2.673	3.017	3.623	3.803	0.7367	0.7967	1.997	2.52	25	25
	X	polluted		0.0281	0.0179	3.833	3.777	4.297	4.15	0.8933	0.9133	4.747	4.91	20	40
	Y	controlled		0.0448	0.0187	3.23	3.503	4.157	4.16	0.7767	0.7767	2.467	2.477	24	29
day-5	A	controlled	58	0.0233	0.0091	3.773	3.647	5.247	5.06	0.72	0.72	2.647	2.577	10	14
	B	controlled		0.0483	0.0247	2.867	2.873	3.85	3.713	0.7433	0.7767	2.17	2.3	25	30
	X	polluted		0.0310	0.0197	3.743	3.77	4.183	4.213	0.8967	0.8967	4.577	4.647	19	20
	Y	polluted		0.0941	0.0337	3.233	3.423	4.137	4.327	0.78	0.7933	2.463	2.793	24	26
day-6	A	controlled	53	0.0118	0.0046	3.533	3.687	4.977	4.937	0.7067	0.7467	2.483	2.757	9	7
	B	polluted		0.1249	0.0462	2.527	2.773	3.7	3.883	0.6933	0.7133	1.75	1.94	31	26
	X	controlled		0.0069	0.0021	3.803	3.763	4.313	4.247	0.8833	0.8867	4.52	4.483	10	13
	Y	polluted		0.0502	0.0180	3.293	2.923	3.987	4.15	0.8267	0.7833	2.957	2.513	18	21
day-7	A	polluted	72	0.1294	0.0772	3.687	3.693	5.013	4.827	0.7367	0.7633	2.653	2.797	18	13
	B	controlled		0.0192	0.0098	2.78	3.123	4.033	4.137	0.69	0.7533	1.877	2.447	29	1
	X	polluted		0.0296	0.0189	3.683	3.713	4.163	4.143	0.8867	0.8967	4.337	4.63	21	20
	Y	controlled		0.0127	0.0053	3.347	3.26	4.2	3.953	0.7967	0.8233	2.747	2.95	30	1


 Spirometry reduced  
 Spirometry increased  
 FeNO reduced or no change  
 FeNO increased

## Conclusion

This research showed that there was no statistically significant impact on volumetric and airway inflammatory lung function from undertaking bicycling on route with different UFP concentration. Key findings was cumulative exposure and air pollutant dosage from the trip are not directly proportional to volumetric and inflammatory response but toxicity and chemical composition of UFP fraction is of primary importance in terms of health impact as also hinted by Lagorio et al., (2006).

# 1 Introduction

## 1.1 Research Background and Rationale

History of air pollution goes hand in hand with that of fossil fuel (Brimblecombe, 1976). Humans have been historically affected by air pollution, evidence of blackened lungs from wood fires in mummified tissue from Egypt, while in A.D. 61 philosopher and statesman Seneca wrote about the change in his health condition after leaving oppressive atmosphere of Rome (Morrison, 2016). Air pollution by WHO, (2019a) is defined as “contamination of the indoor or outdoor environment by any chemical, physical or biological agent that modifies the natural characteristics of the atmosphere”.

Advent of automobiles powered by internal combustion engines resulted in to major shifts in mobility pattern of people, changing the landscape of transport systems in and around cities (Jones, 2014). Sudden burgeoning use of automobiles resulted into increased non-point emissions sources within city (Jones, 2014). For example, Los Angeles by 1940 had more than million cars, subsequently city was smogged in July'43, in 1947, the county established the first air pollution control district in the USA (Morrison, 2016). Historic as well as modern day efforts have been made to reduce adverse effects of air pollution, for example, in 535 AD emperor Justinian initiated a Clean Air Act proclaiming clean air as a birthright, while in 1842 societies such as Citizens smoke control association in Leeds emerged which campaigned against rising smoke in cities (Morrison, 2016). By late 1980's such campaigns were extended to U.S. cities like Chicago and smog incidents across the United States and Great Britain resulted in Clean Air Act in 1970 and U.K. Clean Air Act in 1956 respectively (Morrison, 2016).

Currently as a result of new global challenges like climate change the transport landscape is again undergoing a shift, with efforts to encourage use of active travel modes like bicycling to decarbonize transport sector (Giles-Corti et al., 2010). However, undertaking active travel in polluted environment of cities can adversely affect health (HEI, 2019). In 2017, air pollution was the 5<sup>th</sup> leading mortality risk factor worldwide surpassing deaths from road traffic injuries (HEI, 2019). While there is no safe limit of air pollution, a recent global assessment report showed in 2017, 4.9 million deaths and 147 million years of healthy life lost worldwide were attributed to air pollution (WHO, 2019b; HEI, 2019). In 2016, air pollution in total reduced average life expectancy by 1 year 8 months worldwide (HEI, 2019). The cost of air pollution on

global economy in 2016 was estimated to be at US\$ 225 billion (Worldbank, 2016). Thus, intersection of promoting active travel like bicycling as a commute choice in cities with poor air quality and resulting health impacts from exposure to traffic related air pollution (TRAP) is of key interest requiring robust understanding for active travel policy inputs.

Exposure by Watson et al., (1988) is defined as “any contact between an airborne contaminant and a surface of the human body, either outer (for example, the skin) or inner (for example, respiratory tract epithelium); thus, exposure requires the simultaneous occurrence of two events: a pollutant concentration at a particular place and time, and the presence of a person at that place and time”. Dose by Watson et al., (1988) is defined as “the amount of the pollutant that actually crosses one of the body's boundaries and reaches the target tissue”. Air pollution can be divided in to two key categories, indoor air pollution concerned with the exposure in indoor environment of homes and offices and outdoor air pollution is concerned with the exposure outside of built environment like in streets (NIEHS, 2019). Two key indicator air pollutants for outdoor air pollution are particulate matter (PM) which are airborne PM measuring less than 2.5 micrometers referred as PM<sub>2.5</sub> and ground level tropospheric ozone (O<sub>3</sub>) (HEI, 2019). Short term and long term exposure to PM<sub>2.5</sub> can cause inflammation and worsening of heart and lung diseases, while O<sub>3</sub> irritates airways of lungs increasing symptoms of those suffering from lung diseases (DEFRA, 2019a).

## 1.2 Research Problem

Myriad methods are used for mapping TRAP which are crucial for assessing health impacts (Briggs et al., 1997). Similarly, epidemiological studies have evidenced short term and long term health impacts from exposure to TRAP (Guttikunda and Goel, 2013; Paulin and Hansel, 2016). For example, time series analysis of city level air quality data from monitoring network with hospital admissions (Bell et al., 2009) and emergency visits (Stieb et al., 2009), overall health and economic impact from modal shift to bicycles from cars (Lindsay et al., 2011; Rojas-Rueda et al., 2011), Land Use Regression (LUR) modelling was applied by Hankey and Marshall, (2015) to estimate individual level of exposure over geographical area of interest, Dispersion modelling was applied by McConnell et al., (2010) to understand respiratory health outcomes in children from exposure to TRAP and Comparative risk assessment where measurements from air quality monitoring stations along with chemical transport models are combined with

epidemiological evidence to analyse global burden of disease from PM<sub>2.5</sub> and O<sub>3</sub> (WHO, 2016a; Brauer et al., 2016). However, these methods dependent on monitoring networks and modelling, can underestimate or overestimate actual exposure (Briggs et al., 1997; Johansson et al., 2017).

Very few studies have been undertaken where direct exposure from TRAP was monitored and corresponding health impact was undertaken, for example, Spira-Cohen et al., (2011) did 24 hours continuous exposure studies for PM<sub>2.5</sub> and effects on respiratory health and Brauer et al., (2001) did 24 hour continuous exposure studies for PM<sub>2.5</sub> and PM<sub>10</sub> for COPD patients. In addition, research in transport is primarily limited to TRAP dosage among the modes or comparing TRAP dosage in different locations (Int Panis et al., 2010; de Nazelle et al., 2012; Namdeo Anil et al., 2016). This study will further help understand effects of exposure to ultrafine particles (UFP), which forms PM, a marker of TRAP (Hagler et al., 2009) with typically lower exposure at background site in comparison to kerbside site at a spatial and temporal scale and overcoming limitation of real time dose measurement and TRAP concentration while undertaking active mode of travel (bicycling) in different spatial locations.

### 1.3 Research Scope

Research presented is to study short term impact on lung function from exposure to UFP in locations representative of background and kerbside conditions whilst undergoing bicycling. Urban background site (controlled route) for bicycling for this study is defined as location where pollution levels are not significantly influenced from a single source or street and is representative of contribution from all sources (traffic, industry and natural) (DEFRA, 2019d). kerbside site (polluted route) is defined as site where pollution level is predominantly from traffic (road, motorways and highways) no more than 10 metre away from the centre of road (DEFRA, 2019d).

Participants for the study are all males to eliminate the variation from difference in lung capacity between males and females (Bellemare et al., 2003). It should be noted for participants choice of male/female the key criteria were availability over the study period. Other pollutants such as oxides of nitrogen (NO<sub>x</sub>), Carbon Monoxide (CO), O<sub>3</sub> and Sulphur dioxide (SO<sub>2</sub>) which also affect lung function are not measured as instruments were not

available during study period (DEFRA, 2019a). However, data for this pollutants from stationary Automatic Urban and Rural Network (AURN) with identification code of UKA00527 (Leeds Headingley Kerbside) is accessible and used as required (DEFRA, 2019c).

#### 1.4 Research Objective

This research is undertaken to understand short term lung function impact from UFP exposure and resultant inhaled dose by cyclist in controlled route with urban background conditions and polluted route with kerbside conditions similar to the method adopted for analyzing air quality (DEFRA, 2019d). Following are the three research questions.

1. Is there statistically significant impact on volumetric lung function from undertaking bicycling in controlled route versus polluted route?
2. Is there statistically significant difference in airway inflammation of lungs by undertaking bicycling on controlled route versus polluted route?
3. Is there a combinatory relationship between volumetric and inflammatory lung function impact from undertaking bicycling on controlled route versus polluted route?

#### 1.5 Dissertation Structure

The remaining dissertation is divided in to four sections. Section-2 presents and discusses literature of existing evidence and understanding on air pollution and health impacts relevant to the research questions in section-1.4, discussing sources of emission of particle pollution primarily from transport, how meteorological parameters can influence the particle concentration in the urban realm followed by the various interaction of these particles when inhaled in lungs and current methods used and evidence to understand of impact on lung volumetric and inflammatory function. Section-3 presents the experiment design for data collection for impact on lung volumetric and inflammatory function for the research questions in section-1.4, followed by details of instruments used to collect the data and method for analysis. Section-4 discusses results in terms of cohorts by grouping participants by route and individually followed by discussion where results are interpreted in terms of wider literature as discussed in section-2 including limitation and contribution. Lastly, section-5 presents conclusion for the research question set in section-1.4 and recommendations for future research.

## 2 Literature Review

### 2.1 Chapter overview

This chapter discusses some of the existing literature pertaining to research question set in section-1.4. Section-2.2 discusses sources of particle pollution and makeup of these particles. Section-2.3 discusses dispersion of these particles in urban realm post emission. Section-2.4 discusses interaction of these particles once inhaled in lungs and key considerations for understanding particle pollution dosage. Section-2.5 discusses methods used in epidemiology to study impact on lung volumetric and inflammatory function from exposure to particle pollution.

### 2.2 Sources of particle pollution

PM consist of air pollutants comprising of suspended particles in air which is a complex mixture of organic and inorganic species with varying size as a result of emissions from natural and anthropogenic sources (El Morabet, 2018). Size of these particles can vary from few nanometers to around 100 micrometers in aerodynamic diameter (AQEG, 2005). Aerodynamic diameter by Estokova and Stevulova, (2012) is defined as “diameter of a sphere with unit density and mass equal to the mass of the provided particle” and classification based on which is shown in Table 1. The toxicity of the PM is dependent on factors like bulk chemical composition, trace element content, acid content, sulfate content and particle size distribution (Harrison and Yin, 2000). Particle with size distribution of 3-50 nm range are primarily as a result of emissions from vehicles powered by internal combustion engines (Harrison and Yin, 2000). Kleeman et al., (2000) have shown that particle composition from gasoline powered engines primarily consist of organic compounds while for diesel engines it is equal amounts of organic compounds and elemental carbon. Composition of particles in a UK study showed primary components consist of sodium chloride from sea salt, elemental carbon from fossil fuel combustion, trace metals from metallurgical processes and wear and tear of automobile components and minerals from mining and construction processes along with secondary components of nitrates, sulphates and water (AQEG, 2005). Study by Chow et al., (1993) in California found fine particle composition dominated by nitrate, sulfate and ammonia ions along with elemental and organic carbon contributing 70-80% of FP mass, in

terms of CP fraction it was dominated by aluminum, silicon, sulfur, potassium, calcium and iron contributing to 40-50% of mass.

*Table 1 Particle size type classification adapted from (Estokova and Stevulova, 2012)*

Particle size type	Particle size denomination	Particle Size
Coarse particle (CP)	PM <sub>2.5-10</sub>	Aerodynamic diameter greater than 2.5 micrometers and less than 10 micrometers
Fine particle (FP)	PM <sub>0.1-2.5</sub>	Aerodynamic diameter greater than 0.1 micrometers and less than 2.5 micrometers
Ultra Fine particle (UFP)	<PM <sub>0.1</sub>	Aerodynamic diameter which is less than 0.1 micrometers

PM also has tendency to remain suspended in the atmosphere and can remain or travel to distances over thousands of kilometers from their point of origin depending on their lifetime (Estokova and Stevulova, 2012). Anthropogenic sources of particle origin (point and non-point), one key contributor in urban environment with significant share of non-point emission sources is from road transport used for various purposes (AQEG, 2005). Vehicles produce PM as a result of combustion process in engines where fuel is burned, brake and tyre wear and tear (Garg et al., 2000).

Different vehicle types use different fuels and as a result different combustion techniques produce and release different variety of particles in different proportions in exhaust gases and based on the operating mix in urban area people are exposed to these cocktail of pollutants with different concentrations (Reşitoğlu et al., 2015). Combustion process results in agglomeration of particles from unburned fuel, unburned lubricating oil, ash content in fuel, sulfates and water which can be classified into three categories soot, soluble organic fraction and inorganic fraction (Matti Maricq, 2007). Also, composition of various particles in exhaust varies with operating engine load conditions which would result in different proportion of different types of PM based on condition of traffic (Sharma et al., 2005). Of the various pollutants emitted by automobile exhaust contribution from air borne PM has been suggested as one key contributing factor for exacerbating damage to respiratory system (Marino et al., 2015).

Diesel vehicles can emit up to 100 times more PM in comparison to catalyst equipped petrol vehicles of similar performance (Marino et al., 2015). Historically emission from vehicles have been very high in real world drive cycle and even with more stringent emission standards

adopted across EU many cities in EU are still not achieving WHO air quality standards (EEA, 2018). Peitzmeier et al., (2017) have shown in their comparison of 70000 exhaust plumes in Munster, Germany with Hand Book Emissions Factor for Road Transport (HBEFA) found median Particle Number (PN) per kilometer exceeded Euro-5 and Euro-6 by a factor of 150 for diesel and 15 for petrol, this discrepancy of higher real world emission could be one of the key reasons for EU cities not being able to meet WHO air quality standards.

Brake wear also contributes to PM emissions in urban areas, road simulation and receptor modelling studies for PM<sub>10</sub> brake wear emission factor of 2.0-8.8 mg/km-vehicle and tyre wear emission factor 3.5-9.0 mg/km-vehicle which in total closely correspond to emission factor of modern diesel EURO-6 low displacement vehicle (Theodoros and Giorgio, 2014). Brake wear is a result of mechanical induced wear between brake pad/brake shoe with friction lining material that generates particles which are then released into atmosphere (Boulter, 2006). Depending on the type of brake lining material (Non-asbestos organic, Low-metallic, Semi-metallic and Metallic), severity of braking, driving behavior and ambient conditions (Boulter, 2006). In addition, there is also contribution from wear and tear of clutch plate but since clutch mechanism is enclosed primarily particles remain enclosed (Theodoros and Giorgio, 2014).

Additionally, tyre wear as tread mass loss due to friction with road surface also contributes PM emissions (Grigoratos et al., 2018). Resuspension from paved road dust also contributes to PM<sub>10</sub> and PM<sub>2.5</sub> concentration due to turbulence induced by moving vehicles, measured at 5 locations in UK by Harrison et al., (2001) correlating particle concentration with NO<sub>x</sub> as an indicator of traffic. Non exhaust emission of PM, organic and chemical compounds are increasing as particle emissions from exhaust are reducing with imposition of new standards and their contribution might reduce in comparison to non-exhaust emissions warranting imposition of standards on non-exhaust emissions for reducing air pollution further in urban areas (DEFRA, 2018b).

### 2.3 Particle dispersion in urban realm and influence from meteorology

While understanding exposure to air pollutants and its impacts, it is also necessary to understand the environment in which a person is exposed and myriad factors that influence these interactions between environment and TRAP. Figure 1 shows there is a regional

background concentration after which there is a city level increment due to additional emissions from different sources in city after which there is a street side increment which is typically from TRAP and its interaction with urban topography and meteorological conditions (Tate, 2018).

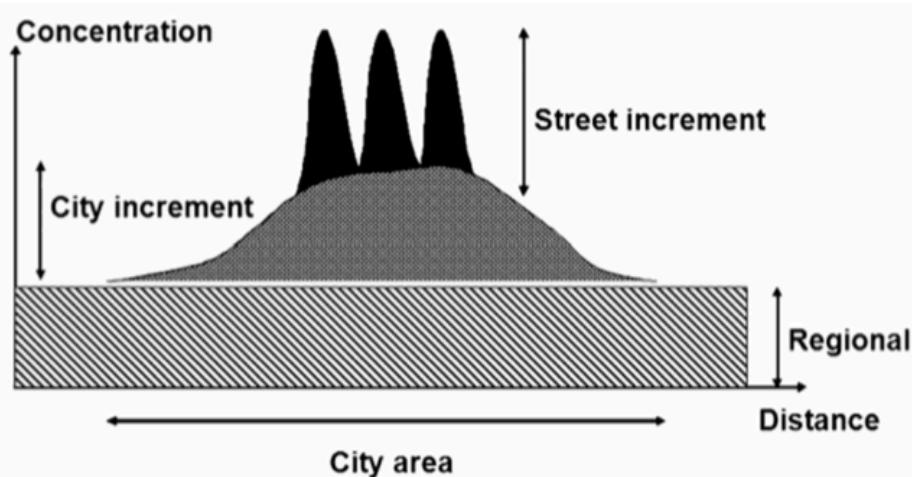


Figure 1 Air pollution concentration based on dispersion (Tate, 2018)

To understand dispersion of particles it is necessary to understand how particles emitted from various sources (primarily from TRAP) are influenced by meteorological conditions (wind speed, wind direction and atmospheric stability), emission height (ground level or high level source) and its interactions with urban topography (EEA, 2016).

Over large urban area, Pateraki et al., (2012) found particle concentration showed weak correlation to meteorological changes (wind speed, relative humidity and temperature) in comparison to PM<sub>2.5-10</sub> or PM<sub>10</sub> based on 53 month long duration in suburban area of Athens using Tapered Element Oscillating Microbalance (TEOM) and continuous air quality monitoring station. Hartog et al., (2005) also found weak correlation between PM<sub>2.5</sub> concentration and meteorological conditions (wind speed, relative humidity and temperature) based on measurements of 229 days in Amsterdam, 177 days in Erfurt and 182 days in Helsinki.

K.H. Kim et al., (2015) in Seoul studied influence of wind speed and direction at three different spatial scales micro-scale, middle-scale and neighborhood scale using particle bound polycyclic aromatic hydrocarbon (PAH) as an indicator of TRAP and found at micro scale concentration is significantly influenced by wind direction, at middle scale there was an exponential decrease from roadway to living environment. Aldrin and Haff, (2005) in their

model to understand relationship between hourly concentration of pollutants ( $PM_{10}$ ,  $PM_{2.5}$ ,  $NO_2$  and  $NO_x$ ) and traffic volumes and several meteorological variables found wind direction and wind speed have large effect on all pollutant concentration with increased relative humidity decreasing  $PM_{10}$  concentration but increasing  $PM_{2.5}$  concentration.

Analysing air quality in microenvironments in urban areas like at street levels is critical to effectively evaluate exposure to TRAP and in a given microenvironment pollution is not representative of only traffic emissions but also influenced by street orientation and prevailing weather within the road network based on study with 122 environmentally pervasive sensors in Newcastle (Galatioto et al., 2014). One of the primary reason for such high concentration in urban areas is poor street ventilation due to high density land use (Uehara et al., 2000). Urban canyon can be defined as space between the buildings and windward and leeward side of the street canyon based on the direction of the wind is shown in Figure 2 (Bourbia and Boucheriba, 2010). Aspect ratio is the ratio of width between the building covering street and pavements ( $w$ ) and height of the building ( $h$ ), if aspect ratio is 1 than it is called symmetric street canyon and if not it is called asymmetric street canyon (Santiago and Martin, 2005). Further, wind flows from modelling results for three different aspect ratio of 1, 0.5 and 0.25 are represented by a, b and c are shown in Figure 3 and Figure 4 (Santiago and Martin, 2005). For symmetric and asymmetric canyons pollution dispersion inside canyon is radically different due to different wind speed inside the canyon and primary vortex continuously sweeping pollutants from windward side pushing them to leeward side resulting in difference in concentration at windward and leeward side (Santiago and Martin, 2005).

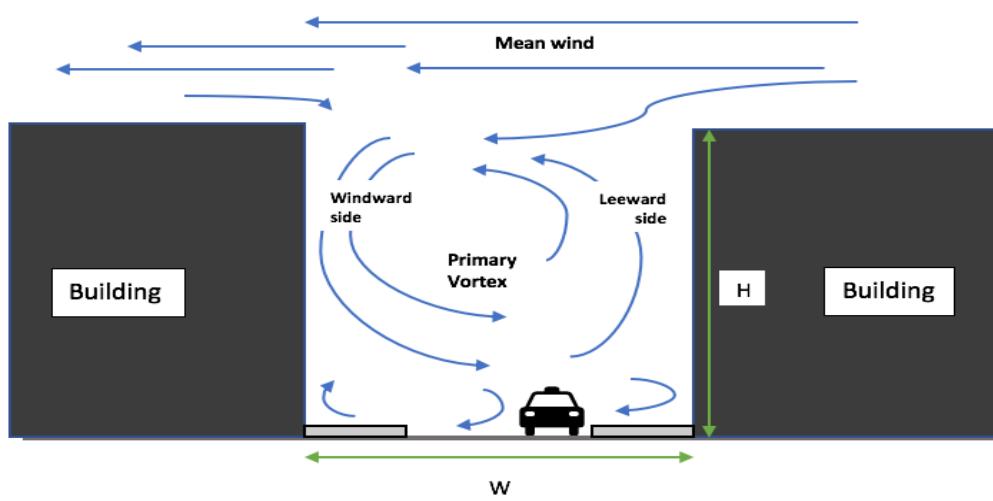


Figure 2 Urban Street Canyon [adapted from (Tate, 2018)]

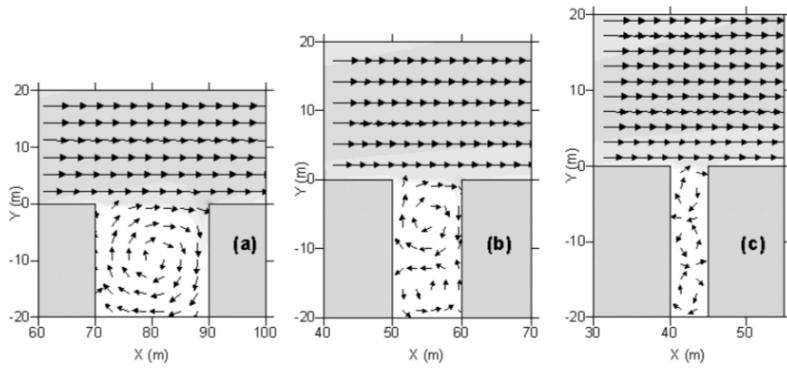


Figure 3 Wind flow in symmetric street canyon. (Santiago and Martin, 2005)

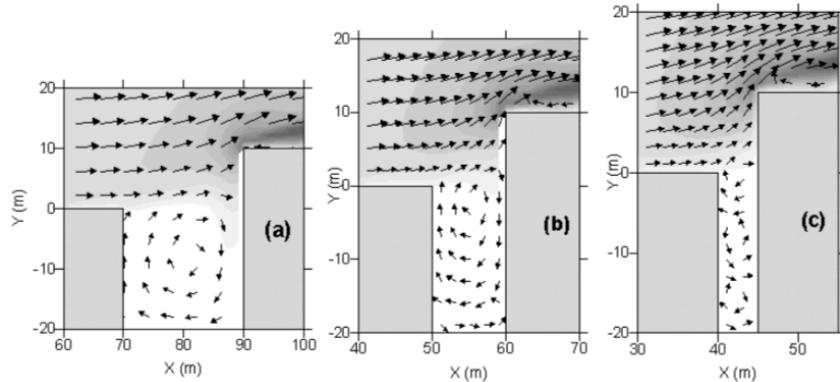


Figure 4 Wind flow in asymmetric street canyon. (Santiago and Martin, 2005)

When wind direction is in parallel to street canyons it results in sweeping of pollution from TRAP gets channeled through the canyon based on the direction of wind where depending on the length of the canyon and aspect ratio, the concentration increases uniformly from centre to the walls of the canyon until it is able to spread out at the end of street or at roof height within canyon affecting exposure within the street canyon from moving traffic (Solazzo et al., 2008; Soulhac and Salizzoni, 2010).

Natural phenomenon like rainfall can also affect the particle concentration, mathematical modelling suggests particles are removed via impaction or entrapment in the rain droplets depending on myriad factors like number density of water droplets and cumulative concentration of pollutants (Shukla et al., 2008). Analytical analysis by Sharma et al., (1983) at three locations in Kanpur for three years found scavenging of particulates during the monsoon season. Similarly, Ravindra et al., (2003) quantified an estimated 40 to 45 % reduction in suspended particulate matter (SPM) before and during initial rain of monsoon in Delhi.

## 2.4 Interaction and dosage of inhaled particles in lungs

As discussed about the complexity of interaction of pollutants in the atmosphere and built up area, similar is the situation inside the lungs where complex interactions happen between the inhaled pollutants and lungs. To understand these interactions, it is vital to define ‘oxidative stress’ and ‘free radicals’. Oxidative stress as per Holguin, (2013) is defined as “imbalance between increased oxidative sources and reduced or defective antioxidant mechanisms”. Free radicals as per Lobo et al., (2010) are defined as “molecular species capable of independent existence that contain an unpaired electron in an atomic orbital”.

In a key study by Kelly, (2003) on oxidative stress from exposure to air pollutants, stated free radicals in a biological setting are very dangerous as they can react indiscriminately with available molecules resulting in to unintended chemical reactions due to activation/deactivation of target molecules. Further, pollutants in the ambient air are free radicals ( $\text{NO}_2$ ) or aid free radical reactions (particulates) and create imbalance between antioxidants and free radicals during oxidative stress resulting into tissue injury or influx of inflammatory cells at the site of injury from a series of complex orchestrated reactions, a simplified process diagram is shown in Figure 5 (Kelly, 2003).

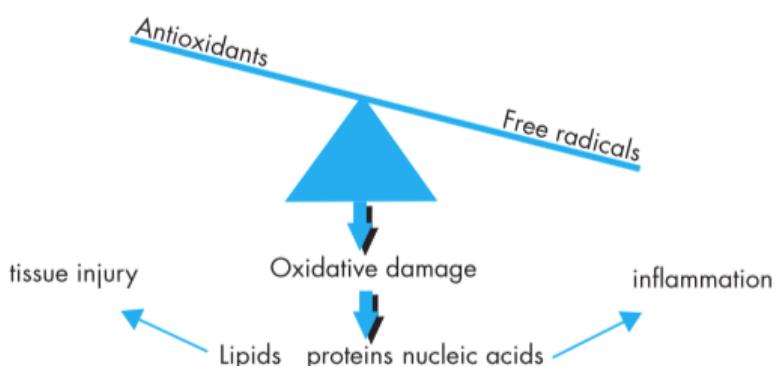


Figure 5 Mechanism of lung inflammation (Kelly, 2003)

However, it is important to note lungs are the only organ of which the largest surface area comes in contact with the ambient air and it has necessary defence systems to compensate the effect as a result of presence of alien/reactive species, yet these mechanism has its own limit and is affected by variety of factors such as individuals health and diet (Gomes and Florida-James, 2014). Size of the air pollutant is also of crucial importance particularly for PM to understand impact of lung function as they can penetrate or get captured in different parts of respiratory track based on their size as shown in Figure 6 (K.-H. Kim et al., 2015). The

resultant impact from pollutants is also influenced from the physical characteristics of individuals such as nose breathing/mouth breathing, breathing rate, ventilation and lung volume) (Brown et al., 2013).

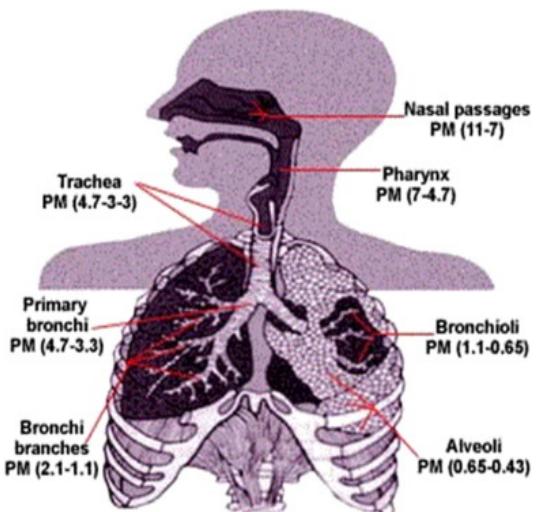


Figure 6 Deposition potential of particle based on size (K.-H. Kim et al., 2015)

One critical parameter for understanding health impact from inhaled pollutant is the amount of dose a subject's lungs actually gets deposited in lungs as there is a probability of particles being expelled out in the process of breathing (Int Panis et al., 2010). The key factor influencing the deposition probability is the size of the particle and hygroscopicity where the particle can increase in size by a factor of 1 (no growth) to 5 because of very high relative humidity (99.5%) in lungs compared to environment in which particles are inhaled from (Anselm et al., 1990; Löndahl et al., 2009). This suggests a measured particle concentration in the urban environment cannot be directly assumed to be dosed inside the lungs as the size may change once it enters into subjects body. After recommendation from National Research Council, (1998) to understand particle dosimetry for particle air pollution, a key study by Daigle et al., (2003) devised a deposition factor (DF) which measured DF for UFP deposition in lungs considering changes in breathing rate, ventilation and diffusion of air with 5 males and 2 females participants undergoing intervals of cycling and rest in controlled lab setting in which participants were exposed to exhaust to arrive at DF of  $0.83 \pm 0.04$  for Ventilation rate (VE)  $38.1 \pm 9.5$  L/min, there were no gender differences for reported DF. To increase the reliability of the measured dose DF is one critical factor and for this research I will use DF of  $0.83 \pm 0.04$  in comparison to contemporary studies (Schiller et al., 1988; Peter A. Jaques, 2000; Löndahl et al., 2009) of DF which were at rest conditions.

Second critical parameter is VE applied to calculate the overall dosage which is being applied differently by different studies producing dosage values which can influence results (Raza et al., 2018). For example, de Nazelle et al., (2012) applied standard VE using EPA handbook developed by office of Air Quality standards (Johnson, 2002) which was later adopted by Rojas-Rueda et al., (2016) and Rabl and de Nazelle, (2012) assumed dose is proportional to air intake of which air intake depends on the bicycling undertaken due to mode specific average Metabolic Equivalent (MET) as per detailed calculation from de Nazelle et al., (2009). Very few studies have actually measured VE and applied to calculate dosage (Int Panis et al., 2010; Namdeo Anil et al., 2016) to estimate reliable dosage values for different mode in same city or comparing values in different cities. Thus, to increase the reliability of results for this study we will be measuring VE.

Epidemiological studies on health impact from active travel and air pollution found exposure for cyclist is lower in comparison to car drivers (van Wijnen et al., 1995; Rank et al., 2001) but the increased dosage from higher VE due to physical effort results into higher dosage for cyclist and thus choice of route can play a critical role for inhaled dosage of cyclist (de Hartog et al., 2010). However, evidence for short term impact for short trips like daily commute are limited in comparison to long term impacts (WHO, 2006).

## 2.5 Methods used and evidence of lung function impact from particle dosage

Air pollutants have short term and long term health impacts and that is the rationale for some pollutants having short term and long term limit values, increased concentration of particulates ( $PM_{10}$  and  $PM_{2.5}$ ) result in to increased mortality or morbidity both daily and over longer duration (WHO, 2018). Currently in UK, limits for  $PM_{10}$  and  $PM_{2.5}$  are set as shown in Table 2.

*Table 2 Short term and long term PM limits for UK (DEFRA, 2018a)*

Pollutant	Objective	Concentration measured as
PM10	50 ug/m <sup>3</sup> not to be exceeded more than 35 times a year	24 hour mean
	40 ug/m <sup>3</sup>	annual mean
PM2.5	25 ug/m <sup>3</sup>	annual mean

Epidemiological studies have used myriad methods and biological indicators to assess the impact on lung function from exposure to TRAP, Table 3 shows key methods used to assess lung function.

*Table 3 Methods to assess lung health (NHLBI, 2019)*

Method	Description
Spirometry/Lung volume test	Most accurate way to measure how much air your lungs can hold and estimates rate of air flow and estimates lung size. Measures fractional exhaled volume in 1 second (FEV1), fractional exhaled volume in 6 seconds (FEV6) and forced expiratory flow (FEF)
Lung diffusion capacity	Assesses how well the oxygen gets into the blood from the air you breathe. Might require drawing of blood to measure haemoglobin level.
Pulse Oximetry	A probe is fitted on the skin surface which measures oxygen level in blood.
Arterial Blood gas tests	Measures level of gases like oxygen and carbon dioxide in blood. Requires blood to be drawn and to be performed under supervision of medical professional
Fractional Exhaled Nitric Oxide (FeNO)	Measures the amount of nitric oxide in exhaled breath which is an indicator of lung inflammation

In a landmark study by McCreanor et al., (2007) in London, 60 adults with mild or moderate asthma walked for 2 hours along Oxford street and on separate occasion walked in Hyde park found reduction in FEV<sub>1</sub> (up to 6.1%) and FVC (up to 5.4%) which were significantly larger than reduction to reduction in FEV<sub>1</sub> and FVC after exposure in park ( $p=0.04$  and  $p=0.01$  respectively). Also, increased inflammatory changes in sputum and exhaled breath condensate (EBC) were noted. Int Panis et al., (2017) in their retrospective cohort study based on data from worker health surveillance program in Belgium of routinely performed lung function measurement on weekdays for 2449 employees (aged 16-70 years with 72% male participants) for four year period (2011-2015) and measuring pollutant concentration from nearby monitoring station showed 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> on the day of examination was associated with 18.9 mL lower FVC (95% CI: -27.5 to -10.3,  $p<0.0001$ ), 28.1 mL lower FEV<sub>1</sub> (95% CI: -19.1 to -6.5,  $p<0.0001$ ) and FEV<sub>1</sub>/FVC did not change. Rice et al., (2013) studied short term exposure to air pollution in 'moderate' range as per EPA guidelines for 3262 non-smoking participants living within 40 km in Boston, USA was associated with 20.1 mL lower FEV<sub>1</sub> for PM<sub>2.5</sub> (95% CI -33.4 to -6.9). Similar association of degradation in spirometry parameters for participants who exercised outdoors regularly versus non-exercisers in Delhi was shown by Kesavachandran et al., (2015). Sharma et al., (2004) assessed impact on lung function for 91 participants within 2 kilometers of three different study site representing different concentration of particulates showed higher reduction in FEV<sub>1</sub> and FVC at sites with higher concentration.

In addition, Adam et al., (2015) studied chronic impact of ambient air pollution on lung function for 7613 adult participants from five cohorts in the European Study of Cohorts for Air Pollution (ESCAPE) using spirometry reported increase in  $10 \mu\text{g}/\text{m}^3$  in  $\text{PM}_{10}$  was associated with lower  $\text{FEV}_1$  (-44.6 mL, 95% CI -85.4 to -3.8) and  $\text{FVC}$  (-59.0 mL, 95% CI -112.3 to -5.6). Downs et al., (2007) randomly selected 9651 adults (18-60 years age) with a validated  $\text{PM}_{10}$  dispersion model assigned to 4742 addresses for assessment comparing years 1991 and 2002 showed net effect of decline in  $10 \mu\text{g}/\text{m}^3$  for  $\text{PM}_{10}$  over a 11-year period would reduce annual rate of decline in  $\text{FEV}_1$  by 9% and  $\text{FEF}$  by 16%.

However, Lagorio et al., (2006) studied changes in lung function of ambient air pollution for adult subjects with pre-existing chronic obstructive pulmonary disease (COPD), asthma and ischemic heart disease (IHD) for 67 days showed negative association between  $\text{PM}_{2.5}/\text{PM}_{10}$  and  $\text{FEV}_1$  and  $\text{FVC}$  but interestingly hinted that the metallic fraction of zinc (Zn), Iron (Fe) and nickel (Ni) in  $\text{PM}_{2.5}$  had negative effect on lung function for subjects with COPD. This could be the result of varying characteristics of PM due to variety of particle species combining in myriad ways and certain resulting species having differing health impacts (Davidson et al., 2005). Jarjour et al., (2013) studied impact on lung function in terms of spirometry parameters based on route choice for bicycling on routes with low and high levels of traffic reported no difference in volumetric lung function for 15 healthy adult participants.

### **Fractional exhaled Nitric Oxide (FeNO)**

Endogenous nitric oxide (NO) is one of the signaling molecule for variety of biological functions within the human body and in lungs NO is an important mediator of the inflammatory response (Flamant-Hulin et al., 2009). Use of FeNO based on increasing development in the field has contributed to management of lung diseases by assessing the level of inflammation and is already in use for treatment in asthma control (American Thoracic Society and European Respiratory Society, 2005). As a result, it is widely accepted that FeNO is a reliable marker of lung airway inflammation (Annesi-Maesano and Dinh-Xuan, 2016). A composite distribution of FeNO results from different studies for population without stable asthma and with stable asthma was developed by Dweik et al., (2011) and is shown in Figure 7, subjects without stable asthma median value was 16 ppb with range of distribution from 2.4 to 199 ppb while for subjects with stable asthma 95% confidence interval of 22 to 44 ppb was reported.

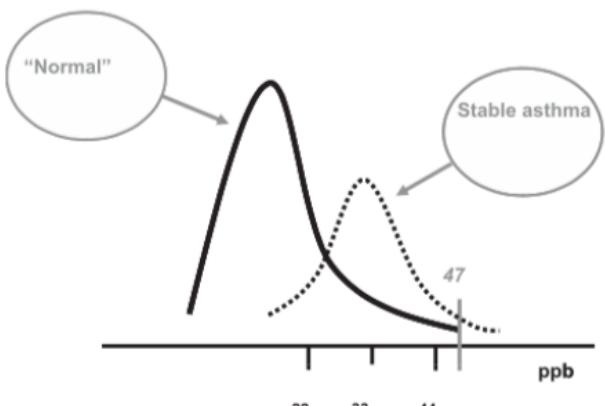


Figure 7 Distribution of FeNO values in population with/without asthma (Dweik et al., 2011)

Kubesch et al., (2015) studied impact on lung function for 28 healthy participants which were subjected to high and low levels of TRAP in real world conditions during 2 hours of intermittent cycling found statistically significant increase of 0.803 ppb in FeNO at 95% confidence interval [0.035-1.571,  $p=0.04$ ] for increased coarse PM exposure. While increase in FEV<sub>1</sub>, FVC and FEF by 34 mL, 29 mL and 91 mL respectively was statistically significant at  $p\leq 0.05$ . Berhane et al., (2014) based on cohort study of 1211 school children in southern California for measurements in year 2006/07 and 2007/08, found increase in annual average concentration of 24 average for PM<sub>2.5</sub> and NO<sub>2</sub> were associated with increased FeNO levels. Further, Barraza-Villarreal Albino et al., (2008) in a cohort study of 158 asthmatic and 50 non-asthmatic schoolchildren used FeNO as indicator of lung inflammation and showed exposure to PM<sub>2.5</sub> resulted into airway inflammation for asthmatics as well as non-asthmatics. Other studies using different TRAP pollutants have also found increase in FeNO and worsening of spirometry parameters (Jansen et al., 2005; Kan et al., 2007; Khatri et al., 2009; Renzetti et al., 2009). The contrary, of improvement in FeNO levels with clean air is also shown by Pedroletti et al., (2009) where 22 adult participants were subjected to controlled air after filtering through HEPA filters during their sleeping hours which resulted in statistically significant decrease in FeNO levels.

However, Gong et al., (2008) for 14 healthy and 13 asthmatic participants did not find significant increase in FeNO when studied UFP exposure in controlled chamber exercising at VE of 15-20 L/min. Interestingly, Hussain et al., (2012) for 16 mild to moderate asthmatics showed post exposure to primary diesel exhaust found increased nitrite in EBC but no elevated FeNO levels indicating presence of non-inflammatory oxidative stress. It should also be noted, determinants affecting results of FeNO are cigarette smoking, allergen sensitization, asthma treatment, height, sex and age (Scarpa et al., 2014). Also, inherently FeNO has a strong

association with flow rate (exhaled flow rate of breath into the instrument) and changes in airway size resulting in ‘bronchoconstriction’ from natural or anthropogenic stimuli which can reduce the reliability of FeNO values as airway wall NO flux and airway NO diffusing capacity is affected (Cattoni et al., 2013).

From above it is evident that health impact studies work in isolation from dosage calculations and tend to associate degradation in health in relation to pollution levels, in contrast dosage studies are limited to understand the commuter’s choice of mode and pollutants inhaled which leads to health impact. These two strands of research are not different but one and I try to reduce this gap by calculating dosage and analyse impact on lung function.

## 2.6 Chapter summary

In this chapter we learnt meteorological parameters along with built up area can significantly affect the dispersion of pollutants resulting in to different dosage that individuals undergo while undertaking various physical activities in urban areas. Lungs have certain immune mechanism to compensate the effect of air pollutants however if exposure creates imbalance in oxidants and anti-oxidants it results in to lung inflammation followed by tissue damage and lung volume capacity. Spirometry parameters (FEV<sub>1</sub>, FEV<sub>6</sub>, FVC and FEF) and FeNO have been reliably used in a variety of epidemiological studies to understand the impact on lung function from TRAP. Thus, we will be using spirometry parameters and FeNO to understand the impact on lung function from different exposure environment for research questions set in section-1.4.

### 3 Methodology

#### 3.1 Chapter overview

This chapter discusses the experiment design along with the instrument used for collecting data for the research objective set in section-1.4. Section-3.2 provides detailed explanation of the design of experiment. Section-3.3 and section-3.4 provides description of polluted and controlled route respectively. Section-3.5 provides overview of participants. Section-3.6 discusses instruments used for data collection. Section-3.7 discusses data processing and cleaning of the recorded data. Section-3.8 shows the computation of common indicator dosage and interpretation of lung function impact for FeNO and spirometry.

#### 3.2 Experiment Design

The experiment was planned for eight days and was undertaken during the AM peak hours (8:00 – 10:00 AM) of week 18 and 19 in June'19 excluding weekends. Two participants were required to bicycle on controlled route while other two on polluted route with same start time and end time during the AM peak. During the duration of bicycling participants were equipped with condensed particle counter (CPC) to record particle concentration. FeNO and spirometry parameters were measured before and after undertaking bicycling. A simplified diagram of experiment design is shown in Figure 8 and a detailed procedure for a particular day is shown in Figure 29 in APPENDIX-1.

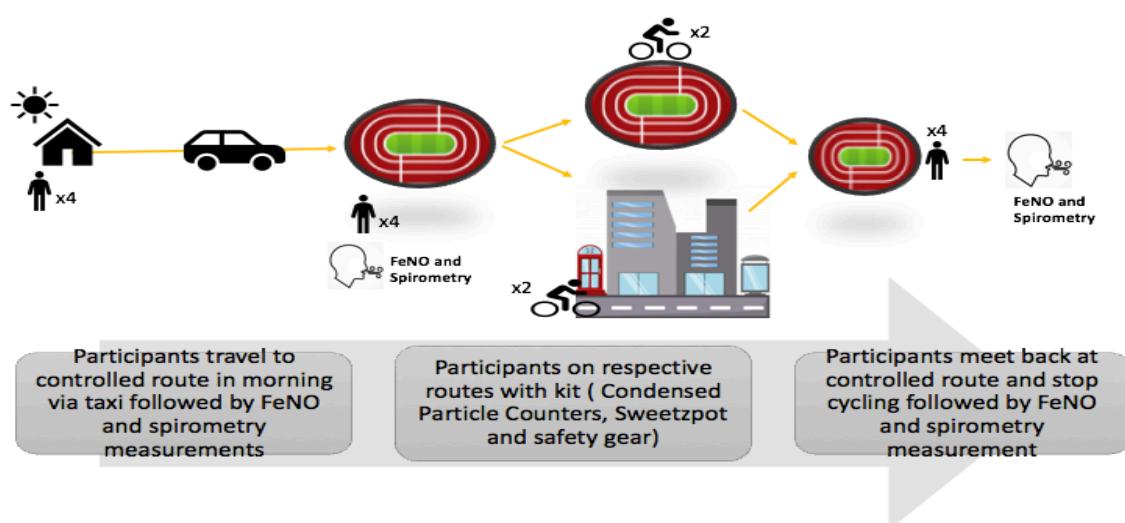


Figure 8 Overview of Experiment Design (Khatri, 2019b)

To avoid contamination of participants from prior exposure to pollution all four participants (A, B, X and Y) were transferred to polluted route via taxi which was powered by internal

combustion engine and windows were kept open during commute. Of the four participants, three participants were located within 20 meter radius while fourth participant was picked up on the way to controlled route. Also, while loading instruments for data collection in the taxi driver was requested to switch off the engine and the same was applicable while unloading upon reaching the controlled route.

Once participants reached controlled route FeNO and spirometry values were recorded for all the participants using data collection sheet shown in Figure 34 in APPENDIX-1. In parallel, dedicated CPC based on route were packed in rucksack with packaging material like bubble wrap, placed in such a way that it would be in position like held in held once the cyclist was on bicycle and can be seen in Figure 36 in APPENDIX-1. For polluted route CPC's a PTFE pipe was assembled to the CPC at inlet which was then exposed to atmosphere at shoulder level as shown in Figure 36 in APPENDIX-1. One CPC in exception was kept on the centre of the controlled route.

After CPC's were made ready, recording for sweetspot devices were initiated and checked if it was recording data. Followed by simultaneously initiating recording in STRAVA and putting CPC into log mode. Participants with their respective rucksack started cycling on their respective route as allocated in Table 4 below.

*Table 4 Participant route allocation*

Route	day-1	day-2	day-3	day-4	day-5	day-6	day-7	day-8
Controlled route	AB	XY	AX	BY	AB	AX	BY	XY
Polluted route	XY	AB	BY	AX	XY	BY	AX	AB

Once the cyclist from polluted route return back to controlled route cyclists on controlled route stopped cycling followed by ending trip on STRAVA. After which FeNO and spirometry data were recorded for each participant resulting in to end of data collection for the day. The same process was repeated for the duration of study. Route allocation to participants was done in a manner to record at least four observations for each participant on each route. Participants were swapped among the routes to establish a possible trend of lung function impact based on route allocated by day to test the research objective set in Section-1.4.

### 3.3 Controlled Route description

Figure 9 shows the ariel view of controlled route (Brownlee center) located at 53.8430° N, 1.5887° W coordinates in Leeds, UK (GoogleMaps, 2019). It is a paved bicycle track of length one mile. There are two roads in the vicinity, A660 on one side and A6120 on the other side. There is a limited built up area around the track thus there is very minimal possibility of building up of pollution from lack of dispersion as discussed in section-2.3. There is no presence of point emission sources in vicinity as shown in Figure 32 in APPENDIX-1 (EPRTR, 2017). With very good dispersion due to no built up area and location being outside the city pollution levels are expected to be low in comparison to kerbside as discussed in section-2.3, making it the most desirable site as controlled route. Zoomed out view of controlled route is shown in Figure 31 in APPENDIX-1.



Figure 9 Controlled Route (GoogleEarth, 2018)

### 3.4 Polluted Route description

Figure 10 shows polluted route with Brownlee centre (marked as flag) as starting point and end point to Brownlee centre via Parkinson building. The total distance covered in round trip 7.4 miles (GoogleMaps, 2019). Route includes features like street canyon and heavily built up areas across certain sections of the road which make dispersion of pollutants difficult thus increasing the dosage as discussed in section-2.3. Gradient profile from Brownlee centre to Parkinson building is shown in Figure 30 in APPENDIX-1 (GoogleMaps, 2019). AURN was located at latitude 53°81'99.72"N longitude 1°57'63.61"W along the route measuring pollutants NO, Nitrogen dioxide (NO<sub>2</sub>), NO<sub>x</sub> and PM<sub>10</sub> and PM<sub>2.5</sub> starting from 17th Feb'08 (DEFRA, 2019c). Annual average daily traffic counts at point-A and point-B in Figure 10 are

15840 and 17884 respectively which shows significant traffic movement (DfT, 2019). Thus, heavy traffic along the route with varying dispersion conditions from built up area, total journey time and presence of AURN with good resolution data made the route most desirable. For some part of the track bicycle lanes and bus lanes coincided in which cyclists were trained to stop if the bus was leaving and if boarding/alighting passenger than overtake it with safety as highest priority. Also, cyclist would stop at the Parkinson building to check if the equipment's were still recording. A zoomed out view of polluted route is shown in Figure 33 in APPENDIX-1 which also provides overview of built-up area along the route. At weetwood roundabout cyclist were advised to walk through a pedestrian crossing for safety reasons, for the rest of the route participants cycled as per traffic rules with visibility vests and helmets.

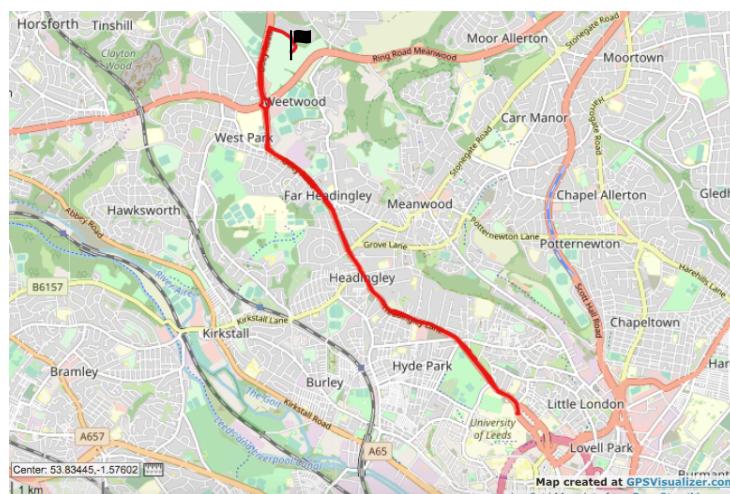


Figure 10 Polluted Route (GPSVisualizer, 2019)

### 3.5 Participant Description

Overview of participants is shown in Table 5. Participant-A has higher height in comparison to other participants while participant-B has history of asthma are two key standout physiological differences. Also, participant-A has been a regular cyclist and participant-B a regular swimmer while other two participants did not undergo additional physical activity apart from walking for their commutes. The single most constrain because of which selection process was not undertaken was availability over the period of two weeks which were blocked for the experiment. Additional days were blocked to account for weather variability. All participants were shown how to use medical equipment's to record FeNO and Spirometry values in a training session along with respiratory consultant. All participants were provided with safety orientation and overview of the route prior to experiment.

*Table 5 Overview of study participants*

	Participants			
	A	B	X	Y
Weight (kg)	72	88	70	62
Height(cm)	193	170	273	162
Ethnicity	British	Indian	Colombian	Colombian
Sex	Male	Male	Male	Male
Smoking history	No	No	No	No
Asthma history	No	Yes	No	No
exercise	Cycling	Swimming	Walking	Walking

### 3.6 Equipment's

#### 3.6.1 Particle Counter

Particle number concentration (PNC) was measured using TSI 3007 CPC. The equipment is capable of measuring particle size in the range of 0.01 to >1.0  $\mu\text{m}$  and concentration range of 0 to 100,000 particle/cm<sup>3</sup> with concentration accuracy of  $\pm 20\%$  (TSI, 2019b). Response time is less than 9 seconds for 95% of the response and ambient temperature operating range is 10 to 35 degree celcius (TSI, 2019b).

Equipment is shown in Figure 11 and working schematic is shown in Figure 12. Flow rate at the inlet of the equipment as shown in Figure 11 is 700 cm<sup>3</sup>/min but particles are detected at 100 cm<sup>3</sup>/min (TSI, 2019b). During operation sample air is drawn via inlet continuously, a heated saturator where isopropyl alcohol is vaporized and diffused with the sample stream, this mixture of air and vaporized alcohol passed on to a condenser where alcohol vapor is supersaturated which then uses the particle sites to condense which then becomes a larger alcohol droplet that pass through optical detector where they are counted (TSI, 2019b). This equipment is sensitive to vibration and position in which they are held for which all the CPC's in the rucksack were surrounded by packaging material like bubble-wrap and kept in a position that would replicate that as if it was held in hand once participant is in cycling stance as shown in Figure 36 in APPENDIX-1. Going forward CPC's will also be referred as ap-x where x is the CPC number for identification.



Figure 11 TSI 3007 CPC (Khatri, 2019a)

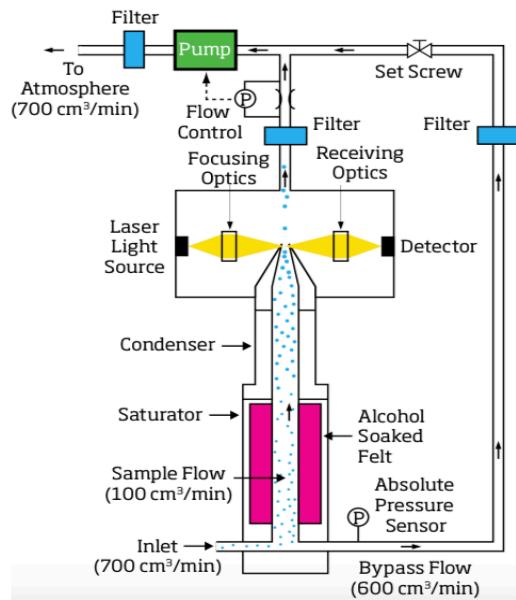


Figure 12 Operating schematic of TSI 3007 CPC (TSI, 2019b)

### 3.6.2 Spirometer

Lung function volumetric parameters ( $FEV_1$ ,  $FEV_6$ ,  $FEV_1/FEV_6$  and  $FEF$ ) were measured with Vitalograph lung monitor usb 4000 (Vitalograph, 2019). The equipment has accuracy of  $\pm 3\%$  and operating temperature range of 17 – 37 deg C (Vitalograph, 2019). Flow impedance of the device is less than 0.15 kPa/L/s at 14 L/s (Vitalograph, 2019). Figure 13 shows the instrument and stator-rotor principle on which it operates. Participant exhales breath in disposable mouthpiece which spins in a swirl induced by multi-vane stator which makes the flat blade (rotor) spin, the blade blocks the light source as it spins and a detector measures mass air flow based on number of revolutions which is approximately proportional to flow and are calibrated at the time of production (Vitalograph, 2019). It is critical that there is no restriction

for air to escape while holding the instrument and was covered during the training and was monitored for daily recordings to be as per protocol. Each participant had to perform the maneuver 3 times for before and after readings.



Figure 13 Spirometer with stator-rotor arrangement (Vitalograph, 2019)

### 3.6.3 NObreath

Lung inflammation with FeNO as a biological indicator is measured via NObreath which measures exhaled nitric oxide in parts per billion (ppb) with accuracy of  $\pm 5$  or 10% whichever is greater at flow rate of 50 mL/sec at 10 cm water and concentration range of 5-300 ppb (Bedfont, 2019). The instrument should be operated within the temperature range of 10° – 30° C (Bedfont, 2019). The sensor drift is 5% per year but since the instrument was bought few months before the experiment the results are expected to be very reliable (Bedfont, 2019). The instrument consist of electrochemical sensor which is sensitive to NO and reacts in its presence to produce an electrical output, NO free air flows into sensor while it is in ‘on’ condition but not during the test (Bedfont, 2019). Figure 14 shows the instrument, mouthpiece through which participant exhales breath looking at flow indicator band within which the ball should stay over the guided visual bar informing participant about completion of test on display followed by display FeNO result. There is a vent hole at the rear of the instrument which should not be blocked under test condition and same was covered under training of participants and test were monitored for daily recordings to be as per protocol based on ATS/ERS guidelines. NObreath performance as a portable device in comparison to induced sputum analysis in clinical setup is already established and shows close correlation for airway inflammation (Deykin et al., 2002; Yune et al., 2015).



Figure 14 Bedfont NOBreath instrument (Bedfont, 2019)

### 3.6.4 Sweetzpot

Sweetzpot flow was used to measure the breathing rate, VE and heart beat (Sweetzpot, 2019). Breathing rate and breathing flow are measured by measuring ventilation which is the amount of air that enters and goes out of the lungs (Sweetzpot, 2019). Sweetzpot with an elastic harness worn around the subjects torso which expands and contracts during the breathing process and reports breathing rate and VE (Laugstol, 2017). The expansion and contraction of the belt is converted into flow and VE value via strain gauge as shown in Figure 15 (Sweetzpot, 2019). Prior to use the instrument needs to be worn tightly and calibrated to maximum and tidal breathing value of an individual whose data is being recorded via manufacturers mobile application and all measurement must be done after calibration (Sweetzpot, 2019). All the participants were calibrated to sweetzpot prior to experiment and were allocated the same device for the study period. Instrument in strapped condition is shown in Figure 37 in APPENDIX-1.

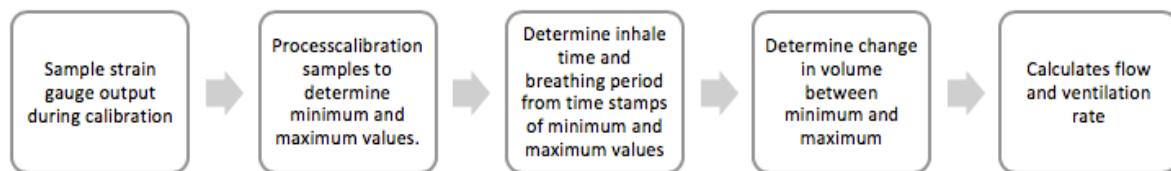


Figure 15 Sweetzpot calculation methodology [adapted from (Laugstol, 2017)]

### 3.6.5 Mobile Phone

Each participant had a mobile phone with STRAVA, (2019) and Sweetzpot application, which were allocated to each participant and same allocation was used for the duration of experiment. STRAVA data GPS data and timestamps were used as master for the experiment.

### 3.6.6 Bicycle

All four bicycles used were with gear mechanism, well maintained and appropriate air in tyres which were not disturbed for the duration of experiment. Each participant was allocated a cycle for the entire experiment and were free to use gears as per his comfort level whilst maintaining speed between 10-15 km/hr except downhill. Figure 38, Figure 39, Figure 40 and Figure 41 represent the cycles used by participant -A, B, X and Y respectively in APPENDIX-1.

## 3.7 Data processing

### 3.7.1 Data record rate

Data collection duration was reduced to 7 days due to heavy rains in second week. Figure 16 shows the overview of data collection rate for the experiment. Weather data for the duration of data collection is shown in Table 6.



Figure 16 Data collection overview

Table 6 Weather data for duration of data collection (Worldweatheronline, 2019)

Day	Weather type	Temp (° C)	Wind (km/hr)	Humidity (%)	Cloud %
day-1	Sunny	14	22	55	4
day-2	Moderate rain	15	12	80	80
day-3	Sunny	14	19	61	55
day-4	Sunny	15	8	53	19
day-5	Cloudy	14	14	69	100
day-6	Moderate Sunny	14	13	71	77
day-7	Rainy	10	18	85	100

CPC data collection rate was at acceptable level except CPC-6 on 4<sup>th</sup> June'19 did not record sufficient readings, however since both the cyclist on the polluted route were cycling relatively close to each other recording of CPC-10 will be applied for analysis. Sweetspot data was

severely compromised as the instrument did not work as intended for reasons unknown. To address this issue 4 more bicycle run with sweetzpot only were done with origin and destination reversed on the same route during morning peak by participant B and Y and same VE will be applied wherever data is lost for respective participant. For participant-X, data from 7<sup>th</sup> and 13<sup>th</sup> July for polluted and controlled route will be applied for the rest of the days for respective routes. Similar strategy was adopted for participant-A. There were no data losses on spirometry and NObreath parameters except for participant-X on day-1 when he not able perform spirometry maneuver 3 times correctly.

### 3.7.2 Data cleaning

Data sets were combined in Microsoft excel and then trimmed down to a common time series based on STRAVA recordings. Units were converted appropriately for analysis. Any extra readings outside the start and end time's of STRAVA were removed from analysis. Since the CPC's and respective STRAVA application on the phone were always started simultaneously CPC recordings were aligned with STRAVA timestamps. Finally, this was converted into comma separated variable file. It is important to note that as described in section-3.4 participants stopped at Parkinson building to check if CPC's were working is not excluded from dosage calculation and treated like an additional stop at junction.

### 3.7.3 Instrument and data Quality Assurance

CPC's underwent flow check and results shown in Table 7 and were found meeting inlet flow requirement of  $1.0 \pm 0.05$  L/min (TSI, 2019a). Also, regression for CPC on polluted (ap-6 and ap-10) and controlled (ap-8 and ap-9) route shown in Figure 42 and Figure 43 respectively in APPENDIX-1. Regression results shown statistically significant positive correlation among the two CPC's used on respective routes. AURN data used should be treated with caution as data was not ratified on the date of download. NObreath, Spirometer and Sweetzpot used for data collection were purchased three to four months prior to data collection and were already calibrated. For NObreath the flow-pipe with valve was replaced post 50 readings as per guidance (Bedfont, 2019).

Table 7 CPC Flow check data

	Trials	ap6	ap8	ap9	ap10
<b>Zero Flow (l/m)</b>	t-1	0.002	0.002	0.008	0.002
	t-2	0.003	0.009	0.003	0.003
	t-3	0.002	0.008	0.005	0.004
<b>Flow (l/m)</b>	t-1	0.692	0.718	0.699	0.655
	t-2	0.691	0.72	0.694	0.656
	t-3	0.695	0.722	0.687	0.656

### 3.8 Analysis

For analysis Minitab, (2019b), open source software ‘R’ (R Team, 2008) along with ‘openair’ (Carlsaw, 2018) and ‘ggplot2’ (Hadley, 2016) packages were used. Analysis of individual trip for spirometry and lung inflammation is restricted to descriptive statistical analysis as sample size restricted us to group participants to apply t-test.

#### 3.8.1 Calculating inhaled dose and Interpreting lung function impact

To understand the impact on lung function from exposure to particle pollution it is necessary to have a common indicator across the participants to compare. As participants differ themselves in various physiological characteristics and different exposure to particle concentration at both the sites. Thus, we will compute dosage for each participant every day for amount of pollution inhaled in terms of particles. As discussed in section-2.3 on how pollution inhaled by participant is not directly dependent on particle concentration and VE only but also DF. For this study I have adopted the method used by Namdeo Anil et al., (2016) using below equations with units in () brackets to calculate the dose as a common indicator which can be used to compare with lung function impact for and amongst the participants.

$$\text{Inhaled air volume (m}^3\text{)} = [\text{Ventilation (L/min)}/1000] * \text{Journey duration (min)}$$

$$\text{Inhaled particles (particles)} = \text{Inhaled air volume (m}^3\text{)} * \text{particle concentration (particle/m}^3\text{)}$$

$$\text{Inhaled dose (particle)} = \text{Inhaled particles (particles)} * \text{DF}$$

$$\text{Inhaled dose (particle/km)} = \text{Inhaled dose (particle) / distance travelled (km)}$$

DF of  $0.83 \pm 0.04$  which was noted at VE of  $38.1 \pm 9.5$  L/min range is the best available DF with an similar experimental setup used by Daigle et al., (2003) where participants did not cycle continuously and were at rest for some part of the route (example: free-wheeling during the slope) and came closest to our experiment conditions. Although VE vary differently for

different participants in different conditions, this is the best available value that I could find and apply for the study and also adopted for dosage studies (Namdeo Anil et al., 2016; Int Panis et al., 2017).

To interpret the level of inflammation in lung airways we will be using established evidence based values as per approved guidelines by American Thoracic Society (ATS) as shown in Table 8 (Dweik et al., 2011). Similar guidance is also suggested in NObreath instrument manual (Bedfont, 2019).

*Table 8 Interpretation of FeNO result [adapted from (Dweik et al., 2011)]*

	FeNO < 25 ppb	FeNO 25 - 50 ppb	FeNO > 50 ppb
Interpretation	Airway inflammation unlikely	Cautious indication of airway inflammation	Inflammation present

Spirometry parameters of FEV<sub>1</sub>, FEV<sub>6</sub>, FEV<sub>1</sub>/FEV<sub>6</sub> and FEF will be used to assess the impact on lung volume function impact as discussed in section 2.5 with one difference of use of FEV<sub>6</sub> instead of FVC. Epidemiological studies have already established this is as a safe surrogate, Vandevoorde et al., (2005) studied the efficacy of surrogate indicator FEV<sub>6</sub> for 11,767 (7010 men and 4666 women) examinations and concluded FEV<sub>6</sub> as valid surrogate indicator for FVC, additionally advantage of using FEV<sub>6</sub> instead of FVC is that it is more explicitly defined in comparison. Swanney et al., (2000) from analysis of 502 (209 men and 128 women) patients also concluded FEV<sub>6</sub> can be used instead of FVC to interpret results. Spirometer used for the study with low flow impedance had limitation of measuring low flow values because of which established surrogate indicator of FEV<sub>6</sub> is calculated instead of incorrectly measuring FVC and not affect reliability of results.

To understand the volumetric and inflammatory change in lung function as an impact from inhaled pollution dose I will be using (Global Initiative for Chronic Obstructive Lung Disease) standard used for COPD patients wherein ratio of FEV<sub>1</sub>/FEV<sub>6</sub> if between 0.7 and 0.8 is deemed normal while below 0.7 indicates airway obstruction. Similarly, FEV<sub>1</sub>/FEV<sub>6</sub> ratio will be used as an primary indicator for volume for this study (GOLD, 2019). FEF is associated with flow in small airways and thus affecting the gaseous exchange which deteriorates prior to impact in volumetric lung function (Newman and Stinson, 2012), meaning change in FEF irrespective but no change in FEV<sub>1</sub>/FEV<sub>6</sub> is not an indication of absence of impact in lung function.

## 4 Results and Discussion

### 4.1 Chapter Overview

In this chapter results are presented for the research objective of assessing lung function impact in different exposure environment as set in section-1.4, followed by discussion of results, limitation and contributions. Divided into two parts, section-4.2 shows the results from data collection for the research objectives. First, by grouping Spirometry and FeNO parameters are grouped for participants by route and t-test performed. Second, discerning a trend in the data for individual participants using graphical visualization. Section-4.3 discusses the results in the wider context of literature with contribution and limitations of the study. Table 18 in APPENDIX-2 shows the computed dosage values as discussed in section-3.8.1 and Table 17 shows reported before and after reported values of lung function parameters for each participant by day will be used throughout the chapter.

### 4.2 Results

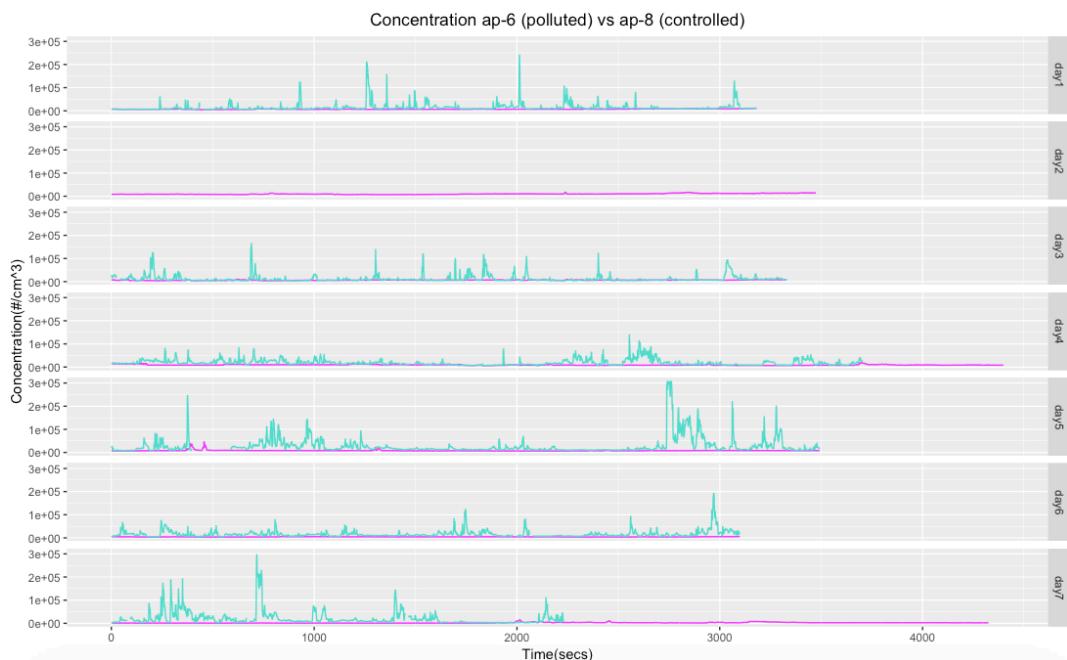
Particle concentration on polluted route was consistently higher in comparison to controlled route.

Table 9 shows if the mean concentration difference of CPC on polluted route in comparison to one on controlled route is statistically significant at 98% confidence interval. Ap-6 on polluted route and ap-8 on controlled route as one pair show average concentration on polluted route was higher for all 6 days (day-2 data not available). Similarly, ap-10 on polluted route and ap-9 on controlled route as second pair show average concentration on polluted route were higher for all 7 days. Figure 17 and Figure 18 shows temporal distribution of particle concentration comparing one CPC on controlled and other on polluted route indicating higher concentration and events of exceedances.

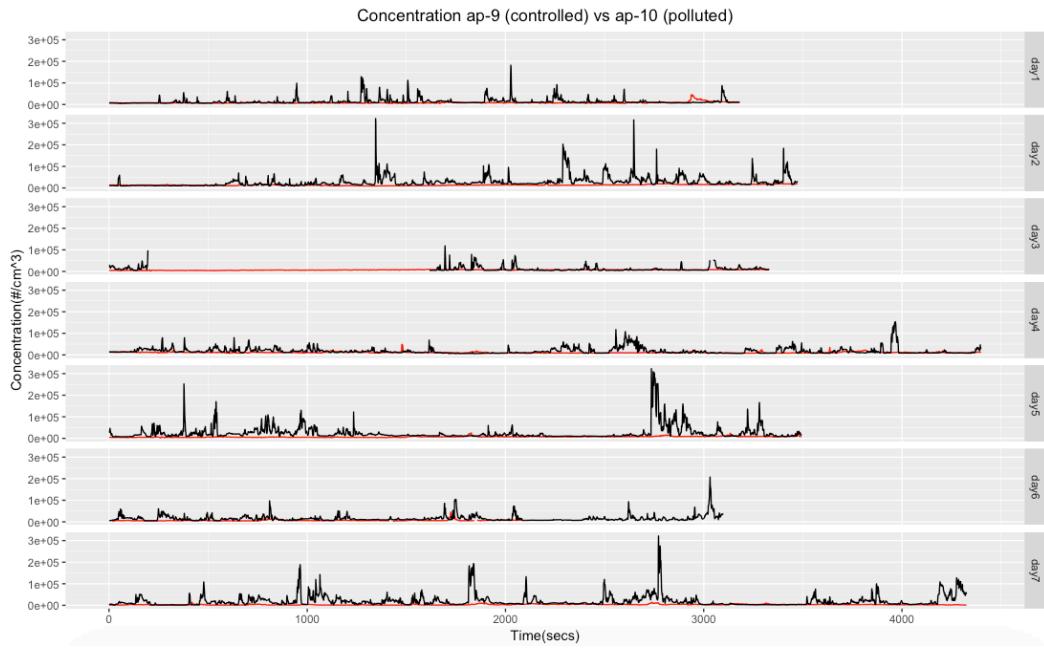
*Table 9 Particle concentration difference between controlled route and polluted route (\* statistically significant)*

Day	ap-6 mean concentrato n (#/cm3)	ap-8 mean concentrati on (#/cm3)	Mean diff (#/cm3)	98% CI	pvalue	ap10 mean concentrati on (#/cm3)	ap-9 mean concentrati on (#/cm3)	Mean difference (#/cm3)	98% CI	pvalue
day-1	14549	6941.8	7607.2*	6889-8325.4	< 0.001	13586	8304.8	5280.8*	4713.2-5848.3	< 0.001
day-2	DL	DL	DL	DL	DL	27385	13720	13666*	12734-14597	< 0.001
day-3	12366	6334.4	6031.3*	5399.5-6663.2	< 0.001	11044	6840.5	4203.8*	3551.1-4856.6	< 0.001
day-4	19190	9126	100064*	9569.9-10559	< 0.001	17931	10995	6936.5*	6423.2-7449.9	< 0.001
day-5	28468	7971.8	20496*	19015-21977	< 0.001	24854	7107.1	17747*	16516-18979	< 0.001
day-6	16618	4432.6	12186*	11552-12819	< 0.001	15640	6484.9	9155.3*	8540.4-9770.2	< 0.001
day-7	26020	2275.2	23744*	21136-26352	< 0.001	19126	3154.9	15971*	15059-16884	< 0.001

Further, Table 19 in APPENDIX-2 shows descriptive statistics for particle concentration on both the routes by each day of experiment. Minimum values remain similar for both the routes because of start/end point for both the routes is the same. However, values between first quartile and third quartile on polluted route do not go beyond 30000 (#/cm<sup>3</sup>). Figure 44 and Figure 45 in APPENDIX-2 shown temporal variation in concentration for polluted route and controlled route respectively, indicating stable concentration levels on controlled route in comparison to polluted route. Results shows characteristics of higher concentration on polluted route in comparison to controlled route based on the site type discussed in section-2.3 which is key requirement to assess the research objective set in section-1.4 and choice of route discussed in section-3.3 and section-3.4.



*Figure 17 Temporal variation in particle concentration between controlled (magenta-ap8) and polluted(turquoise=ap6) route*



*Figure 18 Temporal variation in particle concentration between controlled (red-ap9) and polluted(black=ap10) route*

### Cohort FeNO

For total 28 trips undertaken by 4 participants with equal number of trips on both the routes, Table 10 shows descriptive statistics for FeNO values of participants grouped by route. No instance of FeNO values post bicycling breaching the inflammation range or cautious inflammation range as discussed in section-3.8.1. For polluted route difference in mean value of 1 between before and after samples is not statistically significant at 95% confidence interval [-5.1043 to 7.1043, p=0.739]. Similarly, for controlled route difference in mean value of 2.7143 between before and after samples of size (n=14) is not statistically significant at 95% confidence interval [-5.5829 to 11.011, p=0.50]. Thus, for FeNO results in terms of cohorts there is no evidence of statistically higher inflammation as a result of undertaking bicycling on polluted route in comparison to controlled route. Also, since the sample size is less than 15 normality cannot be checked and can affect the result for t-test requiring the result to be treated with caution (Minitab, 2019c).

Table 10 Descriptive statistics for FeNO results of before/after values for participants by route

		FeNO (ppb)		
		Before	After	Change
Controlled Route	Mean	18.214	15.5	-2.714
	1st Quar	10	6.25	-3.75
	Median	17	15.5	-1.5
	3rd Quar	25	23.25	-1.75
	Minimum	3	1	-2
Polluted Route	Maximim	31	36	5
	Mean	20.571	19.571	-1
	1st Quar	18	13.75	-4.25
	Median	19.5	20	0.5
	3rd Quar	23.75	23.25	-0.5
	Minimum	6	4	-2
	Maximim	32	40	8

Figure 19 shows changes in FeNO parameter over the study period with participants on X-axis and FeNO values on Y-axis with measured value for each participant (grouped by route) is shown in the form of scatter point on which box plot is overlaid for each participant classified under panels indicating values before and after bicycling. For controlled route and polluted route for the duration of experiment there was no instance for any participant when FeNO values cross the mark of inflammation present (>50 ppb) based on limit values set in Table 8 of section-3.8.1. Again for polluted route and controlled route there were 3 and 1 instances respectively, when FeNO values cross the mark of cautious indication of airway inflammation (>25 to <50 ppb) set in Table 8 of section-3.8.1. It should also be noted participant-B with elevated FeNO values (>25 ppb) before bicycling in comparison to other participants in Figure 19 is expected as discussed in Figure 7 in section-2.5 that FeNO values for asthmatics are higher in comparison to healthy participants.

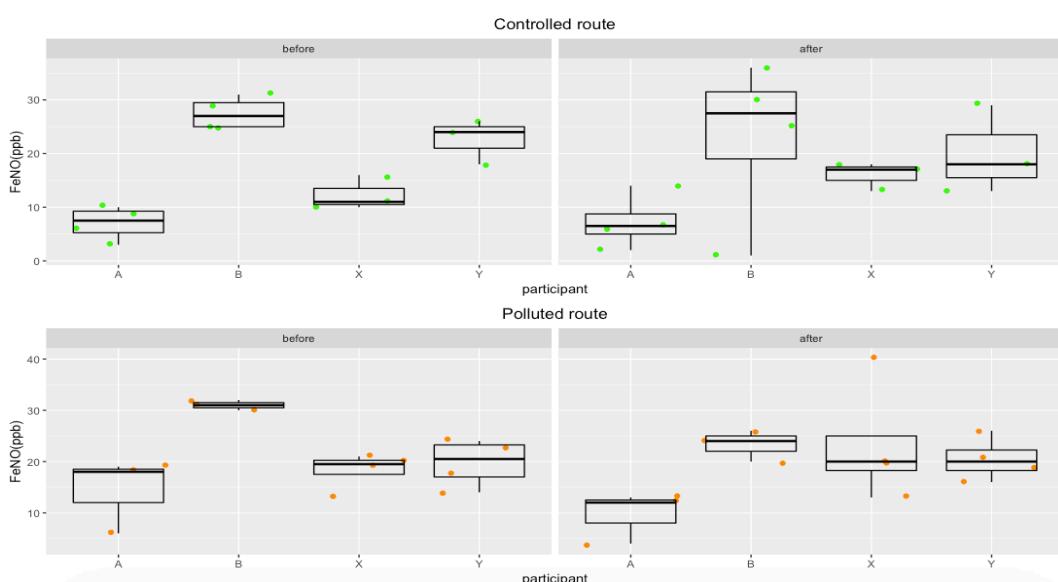


Figure 19 Participant FeNO results post bicycling by route

### Individual Participant FeNO Result

Table 11, Table 12, Table 13 and Table 14 indicates change in FeNO values post-bicycling for participant A, B, X and Y respectively. The same is represented graphically in Figure 46, Figure 47, Figure 48 and Figure 49 in APPENDIX-2.

For participant-A, FeNO values on polluted route always decreased for the given value of inhaled dose. On controlled route, results are mixed but show association with dosage. Thus, on controlled route dose above 0.0221 resulted into increase in FeNO. On polluted route, for maximum dose of 0.1294 there was no increase in FeNO

*Table 11 Participant-A FeNO results*

Day	Route	Inhaled dose (particles)	FeNO (ppb)		
			Before	After	Change
day-1	Controlled	0.0221	3	6	3
day-3		0.0193	6	2	-4
day-5		0.0233	10	14	4
day-6		0.0118	9	7	-2
day-2	Polluted	0.0951	6	4	-2
day-4		0.1209	19	12	-7
day-7		0.1294	18	13	-5

For participant-B, FeNO values on polluted route always decreased for given value of inhaled dose. On controlled route, FeNO values increased for all days except day-7 but dosage on day-7 was also minimum of all the days. Thus, on controlled route, dose above 0.0192 resulted into increase in FeNO. On polluted route, for maximum dose of 0.1249 there was no increase in FeNO.

*Table 12 Participant-B FeNO results*

Day	Route	Inhaled dose (particles)	FeNO (ppb)		
			Before	After	Change
day-1	Controlled	0.0431	31	36	5
day-4		0.0781	25	25	0
day-5		0.0483	25	30	5
day-7		0.0192	29	1	-28
day-2	Polluted	0.0951	32	24	-8
day-3		0.0698	30	20	-10
day-6		0.1249	31	26	-5

For participant-X, FeNO values on polluted route increased or remained same, with day-7 as exception but does not show direct association with dosage. On controlled route, FeNO values always increase for given dosage values. Thus, on controlled route dose value of 0.0039 and

above resulted into increased FeNO. On polluted route, a minimum dose of 0.0281 results in increased FeNO. Day-7 can be treated as an exception due to measuring error of  $\pm 5$  of instrument.

*Table 13 Participant-X FeNO results*

Day	Route	Inhaled dose (particles)	FeNO (ppb)		
			Before	After	Change
day-2	Controlled	0.0144	16	17	1
day-3		0.0039	11	18	7
day-6		0.0069	10	13	3
day-1	Polluted	0.0155	13	13	0
day-4		0.0281	20	40	20
day-5		0.031	19	20	1
day-7		0.0296	21	20	-1

For participant-Y, FeNO values on polluted route always increased, with an exception of day-3 which was also the lowest dose value on polluted route. On controlled route, FeNO values always reduced with day-4 as exception for which again the dose value was highest on polluted route. Thus, on controlled route, a minimum dose of 0.0448 resulted into increased FeNO. On polluted route, a minimum dose of 0.0286 resulted in increased FeNO.

*Table 14 Participant-Y FeNO results*

Day	Route	Inhaled dose (particles)	FeNO (ppb)		
			Before	After	Change
day-2	Controlled	0.0444	26	18	-8
day-4		0.0448	24	29	5
day-7		0.0127	30	1	-29
day-1	Polluted	0.0286	14	16	2
day-3		0.0257	23	19	-4
day-5		0.0941	24	26	2
day-6		0.0502	18	21	3

Thus, different participants have different cut-off dose beyond which inflammation in lung changes. When looked at above results irrespective of the routes, it can be argued dose value as a sole indicator for understanding lung inflammation is not robust to understand health impacts from TRAP.

### Cohort Spirometry

Of the total 28 trips taken by four participants change in spirometry parameters for participants grouped by route are shown in Table 15 in the form of descriptive statistics. For polluted route with sample size (n=41) the difference in mean value of -0.061707 between

before and after samples for FEV<sub>1</sub> is not statistically significant at 95% confidence interval [-0.24297 to 0.11956, p=0.5], mean difference of -0.061951 for FEV<sub>6</sub> is not statistically significant at 95% confidence interval [-0.28476 to 0.16086, p=0.581], mean difference of 0.006 for FEV<sub>1</sub>/FEV<sub>6</sub> is not statistically significant at 95% confidence interval [-0.0355 to 0.0233, p=0.681] and mean difference of 0.14537 for FEF is not statistically significant at 95% confidence interval [-0.57077 to 0.28004, p=0.498]. For controlled route, with sample size (n=42) between before and after samples the difference in mean value of -0.07619 for FEV<sub>1</sub> is not statistically significant at 95% confidence interval [0.24719 to 0.094811, p=0.278], mean difference of 0.052619 for FEV<sub>6</sub> is not statistically significant at 95% confidence interval [-0.19741 to 0.30265, p=0.677], mean difference of -0.023571 for FEV<sub>1</sub>/FEV<sub>6</sub> is not statistically significant at 95% confidence interval [-0.0505 to 0.00336, p=0.085] and mean difference of -0.18738 for FEF is not statistically significant at 95% confidence interval [-0.5418 to 0.16703, p=0.269].

*Table 15 Descriptive statistics for spirometry parameters post bicycling by route*

	FEV1 (L/s)			FEV6 (L/s)			FEV1/FEV6			FEF (L)			
	Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change	
Controlled Route	Mean	3.3919	3.4681	0.0762	4.4398	4.3871	-0.0527	0.7666	0.7902	0.0236	2.8252	3.0126	0.1874
	1st Quar	3.092	3.143	0.051	4.075	4.008	-0.067	0.71	0.75	0.04	2.355	2.522	0.167
	Median	3.355	3.575	0.22	4.235	4.235	0	0.765	0.78	0.015	2.62	2.665	0.045
	3rd Quar	3.78	3.775	-0.005	4.918	4.845	-0.073	0.8	0.82	0.02	2.805	2.947	0.142
	Minimum	2.36	2.69	0.33	3.32	3.51	0.19	0.68	0.68	0	1.69	2.11	0.42
	Maximum	3.94	4.02	0.08	5.58	5.67	0.09	0.9	0.89	-0.01	4.83	4.62	-0.21
Polluted Route	Mean	3.3885	3.4502	0.0617	4.271	4.3329	0.0619	0.797	0.8031	0.0061	3.0305	3.1759	0.1454
	1st Quar	3.092	3.143	0.051	4.075	4.008	-0.067	0.71	0.75	0.04	2.355	2.522	0.167
	Median	3.355	3.575	0.22	4.235	4.235	0	0.765	0.78	0.015	2.62	2.665	0.045
	3rd Quar	3.78	3.77	-0.01	4.918	4.845	-0.073	0.8	0.82	0.02	2.805	2.947	0.142
	Minimum	2.36	2.69	0.33	3.32	3.51	0.19	0.68	0.68	0	1.69	2.11	0.42
	Maximum	3.94	4.02	0.08	5.58	5.67	0.09	0.9	0.89	-0.01	4.83	4.62	-0.21

Table 16 shows change in spirometry parameters by day and route for the given amount of inhaled dose. For none of the participant on any of the day for any route ratio of FEV<sub>1</sub>/FEV<sub>6</sub> post bicycling reduces below cutoff value of 0.7 as discussed in section-3.8.1, indicating no volumetric impact on lung function. Three of the four reductions reported for FEV<sub>1</sub>/FEV<sub>6</sub> ratio are for participant-B who is asthmatic and inhaled higher dose in comparison for each run. Also, for participant-B reduction in FEV<sub>1</sub>/FEV<sub>6</sub> were higher on polluted route in comparison to controlled route. In terms of FEF, there is no cut-off value but interestingly whenever there is reduction in FEF there is a corresponding decrease in more than one spirometry parameter. This hints that start point of primary impact from TRAP is on smaller airways (reduced FEF) resulting into overall lung volume function (FEV<sub>1</sub>, FEV<sub>6</sub> or FEV<sub>1</sub>/FEV<sub>6</sub>) also evidenced by Marseglia et al., (2007).

Table 16 Change in spirometry parameters for participants by day

Participant	Day	Route	Inhaled dose (count)	FEV1 (L/s)			FEV6 (L/s)			FEV1/FEV6			FEF (L)		
				Before	After	Change	Before	After	Change	Before	After	Change	Before	After	Change
A	day1	controlled	0.022139	3.727	3.953	0.226	5.42	5.557	0.137	0.6867	0.71	0.0233	2.553	2.81	0.257
B		controlled	0.043119	3.14	3.127	-0.013	3.943	3.947	0.004	0.7967	0.7933	-0.0034	2.633	2.63	-0.003
X		polluted	0.015479	3.06	3.885	0.825	3.495	4.27	0.775	0.87	0.88	0.01	3.515	4.695	1.18
Y		polluted	0.028608	3.11	3.303	0.193	4.007	4.16	0.153	0.78	0.7933	0.0133	2.383	2.677	0.294
A	day2	polluted	0.095077	3.733	3.873	0.14	5.203	5.207	0.004	0.7167	0.7467	0.03	2.597	2.827	0.23
B		polluted	0.196561	3.177	3.287	0.11	3.947	4.137	0.19	0.8067	0.7967	-0.01	2.693	2.8	0.107
X		controlled	0.011411	3.79	3.827	0.037	4.43	4.363	-0.067	0.8567	0.88	0.0233	4.117	4.507	0.39
Y		controlled	0.044371	3.21	3.23	0.02	4.16	4.12	-0.04	0.77	0.7867	0.0167	2.403	2.5	0.097
A	day3	controlled	0.019266	3.853	3.76	-0.093	5.45	5.1	-0.35	0.7067	0.7367	0.03	2.73	2.74	0.01
B		polluted	0.069814	3.157	3.05	-0.107	3.977	3.94	-0.037	0.7933	0.7733	-0.02	2.617	2.467	-0.15
X		controlled	0.003945	3.76	3.783	0.023	4.353	4.323	-0.03	0.8633	0.8767	0.0134	4.21	4.48	0.27
Y		polluted	0.025692	3.19	3.157	-0.033	4.127	3.997	-0.13	0.7733	0.79	0.0167	2.467	2.517	0.05
A	day4	polluted	0.083856	3.903	3.82	-0.083	5.35	5.237	-0.113	0.73	0.73	0	2.833	2.757	-0.076
B		controlled	0.078076	2.673	3.017	0.344	3.623	3.803	0.18	0.7367	0.7967	0.06	1.997	2.52	0.523
X		polluted	0.028139	3.833	3.777	-0.056	4.297	4.15	-0.147	0.8933	0.9133	0.02	4.747	4.91	0.163
Y		controlled	0.044754	3.23	3.503	0.273	4.157	4.16	0.003	0.7767	0.7767	0	2.467	2.477	0.01
A	day5	controlled	0.023257	3.773	3.647	-0.126	5.247	5.06	-0.187	0.72	0.72	0	2.647	2.577	-0.07
B		controlled	0.048309	2.867	2.873	0.006	3.85	3.713	-0.137	0.7433	0.7767	0.0334	2.17	2.3	0.13
X		polluted	0.030989	3.743	3.77	0.027	4.183	4.213	0.03	0.8967	0.8967	0	4.577	4.647	0.07
Y		polluted	0.09415	3.233	3.423	0.19	4.137	4.327	0.19	0.78	0.7933	0.0133	2.463	2.793	0.33
A	day6	controlled	0.011817	3.533	3.687	0.154	4.977	4.937	-0.04	0.7067	0.7467	0.04	2.483	2.757	0.274
B		polluted	0.124852	2.527	2.773	0.246	3.7	3.883	0.183	0.6933	0.7133	0.02	1.75	1.94	0.19
X		controlled	0.006875	3.803	3.763	-0.04	4.313	4.247	-0.066	0.8833	0.8867	0.0034	4.52	4.483	-0.037
Y		polluted	0.050221	3.293	2.923	-0.37	3.987	4.15	0.163	0.8267	0.7833	-0.0434	2.957	2.513	-0.444
A	day7	polluted	0.089762	3.687	3.693	0.006	5.013	4.827	-0.186	0.7367	0.7633	0.0266	2.653	2.797	0.144
B		controlled	0.019197	2.78	3.123	0.343	4.033	4.137	0.104	0.69	0.7533	0.0633	1.877	2.447	0.57
X		polluted	0.029603	3.683	3.713	0.03	4.163	4.143	-0.02	0.8867	0.8967	0.01	4.337	4.63	0.293
Y		controlled	0.012666	3.347	3.26	-0.087	4.2	3.953	-0.247	0.7967	0.8233	0.0266	2.747	2.95	0.203

Among the participants performance of spirometry parameters is weakest for participant-B who also underwent higher dose (Table 16). Figure 20 shows changes in spirometry parameters on polluted route for the study period with participants on X-axis and spirometry parameter on Y-axis and where measured value of each participant is shown in the form of scatter point, on which box plot is overlaid for each participant classified under panels indicating values before and after bicycling was undertaken. Similarly, Figure 21 represents for controlled route. Both these figures corroborate that recorded as well as change in spirometry parameters for asthmatic participant-B are an exception with larger inter quartile range and variance in comparison to other participants.

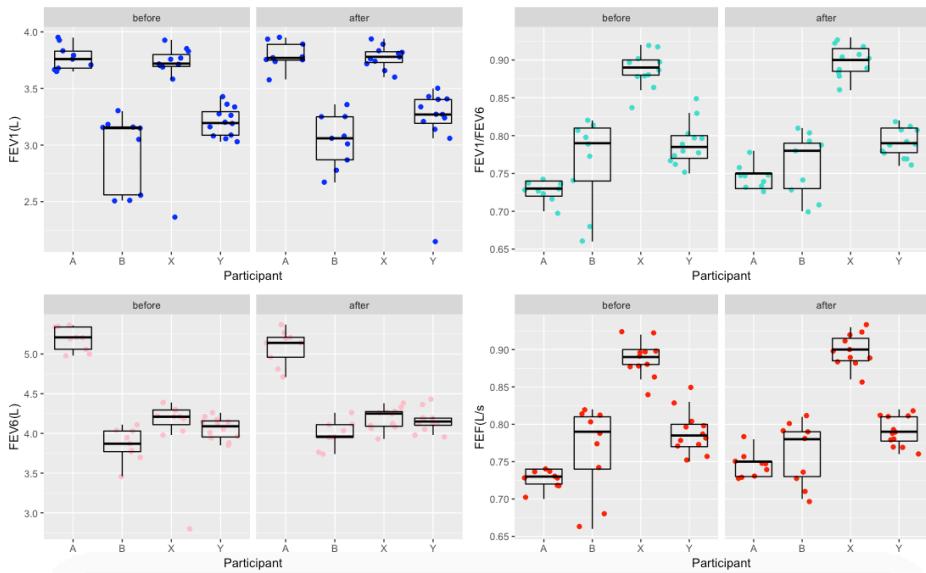


Figure 20 Polluted route cohort spirometry

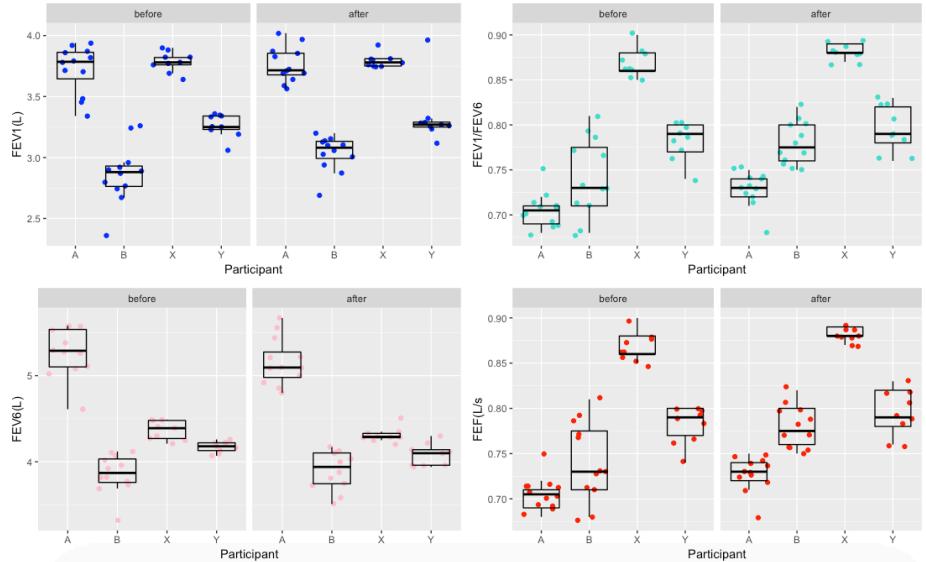


Figure 21 Controlled route cohort spirometry

Thus, from above spirometry results in terms of cohorts by route suggests no statistically significant difference in lung function in terms of spirometry parameters from undertaking bicycling on polluted route in comparison to controlled route. Also, there is no evident trend of route specific deterioration neither it was possible to establish a cutoff value beyond which there was always a deterioration.

#### Individual participant spirometry

Figure 22, Figure 23, Figure 24 and Figure 25 shows change in spirometry parameters for each day by route for participant-A, participant-B participant-X and participant-Y respectively. X-

axis represents the days and before/after spirometry parameter is indicated on Y-axis with average value of three readings taken are plotted as points with error bar (adjusted to Y-axis) for individual days.

Participant-A FEV<sub>1</sub> and FEV<sub>6</sub> performance post bicycling degraded over the duration of experiment. From Figure 22, FEV<sub>1</sub> and FEV<sub>6</sub> values after bicycling show decline over the duration of experiment. FEV<sub>1</sub>/FEV<sub>6</sub> values after bicycling continue to increase for the duration of experiment while there is no discernible trend for FEF. While for individual day performance of all spirometry parameters improves post cycling.

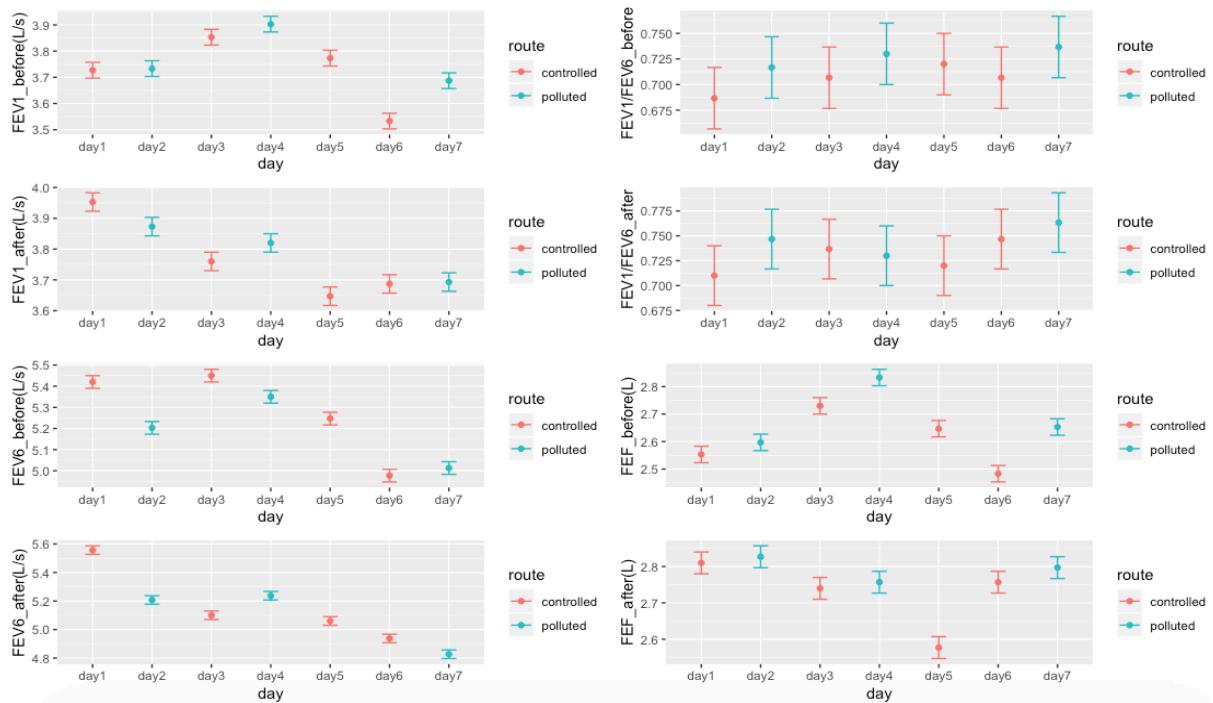


Figure 22 Participant-A spirometry parameter change post bicycling

Participant-B FEV<sub>1</sub>/FEV<sub>6</sub> and FEF performance post bicycling degraded over the duration of experiment. From Figure 23, FEV<sub>1</sub>/FEV<sub>6</sub> and FEF values post bicycling decline for the duration of experiment. FEV<sub>1</sub> and FEV<sub>6</sub> show decline for first six days. While individual day performance of all spirometry parameters improved post bicycling.

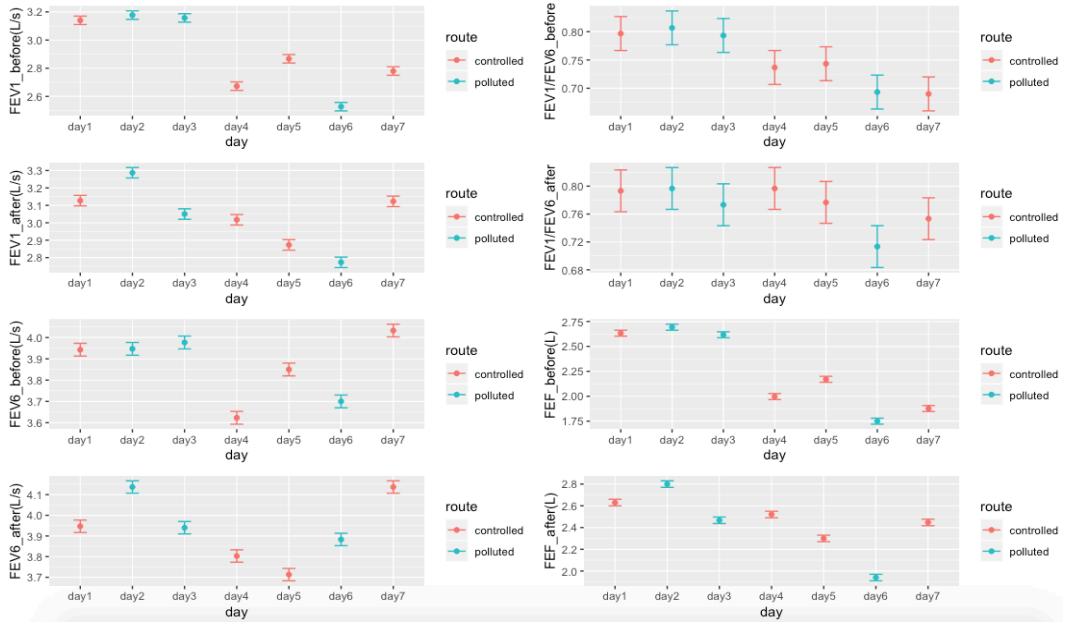


Figure 23 Participant-B spirometry parameter change post bicycling

For participant-X day-1 spirometry value will be excluded from analysis as data was not recorded correctly (Figure 16). From Figure 24, FEV<sub>1</sub> and FEV<sub>6</sub> values post bicycling decline over the duration of experiment. FEV<sub>1</sub>/FEV<sub>6</sub> and FEF values after bicycling show increasing trend over the duration of experiment. While for individual day performance of all spirometry parameters improved post cycling.

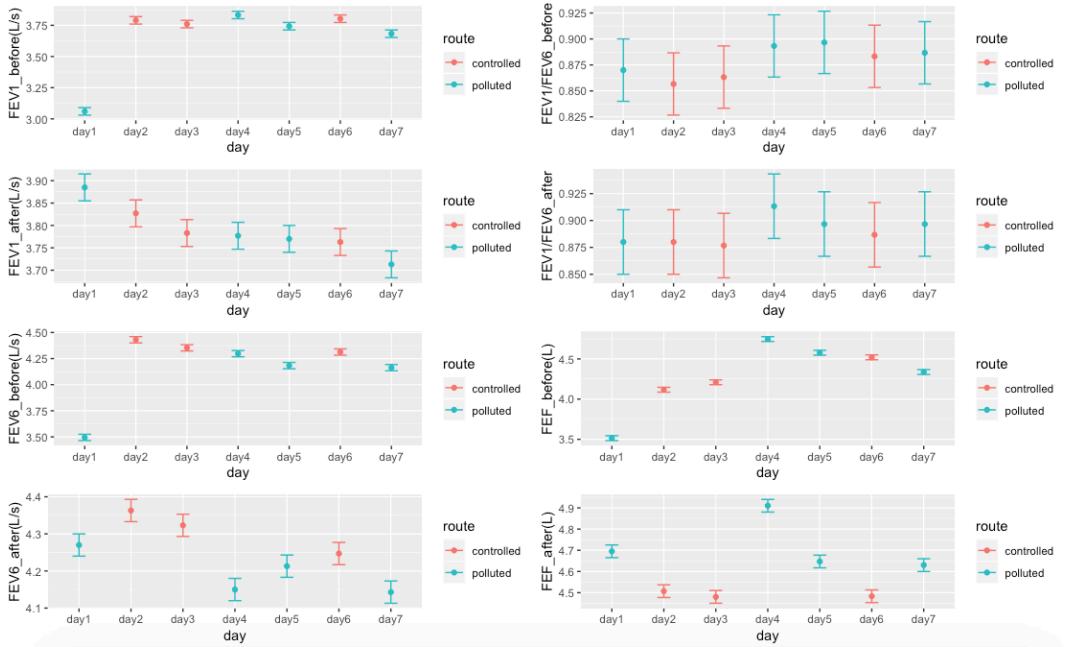


Figure 24 Participant-X spirometry parameter change post bicycling

Participant-Y spirometry parameter performance post bicycling is volatile over the duration of experiment. From Figure 25, FEV<sub>1</sub> and FEV<sub>6</sub> performance post bicycling degraded over the

duration of experiment. FEV<sub>1</sub>/FEV<sub>6</sub> shows minor change except for day-7 while FEF shows increasing trend post bicycling for the duration of experiment.

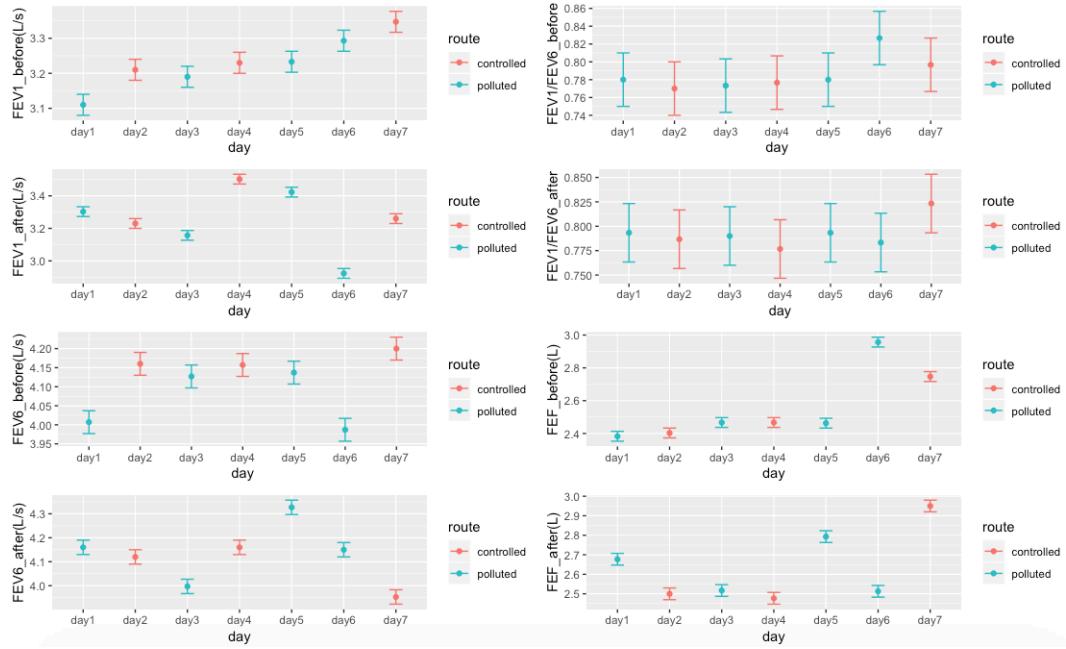


Figure 25 Participant-Y spirometry parameter change post bicycling

### Integrated analysis

From the given results and Table 17 there is no indication of relationship between simultaneous degradation in volumetric and inflammatory lung function. In Table 17 on day-3, except for participant-X there was reduction in FEV<sub>1</sub> and FEV<sub>6</sub> for all participants with simultaneous degraded FeNO. For participant-X, while FeNO degrades, only reduction in FEV<sub>6</sub> was noted. For participant-B, in Table 17, day-1 results show simultaneous degradation in FeNO and spirometry parameters, while on day-4 when inhaled dose was higher than day-1 resulted in improved spirometry and no change in FeNO. For participant-A, in Table 17, day-2 results show simultaneous improvement in FeNO and spirometry parameters, while on day-4 with higher dosage than day-2 all spirometry parameters degrade while FeNO improves. For participant-Y, in Table 17 day-1 results show improvement in spirometry parameters while FeNO degraded, while on day-3 with reduced inhaled dose spirometry parameter degraded and FeNO improved.

Day	Participant	Route	Start time (hh:mm:ss)	End time (hh:mm:ss)	Journey time (mins)	Inhaled dose (count)	Std dev	FEV1 (L/s)	FEV6 (L/s)	FEV1/FEV6	FEF (L)	FeNO (ppb)
day-1	A	controlled				0.0221	0.0086	3.727	3.953	5.42	5.557	0.68667
	B	controlled	08:38:49	09:31:49	53	0.0431	0.0220	3.14	3.127	3.943	3.947	0.7967
	X	polluted				0.0155	0.0099	3.06	3.885	3.495	4.27	0.87
	Y	polluted				0.0286	0.0095	3.11	3.303	4.007	4.16	0.78
day-2	A	polluted				0.0951	0.0811	3.733	3.873	5.203	5.207	0.7167
	B	polluted	08:24:04	09:21:56	58	0.0951	0.0811	3.177	3.287	3.947	4.137	0.8067
	X	controlled				0.0114	0.0035	3.79	3.827	4.43	4.363	0.8567
	Y	controlled				0.0444	0.0185	3.21	3.23	4.16	4.12	0.77
day-3	A	controlled				0.0193	0.0075	3.853	3.76	5.45	5.1	0.7067
	B	controlled	08:25:43	09:21:13	56	0.0698	0.0203	3.157	3.05	3.977	3.94	0.7933
	X	controlled				0.0039	0.0013	3.76	3.783	4.353	4.323	0.8633
	Y	controlled				0.0287	0.0086	3.19	3.157	4.127	3.997	0.7733
day-4	A	polluted				0.1209	0.0721	3.903	3.82	5.35	5.237	0.73
	B	controlled	08:23:41	09:36:58	73	0.0781	0.0399	2.673	3.017	3.623	3.803	0.7367
	X	polluted				0.0281	0.0179	3.833	3.777	4.297	4.15	0.8933
	Y	controlled				0.0448	0.0187	3.23	3.503	4.157	4.16	0.7767
day-5	A	controlled				0.0233	0.0091	3.773	3.647	5.247	5.06	0.72
	B	controlled	08:17:04	09:15:15	58	0.0483	0.0247	2.867	2.873	3.85	3.773	0.7433
	X	polluted				0.0310	0.0197	3.743	3.77	4.183	4.213	0.8967
	Y	polluted				0.0941	0.0337	3.233	3.423	4.137	4.327	0.78
day-6	A	controlled				0.0118	0.0046	3.533	3.687	4.977	4.937	0.7067
	B	polluted	08:15:56	09:08:33	53	0.1249	0.0462	2.527	2.773	3.7	3.883	0.6933
	X	controlled				0.0069	0.0021	3.803	3.763	4.313	4.247	0.8833
	Y	polluted				0.0592	0.0180	3.293	2.923	3.987	4.15	0.8267
day-7	A	polluted				0.1294	0.0772	3.687	3.693	5.013	4.827	0.7367
	B	controlled	08:13:55	09:25:59	72	0.0192	0.0098	2.78	3.123	4.033	4.137	0.69
	X	polluted				0.0296	0.0189	3.683	3.713	4.163	4.143	0.8867
Y	controlled					0.0127	0.0053	3.347	3.26	4.2	3.953	0.7967
												0.8233

 Spirometry reduced  
 Spirometry increased  
 FeNO reduced or no change  
 FeNO increased

Table 17 Change in spirometry, FeNO and inhaled dose for participant

## 4.3 Discussion

### FeNO

On the polluted route, the evidence of inflammation was not conclusive post bicycling, and no discernible trend were observed for any of the participants. A similar study by Gong et al., (2008) for exposure to UFP and PA discussed in section-2.5 also showed similar results. However, this was in contrast to Kubesch et al., (2015) who reported statistically increased inflammation from exposure to coarse PM and bicycling. Difference in results along with results of polluted route of this study indicate increased UFP concentration does not necessarily result in increased inflammation, toxicity in the composition of the particle might be the primary trigger for health impact rather than a simplistic exposure response function based on different particle concentration at sites (Lagorio et al., 2006). For example, an interesting result of participant-X FeNO value suddenly increases post bicycling with no significant change of dose in comparison to other days can be as a result of increased toxicity from higher amount of iron or zinc hinted by Lagorio et al., (2006) and discussed in section-2.5. This cannot be empirically supported as CPC did not have capability to differentiate measured particle fraction.

Further, asthmatic participant-B results show inflammation on polluted route also went down which is contrary to the evidence presented by Barraza-Villarreal Albino et al., (2008) and others discussed in section-2.5. A possible explanation of which could be bronchoconstriction effect affecting airway flux and diffusing capacity of lungs are affected from exposure (Cattoni et al., 2013) and discussed in section-2.5. This decreases the reliability of FeNO results measured under bronchoconstriction effect as FeNO measurement is flow dependent.

For all participants, instances of increased inflammation on controlled route within the no-inflammation range could not be explained. While instances of reduction supports evidence of reduced lung inflammation from undertaking bicycling in less polluted environment (Pedroletti et al., 2009) as discussed in section-2.5. On day-2 and day-7 FeNO values reduce for all the participants irrespective of the routes and it was raining on both the days (Table 6). Change in FeNO on controlled route was significant for both the participants on day-7 when it was more rainy in comparison to day-2, this supports evidence of rain scavenging the

particles from the atmosphere by Ravindra et al., (2003) and Sharma et al., (1983) discussed in section 2.3. The key difference of relative humidity levels in comparison to other days which result in hygroscopic change post emission from tailpipe prior to inhalation which would have not happened in absence of rain as discussed in Section-2.4. Thus, relative humidity levels affecting particle size prior to inhalation influenced inflammation levels for the participants for the duration of experiment.

FeNO as an indicator of lung inflammation is not limited to a single flow rate (50 mL/s) which was used for this study, but different flow rates indicate inflammation in different parts of lungs (Bazeghi et al., 2011). Also, evidence on elevated lung inflammation biomarkers have been associated with air pollution (Delfino Ralph J. et al., 2008) but has not been consistently positive for short term (Hoffmann Barbara et al., 2009). This could be as a result of underlying influence on these biomarkers from underlying conditions like obesity, hypertension and diabetes which are associated with elevated mean inflammatory markers (Dubowsky Sara D. et al., 2006). Also, change in lung inflammation indicated by lower FeNO though not significant to indicate lung inflammation as a result of short term exercise and can vary between individuals who exercise regularly versus individuals who don't and this was the case for participants of this study (Table 5) (Kasapis and Thompson, 2005).

It is methodically difficult to eliminate effects of air pollution from other stressors, like diet and infections and requires larger sample size of participants which increases reliability of statistical test and isolate outliers (Annesi-Maesano and Dinh-Xuan, 2016). For this study with small number of participants information like diet, alcohol consumption etcetera were only recorded in data collection sheet (Figure 34) and considered during analysis.

### Spirometry

No short term effect on spirometry parameters is in contrast to the existing evidence discussed in section-2.5 that shows no change including for asthmatic participants, in a similar study by McCreanor et al., (2007) with walking where degradation in spirometry parameters were observed, but levels of pollution on polluted route were more than double median values than polluted route on this study suggesting the defense mechanisms in the linings of lung as discussed by Kelly, (2003) reducing the negative impact from harmful species of particles or lack of exposure to species of pollution like metallic fraction of iron and zinc as

hinted by Lagorio et al., (2006). Yet, if we refer to daywise trend of after values for FEV<sub>1</sub> for participant A, B and Y we see FEV<sub>1</sub> reduces everyday irrespective of improvement/degradation from the before value on the same day suggesting there is a possibility of underlying impact but unable to conclude due to small sample size. Similar is the case for participant-X excluding day-1 from the analysis when participant-X was unable to perform spirometry maneuver for three times correctly. Weak statistically significant change from lung impact for short term as evidenced by Bell et al., (2013) who used effect modifiers like age, sex and race to understand lung function impact and found age to be a significant modifier stating very young and old people are more susceptible to short term effects from exposure to PM than young individuals and participants of these study being young adults (Table 5) could be a possible reason for no change.

At a gap of 8 weeks post experiment completion I recorded spirometry parameters for participant-B in the morning over a duration of two hours of sample size equivalent to the experiment data for spirometry combining before and after readings. I found increase of mean difference of 0.305 for FEV<sub>1</sub> among the sample statistically significant at 95% confidence interval [0.221 to 0.289, p<0.001], increased mean difference of 0.2431 for FEV<sub>6</sub> is statistically significant at 95% confidence interval [0.1625 to 0.3236, p<0.001], increased mean difference of 0.0314 for FEV<sub>1</sub>/FEV<sub>6</sub> is statistically significant at 95% confidence interval [0.0154 to 0.0473, p<0.001] and increased mean difference of 0.3497 for FEF is statistically significant at 95% confidence interval [0.228 to 0.4715, p<0.001]. This further corroborates the evidence of spirometry parameters FEV<sub>1</sub>, FEV<sub>1</sub>/FEV<sub>6</sub> and FEF reducing over the experiment days as shown in Figure 21. Unfortunately, this was not done for other participants. This also makes the case for establishing baseline readings before and after the experiment period to establish the impact more robustly for following reasons. First, any effect from bronchoconstriction will be reduced from measurements (Cattoni et al., 2013). Second, any increased pulmonary function from exercise can be appropriately considered in analysis (Fairbarn et al., 1991).

### Contribution

The strength of this research is in its design where comparison between polluted and controlled routes and measuring impact on lung function based on real time dosage values has not been undertaken to my knowledge, Kubesch et al., (2015) study was fairly similar but

did not account the increased VE from bicycling for their dosage calculation and this study adds more evidence to their findings . Results indicate short term lung function is not directly dependent on the dosage of UFP but hints the toxicity or makeup of particles is of importance (Lagorio et al., 2006). Further, results showing no inflammation and spirometry parameters for undertaking activity on polluted route where hourly average concentration levels for PM<sub>2.5</sub> were meeting target of 25 µg/m<sup>3</sup> hourly mean (Table 20 in APPENDIX-2) evidence the validity of proposed air quality guidelines by WHO, (2016b).

Further, I have following lessons learned conducting this research that will help guide future studies and produce better results. First, choosing a different route which is more polluted (eg: passing through city centre) or conduct data collection during PM peak for same route. Second, FeNO and spirometry parameters should also be measured at a lag of few hours also pre/post bicycling to understand the change after muscles are relaxed post exercise. Third, few weeks recording of baseline FeNO and spirometry parameters for involved participants prior to data collection which can be used to compare with experimental findings later. Fourth, if possible use of electric cabs for transportation to avoid any prior contamination. Lastly, it is also beneficial to measure relative humidity.

### Limitation

First limitation of the study is that it was limited to male participants however it is highly recommended that similar study including female participants is undertaken to check similarity of results. Second, one of the key suspect for the lung function impact to be negative is lower concentration of pollution on polluted route based on recorded data from AURN as shown in Figure 50 and Figure 51 in APPENDIX-2. Indicating june had very low level of PM concentration in comparison to prior months of year 2019, PM<sub>2.5</sub> and PM<sub>10</sub> concentration during the AM peak on weekdays were again lower in comparison to PM peak. Daily PM<sub>10</sub> and PM<sub>2.5</sub> average values for study hours is shown in Table 20 in APPENDIX-2 which were meeting air quality guidelines discussed in Table 2. It is not the case that route is not polluted as it can be seen pollution levels were high at the start of the year and were continuously decreasing until June (Figure 50 and Figure 51). Further, I only had access to the track in the morning and we see that pollution levels are higher during the PM peak which could have influenced the result from higher exposure. Third, we were not able to differentiate what fraction of particle

were product of primary combustion or not. Fourth, as discussed in section-2.3 how dispersion of pollutants is affected by meteorological parameters like wind speed and wind direction. From Figure 26 the wind direction was westerly dominated which reduced the dosage of participants at least for one leg of the round trip and same was the case for north eastern wind which was channeling pollution in opposite direction of cyclists for one leg of journey reducing the dosage further and influencing results from lowered dosage.

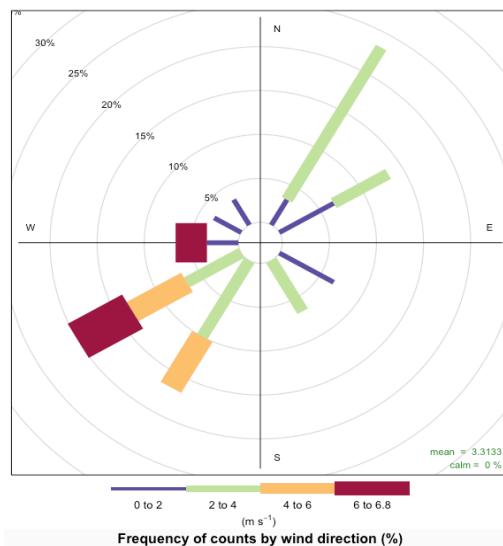


Figure 26 Windrose plot for the study duration (DEFRA, 2019b)

Lastly, performance of sweetzpot device was very poor which restricted us to limit the analysis with average dosed value of particle and not report and compare cumulative dosage with biomarkers of lung function impact and provide a better quantification. Real time VE and concentration values measured together used to calculate total dose indicate how cumulative dose varies substantially for same participant in different exposure route environment as shown for participant-X in Figure 27 and Figure 28. Increasing the uncertainty of the results from studies such as by de Nazelle et al., (2012) using reference VE from published handbooks like of EPA's as discussed in section-2.4.

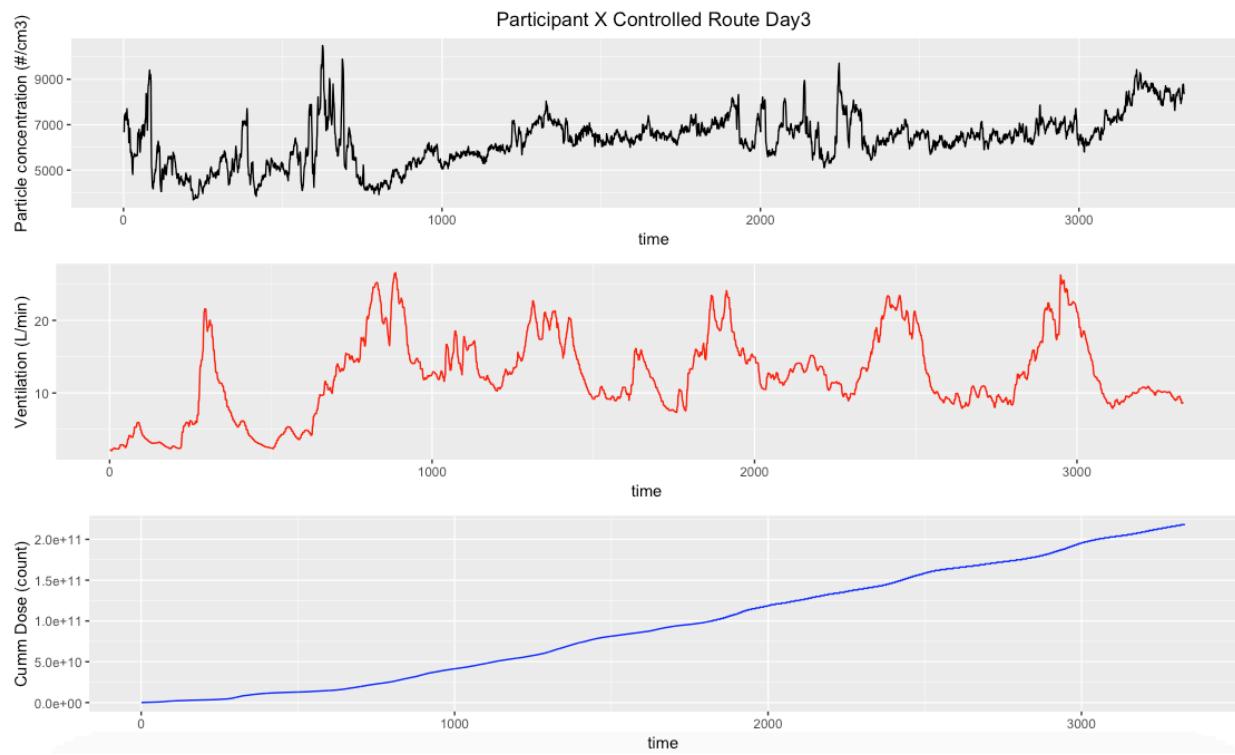


Figure 27 Particle concentration, VE and cumulative dose for participant-X on controlled route on day-3

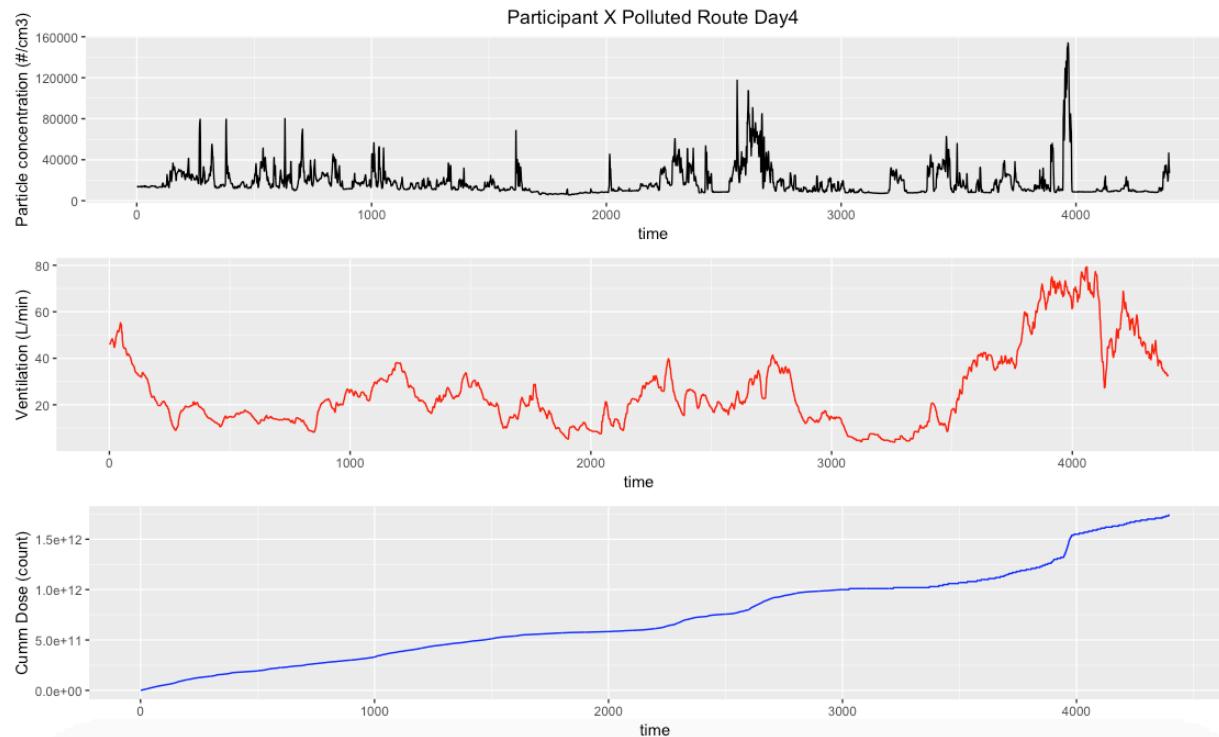


Figure 28 Particle concentration, VE and cumulative dose for participant-X on polluted route on day-4

## 5 Conclusion and further research

This thesis aimed to answer three research questions in section-1.4 found there was no statistically significant impact on lung volumetric function or airway inflammation or combined impact on lung function from undertaking bicycling on polluted versus controlled route as discussed in section-4. Results show, in terms of volumetric impact although there was no impact, overall volumetric impact ( $FEV_1$  and  $FEV_6$ ) always degraded with small airway degradation ( $FEF$ ) also evidenced by Marseglia et al., (2007). In terms of airway inflammation, a certain fraction of chemical composition plays a vital role as evidenced by Lagorio et al., (2006) rather than being proportional to concentration. In terms of combinatory impact there was no consistent evidence neither any interesting observations.

I highly recommend a similar study involving more participants, different busy traffic hours and routes to add further evidence to the findings of this study with methodological improvements proposed in section-4.3. Also, impact from other pollutants like  $NO_x$  and  $O_3$  should be undertaken with larger sample size of participants to better understand health impact from different pollutants. This study presents an opportunity to further explore a new strand of research in health impacts from TRAP with its novel design and recommendations. With new evidence from this strand of research along with other studies will serve as robust evidence for active travel policy inputs to abate negative health impact from improved air quality.

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## 7 Appendices

### 7.1 APPENDIX-1

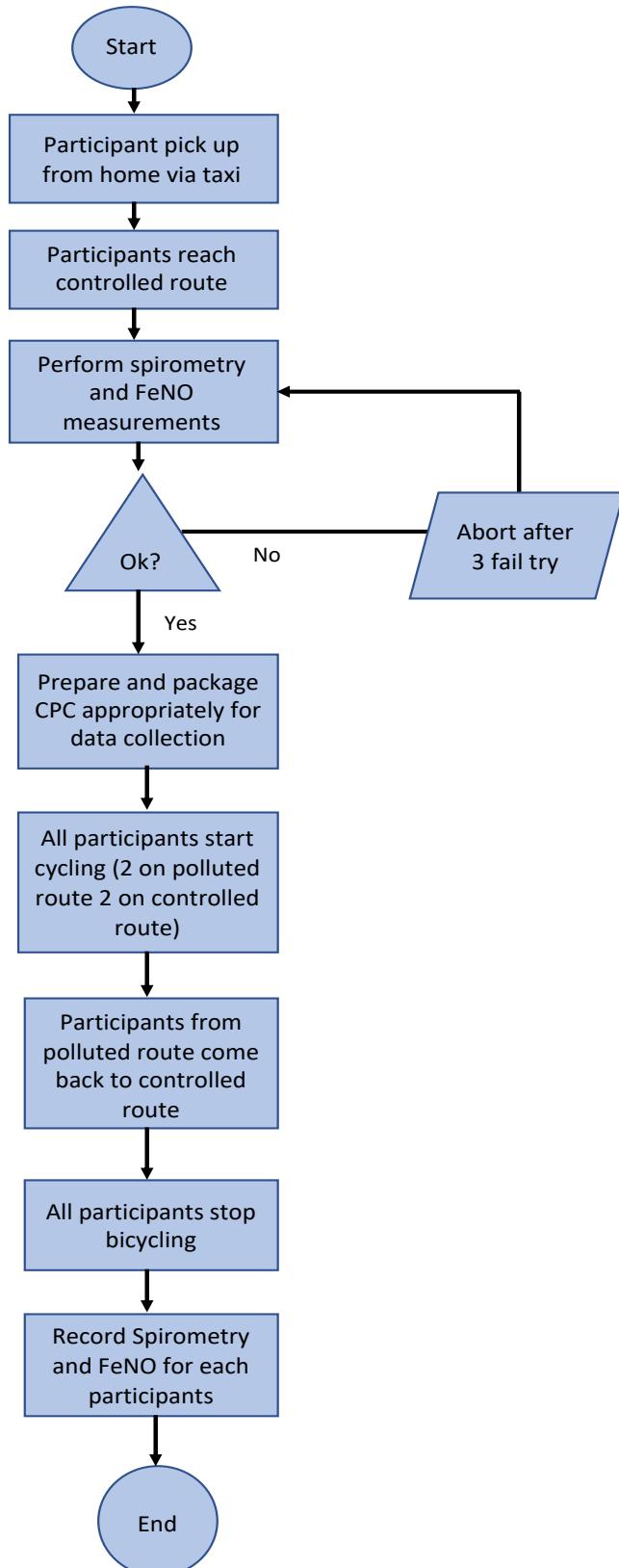


Figure 29 Step by step guidance of data collection for a given day

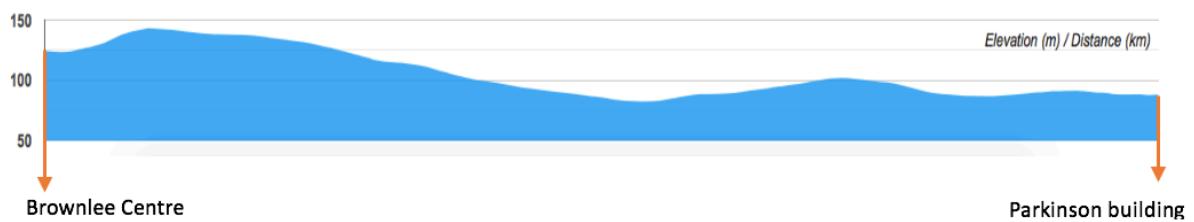


Figure 30 Gradient profile from Brownlee centre to Parkinson building (BikeRoll, 2019)



Figure 31 Ariel view of Controlled route(GoogleEarth, 2018)

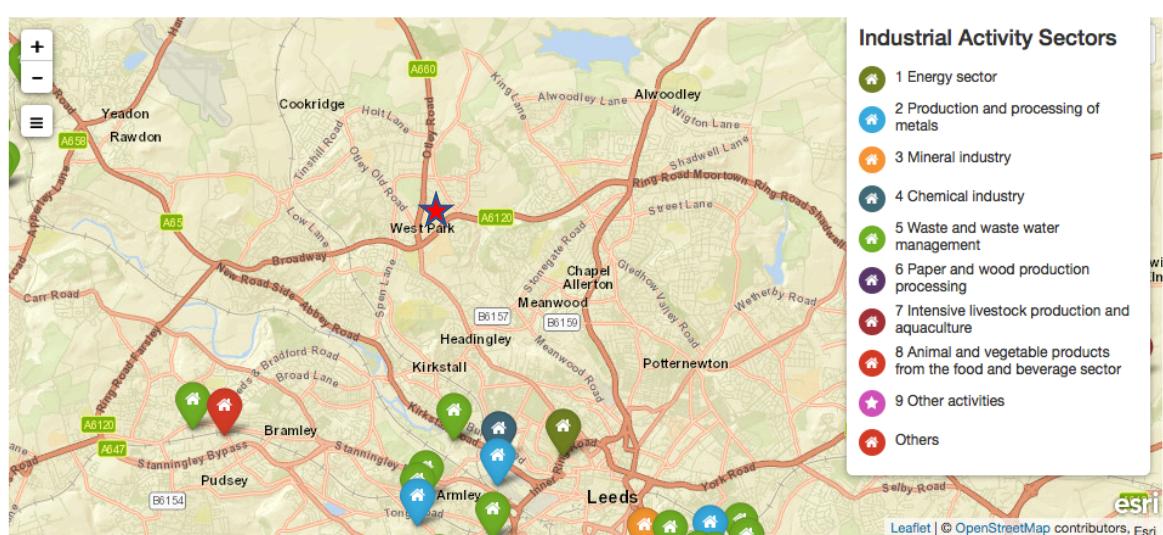


Figure 32 Point emission sources around controlled route (red star= controlled route location) (EPRTR, 2017)

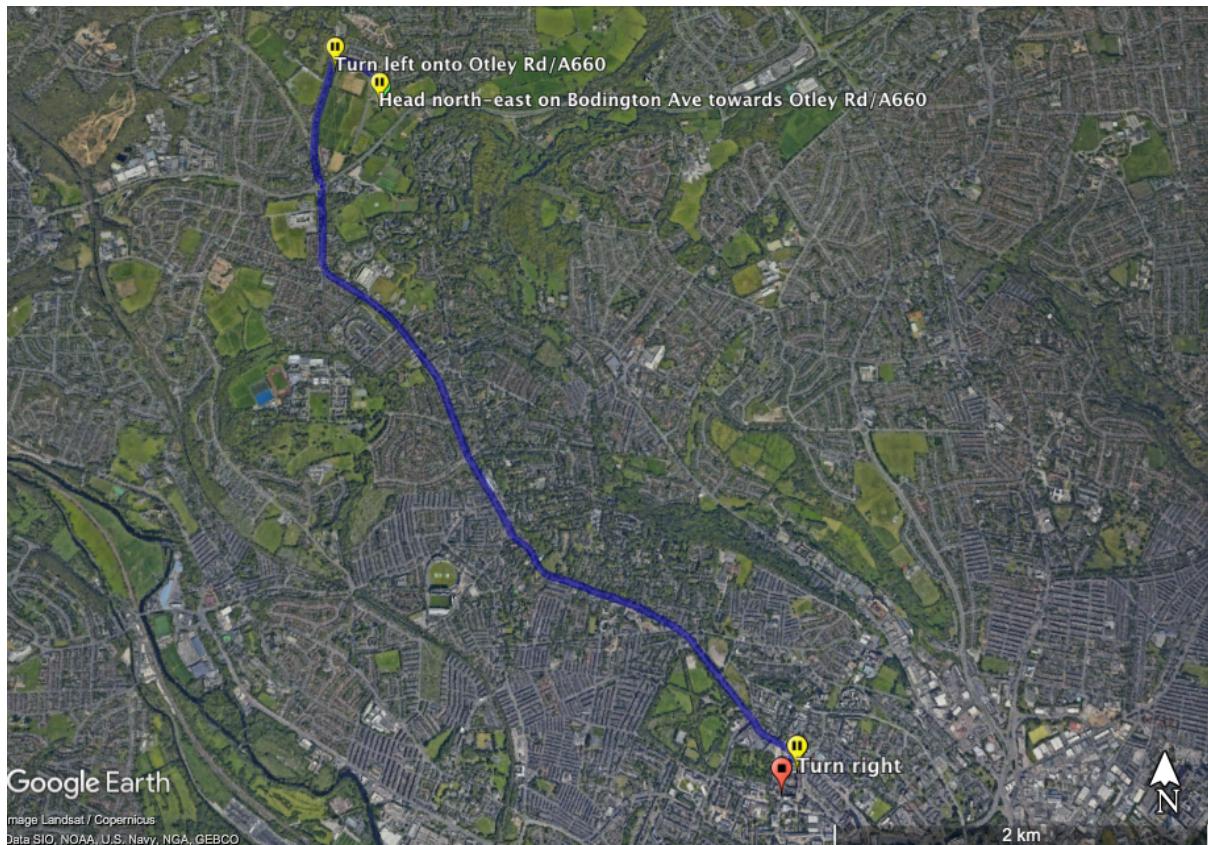


Figure 33 Ariel view of polluted route (GoogleEarth, 2018)

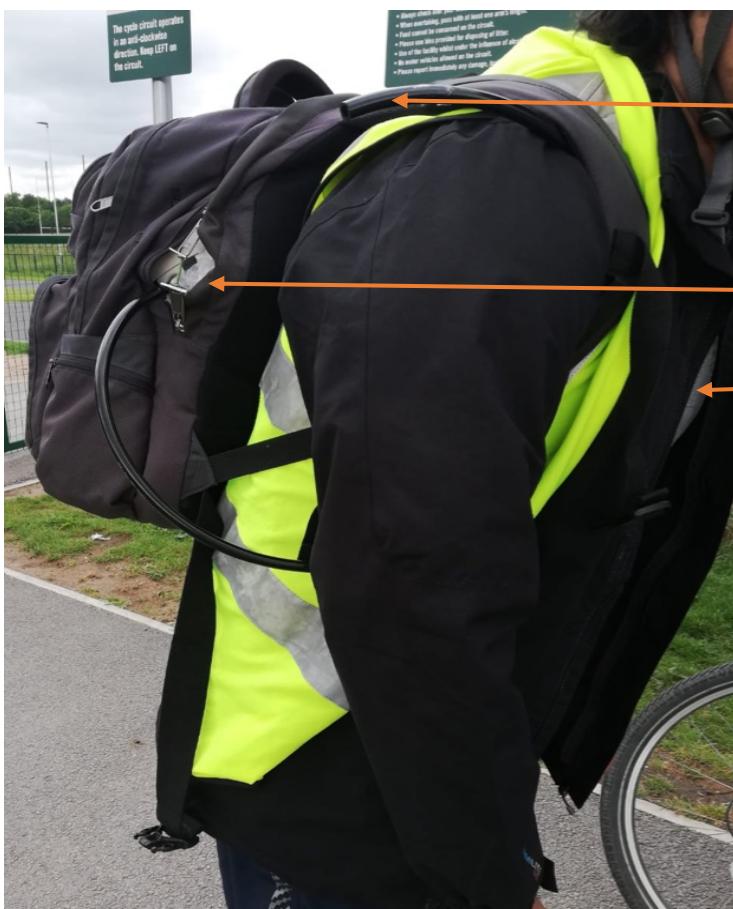
Air Quality Data Collection Sheet							
Date	Participant	Route		Bicycle			
		Start Time		End Time			
Spirometry	Before	FEV1				Sweetzpot	
		FEV6				Noise Badge	
		FEV1/FEV6				Speedometer	
		FEF				CPC	
						Mobile Phone	
	After	FEV1					
		FEV6					
		FEV1/FEV6					
		FEF					
NOBreath	Before	FeNO					
	After	FeNO					
Comments	Dietary						
	Any prior contamination						
	Observations from Data Collection						
	Post Data Collection Observation						

Figure 34 Data collection sheet



Rucksack mounted on participant

Figure 35 Participant with the kit back view (Fonseca, 2019a)



PTFE tube at shoulder level connected to CPC

CPC in rucksack

Sweetspot location (not visible)

Figure 36 Participant with key side view (Fonseca, 2019b)

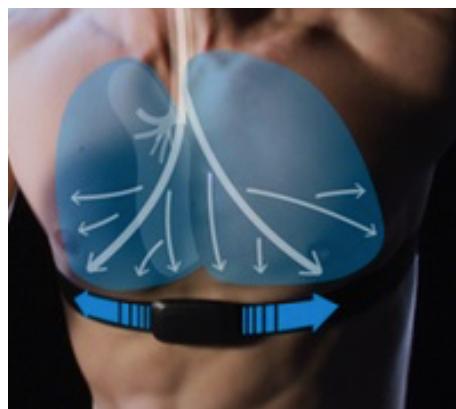


Figure 37 Sweetspot in strapped condition via elastic harness (Fisher, 2018)



Figure 38 Cycle for participant-A (Khatri, 2019a)



Figure 39 Cycle for participant-B (Khatri, 2019a)



Figure 40 Cycle for participant-X (Khatri, 2019a)



Figure 41 Cycle for participant-Y (Khatri, 2019a)

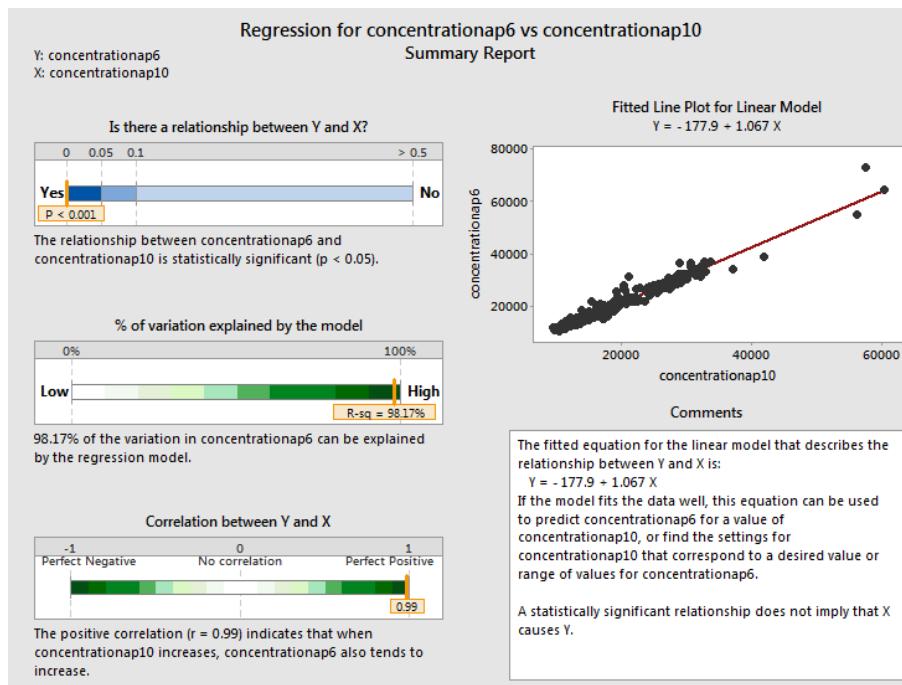


Figure 42 Regression for ap6 and ap10 (Minitab, 2019a)

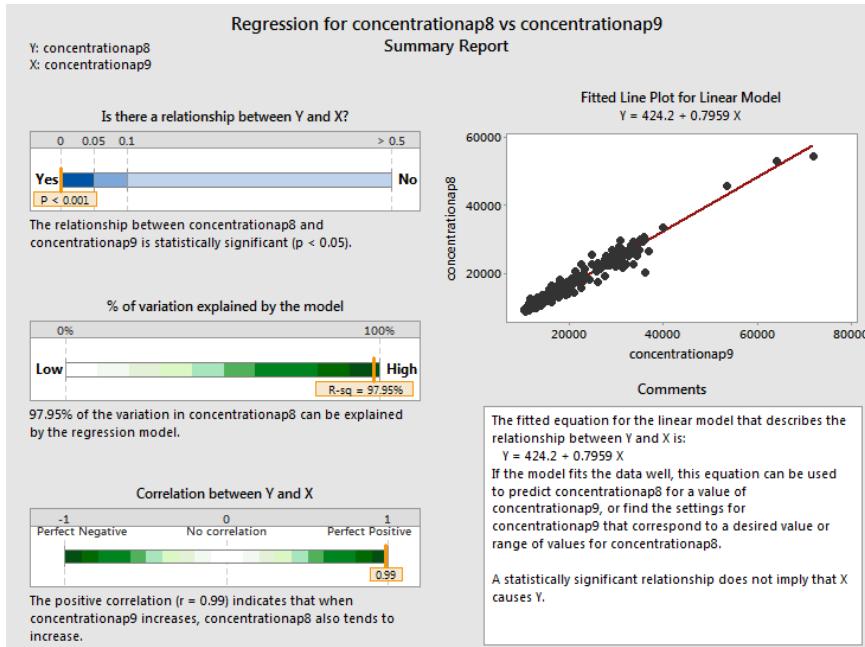


Figure 43 Regression ap8 and ap9 (Minitab, 2019a)

## 7.2 APPENDIX-2

Participant	Day	Route	Start time	End time	Journey time	Avg ventilation rate	Std Dev vent rate	Avg particle concentration	Std Dev particle concentration	Std Dev Particle	Inhaled air (m³)	Std dev Inhaled air (m³)	Inhaled pollutant (count/km)	Std Dev Inhaled pollutant (count/km)	Inhaled dose (count)	Std Dev Inhaled dose (count)
			(hh:mm:ss)	(hh:mm:ss)	(mins)	(litre/min)	(#/m³)	(#/m³)	(#/m³)	(count)	(count)	(count)	(count)	(count)	(count)	
A	controlled		08:38:49	09:31:49	53	60.6	23.6	0.0083	0.0037	3.2118	1.2508	0.0267	0.0104	0.00187	0.0221	
	B	day-1	08:24:04	09:21:56	58	141.2	72.1	0.0069	0.0011	7.4836	3.8213	0.0620	0.0265	0.00364	0.0431	
	X	polluted				25.9	16.5	0.0136	0.0132	1.3727	0.8745	0.0186	0.0119	0.00131	0.0220	
	Y	polluted				44.7	14.9	0.0145	0.0173	2.3691	0.7897	0.0345	0.0115	0.00242	0.0099	
A	polluted					72.12	61.5	0.0274	0.0234	4.18296	3.567	0.11455	0.0977	0.00803	0.0951	
	B	polluted				149.1	65.4	0.0274	0.0234	8.6478	3.7932	0.2368	0.1039	0.01660	0.1966	
	X	day-2	08:25:43	09:21:13	56	24.1	7.3	0.0098	0.0021	1.3978	0.4234	0.0137	0.0042	0.00096	0.0114	
	Y	controlled				119.16	34.8	0.0137	0.0026	6.91128	2.0184	0.0948	0.0277	0.00665	0.0787	
A	controlled					60.6	23.6	0.0068	0.0014	3.3936	1.3216	0.0232	0.0090	0.00163	0.0193	
	B	polluted				130.6	38	0.0115	0.0109	7.3136	2.128	0.0841	0.0245	0.00590	0.0698	
	X	controlled				13.4	4.4	0.0063	0.0011	0.7504	0.2464	0.0048	0.0016	0.00033	0.0013	
	Y	polluted				44.7	14.9	0.0124	0.0156	2.5052	0.8344	0.0310	0.0103	0.00217	0.0230	
A	polluted					72.12	61.5	0.0192	0.0128	5.26476	4.4895	0.1010	0.0862	0.00708	0.0839	
	B	controlled				141.2	72.1	0.0091	0.0019	10.3076	5.2633	0.0941	0.0480	0.00659	0.0203	
	X	polluted				25.9	16.5	0.0179	0.0144	1.8907	1.2045	0.0339	0.0216	0.00238	0.0281	
	Y	controlled				119.16	34.8	0.0110	0.0026	8.89888	2.5404	0.0956	0.0279	0.00670	0.0794	
A	controlled					60.6	23.6	0.0080	0.0025	3.5148	1.3688	0.0280	0.0109	0.00196	0.0233	
	B	controlled				141.2	72.1	0.0071	0.0026	8.1896	4.1818	0.0582	0.0297	0.00408	0.0483	
	X	polluted				25.9	16.5	0.0249	0.0312	1.5022	0.957	0.0373	0.0238	0.00262	0.0310	
	Y	polluted				68.7	24.6	0.0285	0.0364	3.9846	1.4288	0.1134	0.0406	0.00795	0.0941	
A	controlled					60.6	23.6	0.0044	0.0004	3.2118	1.2508	0.0142	0.0055	0.00100	0.0118	
	B	polluted				181.47	67.2	0.0156	0.0142	9.61791	3.5616	0.1504	0.0557	0.01054	0.1249	
	X	controlled				24.1	7.3	0.0065	0.0031	1.2773	0.3869	0.0083	0.0025	0.00058	0.0069	
	Y	polluted				68.7	24.6	0.0166	0.0151	3.6411	1.3038	0.0605	0.0217	0.00424	0.0502	
A	polluted					72.12	61.5	0.0208	0.0304	5.19264	4.428	0.1081	0.0922	0.00758	0.0898	
	B	controlled				141.2	72.1	0.0023	0.0257	10.1664	5.1912	0.0231	0.0118	0.00162	0.0192	
	X	polluted				25.9	16.5	0.0191	0.0017	1.8648	1.188	0.0357	0.0227	0.00250	0.0296	
	Y	controlled				119.16	34.8	0.0032	0.0019	8.57952	2.5056	0.0271	0.0079	0.00190	0.0225	

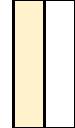
 values from other days for same participant on respective route or proxy run  
 captured data

Table 18 Pollution dosage results for each participant by day and route (430)

Table 19 Descriptive statistics for particle concentration for study duration (DL= Data Loss) (218)

Route	Day	CPC	Mean (#/cm3)	1st Quar (#/cm3)	Median (#/cm3)	3rd Quar (#/cm3)	Min (#/cm3)	Max (#/cm3)
Polluted Route	day1	ap6	14549	8693	9951	13613	2944	240682
		ap10	13586	8366	9593	13056	5039	182239
	day2	ap6	DL	DL	DL	DL	DL	DL
		ap10	27385	14989	20834	29967	9101	321502
	day3	ap6	12366	5659	7288	11604	3561	164959
		ap10	11501	5867	7499	12299	3739	118458
	day4	ap6	19190	10685	15285	23184	5425	138502
		ap10	17931	9520	13518	20557	5579	153901
	day5	ap6	28468	12087	16245	27347	7837	308235
		ap10	24854	11150	14746	24203	7220	323351
	day6	ap6	16618	9038	12111	18545	5031	192507
		ap10	15640	8547	11335	17529	4861	207731
	day7	ap6	20827	5566	10946	22642	1771	296259
		ap10	19126	5522	10255	22702	1202	321324
Controlled route	day1	ap8	6941.8	6215	6628	7510	4674	10933
		ap9	8305	6726	7363	8604	3657	45316
	day2	ap8	9836	8220	9473	10836	6328	17167
		ap9	13720	12317	13270	15009	9052	27479
	day3	ap8	6334	5668	6383	6951	3691	10483
		ap9	6840	5755	7148	7980	2307	10548
	day4	ap8	9126	7888	8521	9999	5875	19305
		ap9	10995	9215	10554	12116	6930	48457
	day5	ap8	7972	7025	7764	8155	6156	47069
		ap9	7107	4921	7190	8476	1820	24850
	day6	ap8	4433	4018	4443	4752	3612	5882
		ap9	6485	5157	5842	6835	1458	45860
	day7	ap8	2275	1016	2060	2635	798	14445
		ap9	3155	1574	2860	3967	966	14643

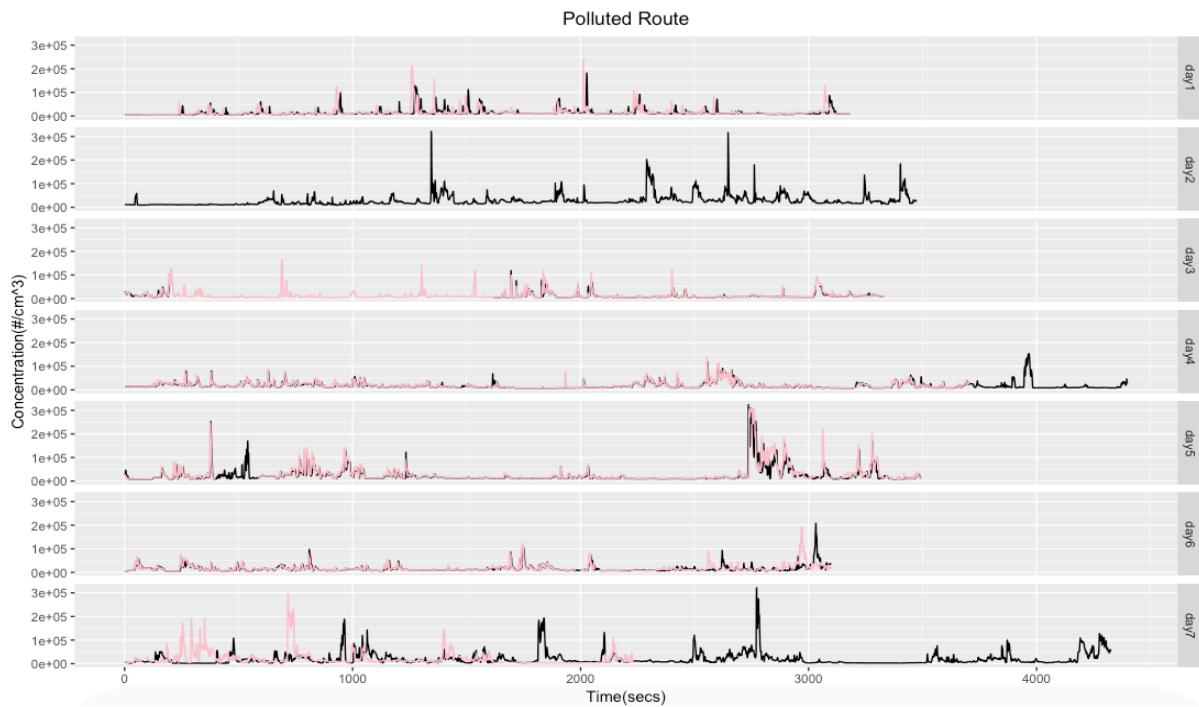


Figure 44 Polluted route concentration (ap6: pink and ap10: black)

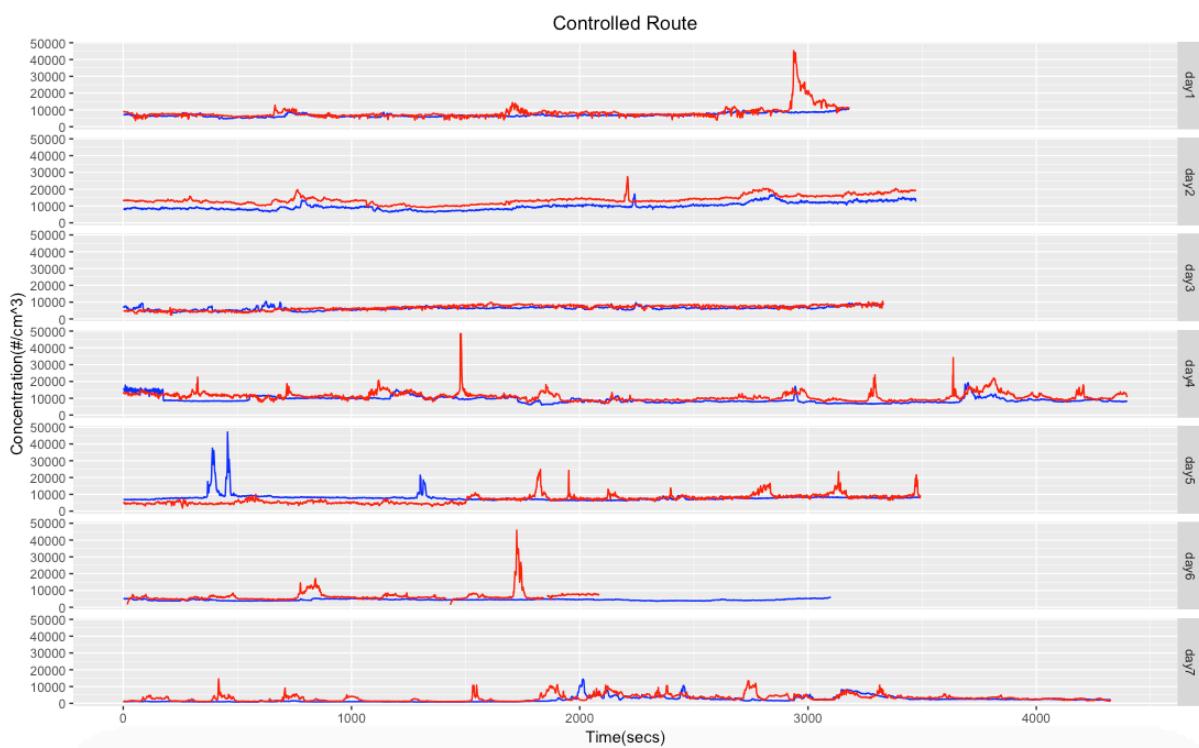


Figure 45 Controlled route concentration (ap8: blue and ap9: red)

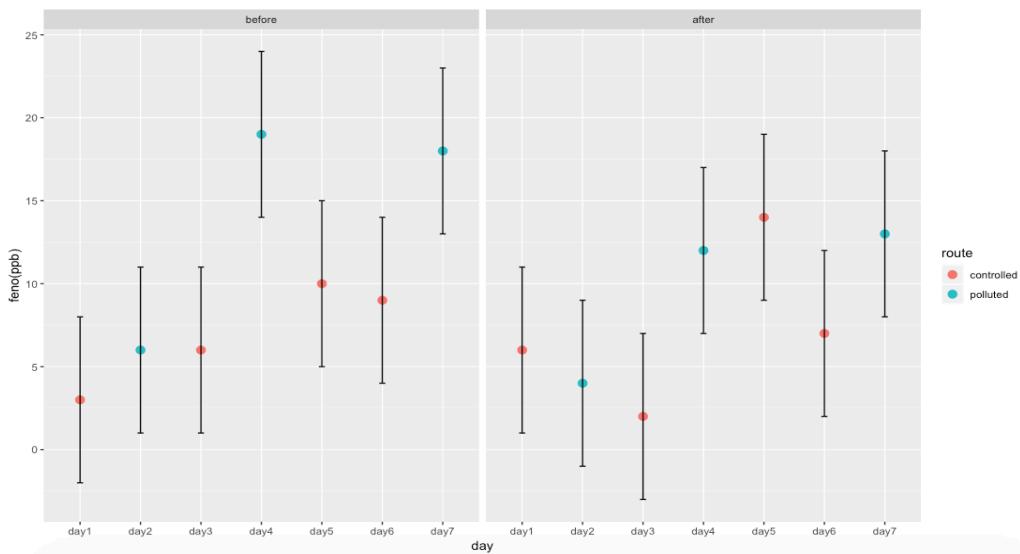


Figure 46 Change in participant-A FeNO post bicycling

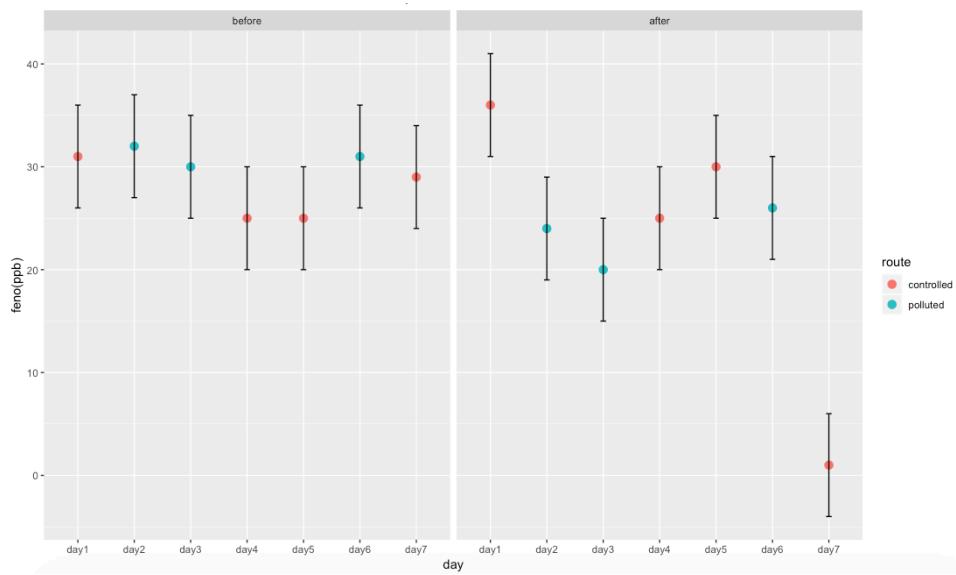


Figure 47 Change in participant-B FeNO post bicycling

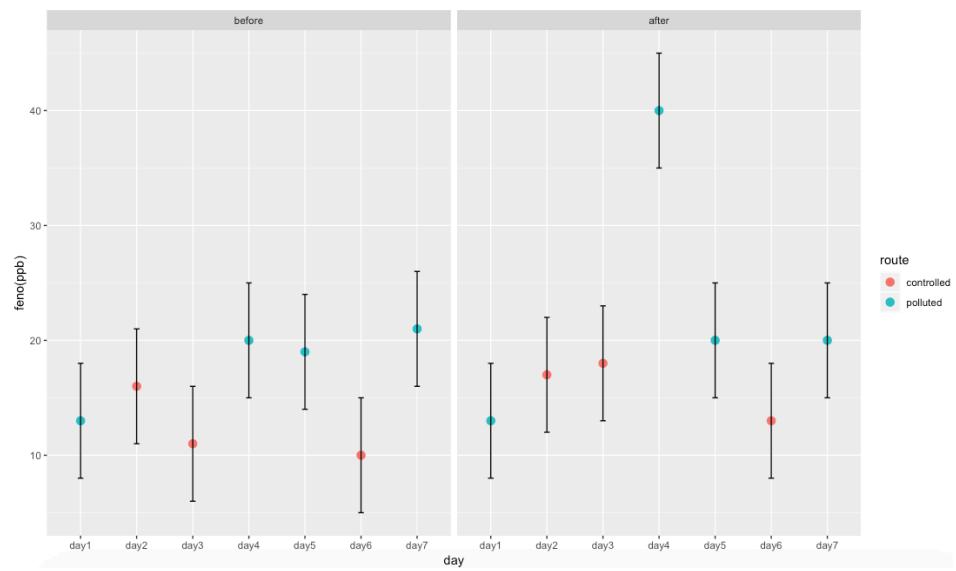


Figure 48 Change in participant-X FeNO post bicycling

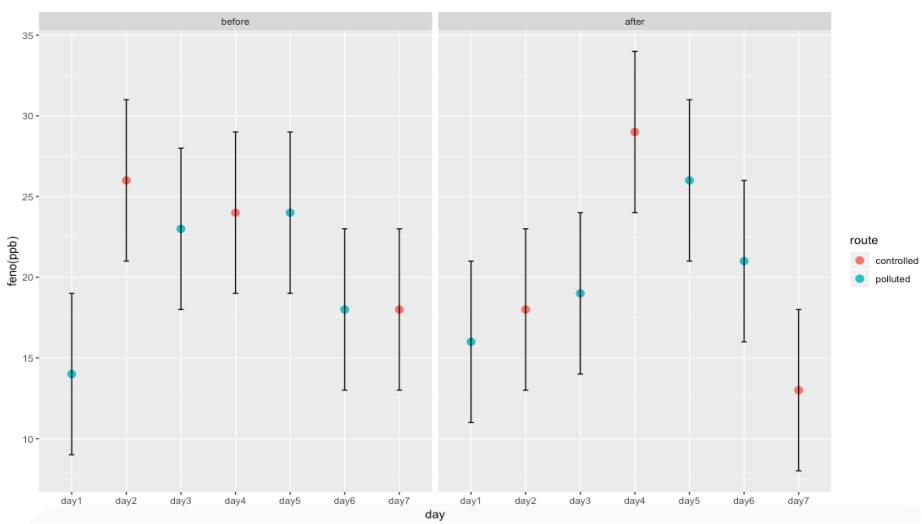


Figure 49 Change in Participant-Y FeNO post bicycling

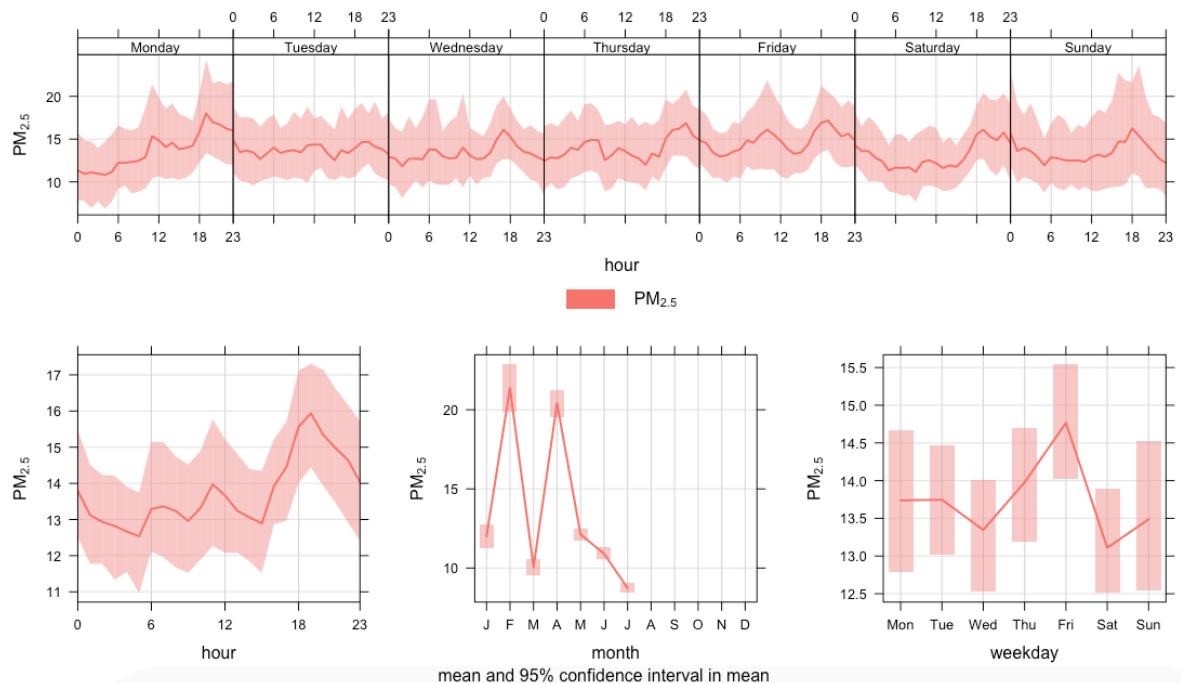
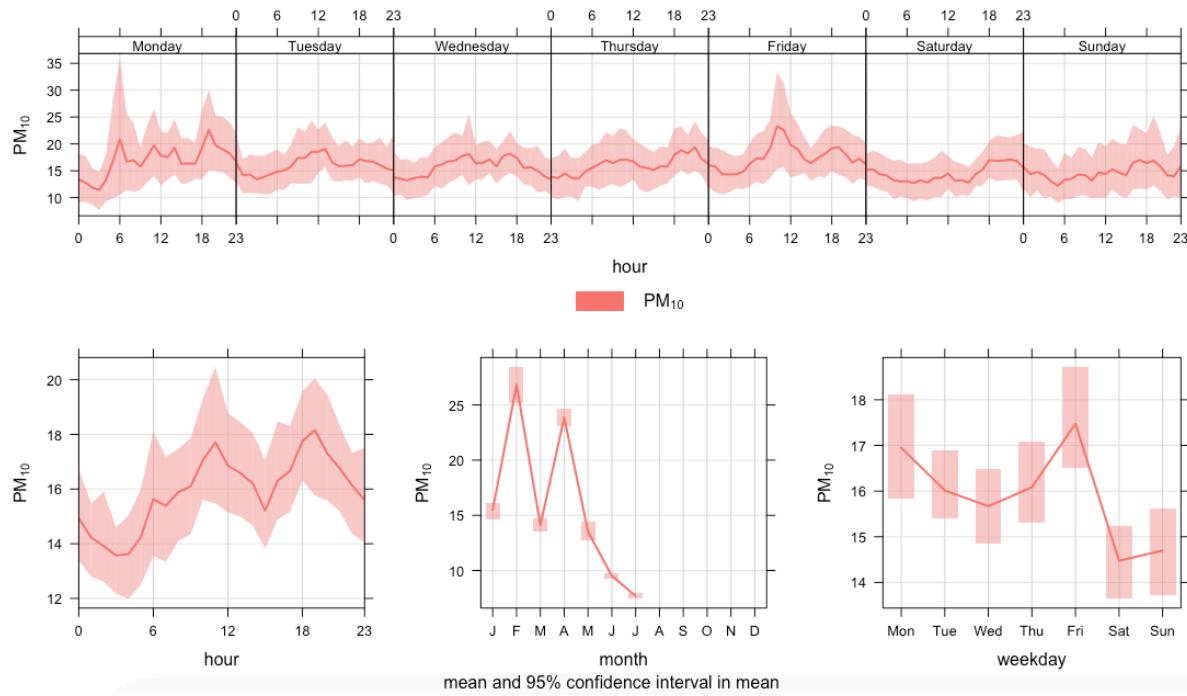


Figure 50 Headingley Kerbside AURN Timevariation plot data for PM<sub>2.5</sub> grouped by time of day across weekdays, week, month and hours with 95% confidence interval range (DEFRA, 2019b)



*Figure 51 Headingley Kerbside AURN Timevariation plot data for PM<sub>10</sub> grouped by time of day across weekdays, week, month and hours with 95% confidence interval range (DEFRA, 2019b)*

*Table 20 Average PM<sub>2.5</sub> and PM<sub>10</sub> concentration for 06:00 to 10:00 AM for study days (DEFRA, 2019b)*

	PM2.5 ( $\mu\text{g}/\text{m}^3$ )	PM10 ( $\mu\text{g}/\text{m}^3$ )
day-1	6.16	6.88
day-2	10.86	10.58
day-3	5.25	5.62
day-4	9.84	7.92
day-5	15.02	13.1
day-6	8.22	7.86
day-7	7.56	5.7