APPLICATION OF MERSEYSIDE COMMUNITY CHILD HEALTH SURVEY DATA FOR ESTIMATING HEALTH RISKS FROM AIR POLLUTION AND EXPOSURE TO CIGARETTE SMOKE DURING PREGNANCY

By

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DEDICATION

to

All my dearest family members for being with me and for their constant support, assistance and encouragement throughout the work.

My friends and colleagues who gave me lots of support to carry out this work and for encouragement.

Most of all I thank God Almighty for helping me bring this work to a successful completion.

In memory

of

GARY MAHONEY

24.6.1958 – 10.11.2014

I am deeply sorry to mention the death of my project team member, Dr Gary Mahoney, from the Environmental Protection Department, Sefton Council, who gave us all the support. Gary was a very dedicated professional, whose outstanding and innovative work has left a lasting legacy. Gary played a leading role in the Sefton council bid to achieve Beacon Authority status, which led the Sefton Council receiving national recognition. He represented Merseyside at Regional Air Quality Group meetings nationally. He suggested the possibility of conducting an analysis of the community child health data after linking with the air pollution data to look at the health outcomes in relation to air pollution in Sefton area. He was such a dedicated and nice friend to me and quite enthusiastic about this work and project. I will be ever grateful for his assistance, and I am sorry that he has not lived to see me complete this work. Gary's memory will be with us always.

“The woods are lovely, dark, and deep,

But I have promises to keep,

And miles to go before I sleep,

And miles to go before I sleep”.

- ***Robert Frost***

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THESIS STRUCTURE

Chapter one outlines the introduction and background, rationale, study aims and objectives. **Chapter two** presents a review of literature which includes the details of air pollution and related health consequences and combined air pollution and cigarette smoke exposure during pregnancy. **Chapter three** describes the survey methodology, study sample, sample size, data collection, dataset linkage, analysis and statistical methods. **Chapter four** outlines study results, with sections on spatial mapping and Venn diagram representation. **Chapter five** is the discussion section which compares observations with results from other studies followed by **Chapter six** which presents the conclusions, future research implications, limitations, and recommendations.

Statement of involvement in the analysis of the 2006 Merseyside Respiratory Health Survey and air pollution data and differences between MPhil and PhD

* This MPhil project reports a descriptive cross-sectional study based on a new dataset formed by linking two different related datasets using post code sectors: Child health data with extracted relevant variables from a survey of 10 Liverpool schools conducted in 2006 and the corresponding air pollution data for the same area and same period collected by Sefton council.

* The MPhil candidate visited the Merseyside Atmospheric Emissions Inventory (MAEI) on a regular basis to understand the methodologies and processes of the working system for deriving the emissions for oxides of nitrogen (NO_X) and particulate matter (size <10mm) (PM_{10}), for measuring nitrogen dioxide (NO_2) and PM_{10} concentrations, and to learn more about the working air pollution data needed to create the final MPhil dataset.

*He also had to visit NHS Sefton and Sefton council on several occasions to get special permission to access the air pollution data and to learn more about the birth and child health outcome data available in the study area over the relevant time period. He also attended seminars and symposiums to learn more about the air quality assessments and air quality issues in the Sefton area and held meetings with the different teams monitoring air pollution and climatic changes in the study area.

* Ultimately, after several meetings and a considerable amount of time, permission was obtained from the local authority and Sefton council to access the relevant air pollution statistics, but not in time to address the issue of possible statistical linkages between important health outcomes and air pollution levels as part of the submitted PhD dissertation. Nevertheless, this was an essential issue requiring study as air pollution in the dock area near to the study area where the schools were located was a well-recognised problem. When the air pollution data subsequently became available, this new project was needed and was carried out as the MPhil project. Air pollution data in the form of emissions for NO_X and PM_{10} and concentrations data for NO_2 and PM_{10} were collected from Sefton council; these were based on data produced from the Merseyside Atmospheric Emissions Inventory based in Sefton.

*The candidate performed a series of completely new statistical analyses, the results of which are reported and discussed in this MPhil thesis. This MPhil thus extends and complements the work reported in the previous PhD, and is separate from that previous work, examining completely new and novel research questions relating to a possible statistical analytical correlation between the health outcome measures and concomitant air pollution levels that could not be addressed in the PhD due to the lack of data as outlined above.

*To emphasize, the PhD project included the pregnancy smoking exposure only, as air pollution data was not available in usable form. Air pollution data was needed for further evaluation and for assessment of the substantial impact on birth and child health outcomes independently and when combined with pregnancy smoking exposure.

*The Merseyside Child Health Survey was completed by the candidate under supervision in 15 primary schools located in Bootle, Liverpool in 2006, which was the final part of follow up surveys carried out in 1991, 1993, and 1998. This was possible through grants from the Sefton Health Authority, the Liverpool Children's Research Fund, and the Leverhulme Fund awarded to the Liverpool School of Tropical Medicine (Principal Investigator, Professor Bernard Brabin).

*The candidate participated in the field data collection for the 2006 survey, taking responsibility for visiting the study schools in Sefton and for providing parental questionnaires for distribution through those schools. The collation of the database, cleaning and its utilisation for the analysis, the statistical methodologies and the specific analyses all have been carried out solely by the candidate under the supervision of the principal investigator with co-supervision from Professor Brian Faragher.

*The candidate was responsible for data capture using optical reading of the questionnaires, for the data cleaning and manual checking of all entries, and for completing the statistical analysis using SPSS. The initial child survey undertaken in 1991 examined the respiratory health in a large sample of 5 to 11 year old primary school children. This survey and the subsequent Respiratory Health Surveys provided a large and comprehensive data set on smoking patterns of mothers during their pregnancy, history of childhood asthma, weight and height of children, birth weight and gestational age.

* The work completed for and reported in the PhD needed substantial further development by linking two datasets from the same period using post code sectors, and further detailed analysis of the final dataset using different and advanced statistical techniques. These were new skills and methodologies that the candidate had to acquire to enable him to apply analytical methods (such as Venn diagram illustrations, Structural Equation Modelling, and Spatial mapping) to this MPhil thesis that were substantially different to those used in the PhD.

*The pregnancy smoking exposure data set was studied extensively as part of the PhD project and has been reported in six papers in peer reviewed journals; these papers have been fully cross-referenced in this MPhil thesis along with the reference for the published PhD thesis in order to indicate how the previous work has been amplified and substantially developed in the present analysis. The work was original, innovative and facilitated practical use of unused public health data.

*The issues involved in getting access to the air pollution data and creating the new dataset are all summarised in section 3.9 in the methodology chapter, along with details relating to the several meetings the candidate had to attend, the regular visits made to the air pollution monitoring stations and the sources of air pollution near to the dock area, and the training sessions the candidate had to attend to complete the MPhil thesis. The differences between the PhD and MPhil thesis have been summarised as a table in Appendix L, Table A.

Abstract

APPLICATION OF MERSEYSIDE COMMUNITY CHILD HEALTH SURVEY DATA FOR ESTIMATING HEALTH RISKS FROM AIR POLLUTION AND EXPOSURE TO CIGARETTE SMOKE DURING PREGNANCY

Introduction: Air pollution is a serious public and environmental health risk that can result in child health problems. Environmental exposures have been well documented in the Sefton area of Liverpool with increased concern about air pollution effects on child health near to the Bootle Dock area.

Rationale: The main priority is to improve child environmental health indicators using existing information and to identify the knowledge gaps and risk groups for developing new policies. There is need for improving linkage and analysis of data from currently available resources.

Aim: To develop and establish a combined database of air pollution indicators, cigarette smoke exposure and health variables for the first time in UK using the 2006 Sefton Community Child Health Survey, and to complete a descriptive analysis of environmental exposures and child health.

Objectives: (1) To compute odds ratio estimates for adverse birth and child health outcomes in relation to PM_{10}, NO_x emissions and PM_{10}, NO_2 concentrations and combined with pregnancy smoking categories and to estimate mean emissions for NO_X and PM_{10}, and mean concentrations for PM_{10} and NO_2 in areas with high and low prevalence of adverse birth and child health outcomes. (2) To estimate population attributable risks for relevant child health outcomes and to develop Venn diagrams and spatial maps of health risk profiles.

Methodology: A descriptive cross-sectional study was conducted by linking air pollution data from Sefton area with data available from the Community Child Health Survey in 2006 using postcode sectors. PM_{10} and NOx monitoring data were available as total and average emissions measured in tonnes per annum and NO_2 and PM_{10} concentrations data in microgram per metre^3. Outcome variables included birth and child health outcomes. SPSS 20 was used for univariate analysis and backward stepwise logistic regression, and results were illustrated using Venn diagram and spatial mapping techniques. The research project had ethical approval from the Royal Liverpool Children's Hospital NHS Trust Ethical Committee, Alder Hey.

Results: Data from 792 school children aged 5 to 11 years from 10 schools combined with corresponding air pollution data were used. There was independent association of childhood obesity with combined high NO_X-PM_{10} emissions + maternal pregnancy smoking (Adjusted OR 4.47, 95% CI 1.22-16.43, p = 0.024); and croup with high NO_X-PM_{10} emissions + paternal smoking during pregnancy (AOR 0.02, 95% CI 0.01-0.84, p=0.034), and high NO_X-PM_{10} emissions + household smoking during pregnancy (AOR 0.15, 95% CI 0.04-0.70, p=0.015) after adjustment for confounding factors.

Conclusion: Combined high NOx-PM_{10} emissions and maternal smoking during pregnancy were associated with an increased risk of childhood obesity. Sequential community surveys of child health linked with air pollution data have an important role in assessing air pollution exposures and outcomes with the potential use of visual illustrations in child health promotion activities.

LIST OF ABBREVIATIONS

AADT	*Average Annual Daily Traffic*
AAP	*Ambient Air Pollution*
AOR	*Adjusted Odds Ratio*
ADHD	*Attention Deficit Hyperactivity Disorder*
AMOS	*Analysis of Moment Structure*
AQMA	*Air Quality Management Area*
BMI	*Body Mass Index*
CO	*Carbon Monoxide*
COMEAP	*Committee on Medical Effects of Air Pollutants*
CDC	*Centre for Disease Control and Prevention*
DDA	*Doctor Diagnosed Asthma*
DEFRA	*Department for Environment, Food and Rural Affairs*
DOH	*Department of Health*
ECMO	*Extra Corporeal Membrane Oxygenation*
ETS	*Environmental Tobacco Smoke*
FEV1	*Forced Expiratory Volume*
HSDP	*Household Smoking during Pregnancy*
IFS	*Infant Feeding Survey*
INEMAR	*Inventory of Emissions into the Atmosphere of the Veneto*
iNO	*Inhaled Nitric Oxide*
IUGR	*Intrauterine Growth Restriction*
ISAAC	*International Study of Asthma and Allergy in Children*
LAQM	*Local Air Quality Management*
LBW	*Low Birth Weight*
MIMAS	*Manchester Information and Association Services*
MSDP	*Maternal Smoking during Pregnancy*
MIEP	*Merseyside Improvement and Efficiency Partnership*
NO_2	*Nitrogen Dioxide*
NO	*Nitric Oxide*
NO_X	*Nitrogen Oxides ($NO + NO_2$)*
NCHS	*National Centre for Health and Statistics*
NHES	*National Household Education Survey*
OR	*Odds Ratio*
PAR	*Population Attributable Risk*
PM_{10}	*Particulate Matter less than 10μm aerodynamic diameter*
$PM_{2.5}$	*Particulate Matter less than 2.5 micrometres in aerodynamic diameter*
PPHN	*Persistent Pulmonary Hypertension*
PEF	*Peak Expiratory flow*
SD	*Standard Deviation*
SEM	*Structural Equation Modelling*
SIDS	*Sudden Infant Death Syndrome*
SPSS	*Statistical Package for Social Sciences*
SO2	*Sulpher Dioxide*
TEA	*Triethanolamine*
TRAP	*Traffic Related Air pollution*
WHO	*World Health Organisation*

CHAPTER 1

INTRODUCTION AND BACKGROUND

1.1 Introduction and background

Air pollution is a serious public and environmental health risk that can lead to child health problems (Landrigan et al, 2004). It has been reported to cause more number of deaths than the number of deaths resulting from AIDS, tuberculosis, malaria or breast cancer (WHO 2012; WHO 2014a; Yang et al 2013). These have been well documented in urban settings within Europe including UK, even though it has been an emerging problem for much of the developing world. Reports have suggested that UK has been badly affected with possibly 60,000 early deaths resulting from air pollution as reported by official advisory body the Committee on the Medical Effects of Air Pollutants (COMEAP 2015), whereas the annual mortality burden in the UK from exposure to outdoor air pollution has been reported to be equivalent to around 40,000 deaths (RCP and RCPCH Joint Air Pollution Report, 2016). Air pollution has now been declared as a "public health emergency" across the globe by the World Health Organisation which could have long lasting untoward effects on future generations (WHO, 2014). It has also been reported that around 7 million people died as a result of exposure to air pollution, out of which a total of 3.3 million died prematurely due to hazardous effects of air pollution, especially indoor pollution, with most of the deaths reported from highly polluted cities in China, India and Pakistan. This is more than double of the previous estimates and confirms air pollution as the world's largest single environmental health risk (WHO, 2014).

A joint study conducted by the World Bank and the Institute for Health Metrics and Evaluation (IHME) has reported air pollution as the world's fourth leading fatal health risk causing one in ten premature deaths in 2013 and has estimated that nearly 5.5 million lives were lost due to diseases associated with outdoor and household air pollution in 2013 causing human suffering especially to young children (World Bank and Institute for Health Metrics and Evaluation, 2016). There has been a drastic rise in the number of deaths related to ambient air pollution in heavily populated urbanized regions compared to the deaths from pollution resulting from cooking and heating sources, even though both types of air pollution caused 1 in 10 deaths in 2013 equivalent to more than six times the deaths caused by malaria (World Bank and Institute for Health Metrics and Evaluation, 2016). Researchers from Kings College reported that in London alone, nearly 9500 people died prematurely due to air pollution with 3537 deaths from exposure to NO_2 and 5,879 deaths from exposure to $PM_{2.5}$ (Walton et al, 2015). The legal limit on air pollution being crossed for hourly Nitrogen dioxide (NO_2) for the whole of 2016 by London for the entire year 2016 just within eight days, 'red alert' being issued

in Beijing, China leading to close down of schools and businesses and people staying indoors to keep away from harmful smog, odd even scheme trial for cars introduced in early January 2016 as part of the by Delhi government's road rationing policy – all suggests the emergence of air pollution as a major public health problem (Birkett S, 2016; Radio Free Asia, 2015; Ray & Kumar 2016). Latest data has revealed that the NO_2 concentrations exceeded EU guidelines at 86% of monitoring locations in Liverpool in 2011 (Liverpool City Council Air Quality Report, 2012), and it has been estimated that the deaths of 239 people aged 25+ in Liverpool in 2010 was attributed to fine particulate matter, with an estimated 2,440 years of life lost (Public Health England, 2014).

Depending on the age dependant factors and the nature of chemical substances to which children are exposed they may exhibit greater vulnerability than adults to toxic substances including tobacco smoke and air pollutants (Horak et al, 2002; Landrigan et al, 2004). Studies on outdoor air have shown that pollutants may disrupt the immune system and the proper development of the lungs in foetuses and young children (Schwartz, 2004). This effect on child lung development has been observed at a level at which no adverse effects occurred in adults highlighting the vulnerability of children (Gauderman et al, 2000). Exposure to cigarette smoke, a different form of indoor air pollution during pregnancy period increases the risk of various birth and child health problems including preterm birth, intrauterine growth restriction and respiratory health problems later in life (De Franza et al, 2004; Dewan et al, 2003; Nabet et al, 2005). There is a possibility that suspended particles in air emitted from cigarette smoke contribute to air pollution and the presence of thousands of toxins in cigarette smoke combined with toxins from air pollution exposure could lead to increased exposure among pregnant women and children, especially, in homes with household smokers and when located in areas with increased air pollution. Environmental pollution exposures to tiny airborne dust particles PM_{10} and oxides of Nitrogen (NOx) may have additional health effects including respiratory health problems such as asthma, bronchitis and cardiovascular problems in individuals who also smoke (Vondra et al, 1998; Ward et al, 2004; Tunnicliffe et al, 1994; Seaton et al, 2003).

Studies on factors influencing the respiratory health of children in the Sefton area of Merseyside, including airborne dust, had been reported twenty years ago (Milligan et al, 1994). This study showed that there was increased respiratory illness among children related to increased exposure to airborne dust in this area. Over the last decade (2000-2010), the Environmental Protection Department of Sefton Borough Council has monitored airborne

pollutant and dust levels in those areas that were included in sequential Respiratory Child Health Surveys during the 1990s. This new environmental data, and information from more recent child health surveys in this area, allowed a new analysis of the combined effects on child health of airborne dust, of airborne pollutants and cigarette exposures in children from these communities. The present thesis addresses this analysis utilising the selected study specific data extracted from the 2006 child health survey.

1.2 Rationale

Airborne dust exposures have been previously well documented in the Sefton area of Merseyside, with evidence for worse respiratory health in children in relation to airborne dust (Milligan et al, 1994). In view of this, there have been long standing concerns among residents in these Merseyside communities concerning air pollution, and particularly near to the Bootle Dock area. But previous studies have never looked into birth and child health outcomes in relation to air pollution and only the pregnancy smoking exposure and its associations were considered. There is need to address the air pollution issue and to look at the combined and confounding effects of pregnancy smoking exposure along with air pollution, when determining their association with birth and child health outcomes.

There is also emphasis on prevention as well as equity and health promotion as part of the NHS Environmental Protection of Children's Health Strategy. There is need for improving children's environmental health indicators using existing information by identifying knowledge gaps and risk groups in order to develop relevant policies. Linkage and analysis of data from various currently available sources is essential for developing modern methods with practical utility, which in turn, can be used as tools for future local applications and risk profiling. Although it has been recognised that air pollution and smoking can lead to health problems in children and adults, there have been no previous studies in the UK assessing both air pollution and cigarette smoke exposure and their combined effects on birth and child health outcomes using post-code sector based data.

Research Question

What are the health associations of exposure to the environmental airborne pollutants of NOx, NO_2, airborne dust (PM_{10}), and cigarette smoke in pregnancy, for mothers and young children in Sefton, Merseyside?

1.3 Aim

To determine the association of birth and child health outcomes with combined air pollution and exposure to cigarette smoke during pregnancy after establishing a combined database of pollution indicators, cigarette smoke exposure and health variables using the locally available and selected data from the 2006 Community Child Health Survey and air pollution data from Sefton Council for a descriptive analysis of environmental factors and child health.

1.4 Objectives

1.4.1 Primary objectives

For the Sefton Metropolitan area of Liverpool:

1.4.1.1 To compute odds ratio estimates for adverse birth and child health outcomes in relation to levels of PM_{10}, NOx emissions combined with pregnancy smoking categories

1.4.1.2 To estimate population attributable risks due to these exposures for relevant child health outcomes.

1.4.2 Secondary objectives

1.4.2.1 To compute odds ratio estimates for adverse birth and child health outcomes in relation to levels of PM_{10}, NO_2, concentrations combined with pregnancy smoking categories.

1.4.2.2 To estimate mean emissions for NO_X and PM_{10}, and mean concentrations for PM_{10} and NO_2 in areas with high and low prevalence of adverse birth and child health outcomes.

1.4.2.3 To develop Venn diagrams and spatial maps of health risk profiles of public health importance for potential use in child health promotion in the Sefton Merseyside area.

CHAPTER 2

REVIEW OF LITERATURE

2.1 Overview

This chapter focuses on the literature on birth and child health outcomes related to environmental air pollution exposures. The search strategy is outlined, and definitions are provided for air pollution, and the types, sources, chemical nature, and measurement of airborne pollutants, and their health effects in children have also been included. Details on regulation and National Programs for monitoring are provided. A separate section on combined air pollution and pregnancy smoking exposure and related toxicology has also been included.

2.2 Search strategy

A search strategy was formulated based on relevant key words using online databases including PubMed, Scopus, Embase, and Web of knowledge in order to provide a broadly relevant contemporary review based on information extracted from selected articles. Information from potential conference proceedings, abstracts and relevant journals in English were manually searched. The wild card search terms used were air, smoking, pregnan*, pollut*, child*, health, problem*, and outcome* for this review. Special emphasis was given to articles based on oxides of nitrogen (NOx) and particulate matter (PM_{10}), and cigarette smoke. The search terms used included air, smoking, pregnant/ pregnancy, pollution / pollutant, child / childhood, health, problem / problems and outcome / outcomes. The search criteria used for the search for relevant articles related to air pollution and pregnancy smoking using different search engines are summarised as follows:

(air AND pollut*) OR (smoking AND pregnan*) AND (child* OR birth) AND health AND (outcom* OR problem*)

The details of the search terms are summarised in Appendix A. Human studies published between February 1990 and February 2017 were included along with selected studies published before that period. The final list of articles from results obtained by this search strategy were cited after scanning through all the abstracts, removal of duplicate studies and after narrowing down to selected relevant articles matching the specific research aims and objectives. Abstracts were also examined to exclude studies which reported policies and commentaries. Studies, repetitive of the original research study and issued in different publications, abstracts or conference proceeding were recorded only once, after matching with

the original article. Those excluded were mostly not within the specified period, or articles not relevant to the research topic. Unrelated studies, not addressing air pollution, smoking and child health were removed. Studies involving experimental animals, or skin tests for sensitisation were mostly excluded. Other resources including study related published reports were searched and unpublished and on-going studies were also checked. Reference lists of identified studies were checked using the search terms. Other information, relevant to the present study aims, was included in the literature review including information from some articles published before 1990, and related information available from published or unpublished reports by the Environmental Protection Department of the Sefton Borough Council and were considered when relevant to the local Merseyside situation as well.

2.3 Air pollution

2.3.1 Definition

There is no ideal definition for air pollution. It may be considered as a process in which emission of any substance into the air occurs from an anthropogenic, biogenic, or geogenic source, that is either not part of the natural atmosphere or is present in higher concentrations than the natural atmosphere, and that may cause a short-term or long-term adverse effect (Daly and Zannetti, 2007). This includes chemicals, biological material, or particulate matter, which could cause harm to other organisms by altering the natural environment and ecosystems. For quantification of the level of atmospheric pollution, the atmospheric pollutant emissions have to be differentiated from atmospheric pollution concentrations. The definition for emissions and concentrations and their categories have been summarised in table no 3.01, pages 71-77 in the methods chapter. Emissions and concentrations represent the value of air quality (INEMAR, 2010).

The 2005 WHO Air quality guidelines (AQGs) were first produced in 1987 and were designed to offer global guidance on reducing the health impacts of air pollution. This was updated in 1997 and the new 2005 guidelines apply worldwide and are based on expert evaluation of current scientific evidence. It was concluded that even relatively low concentrations of air pollutants have been related to a range of adverse health effects. Guideline values for each air pollutant were finalised with the need for achieving progress towards these values as the ultimate objective was emphasised. The guideline values for each air pollutant are summarised in table 2.01.

Table 2.01 Air Quality guideline values (WHO, 2005)

Air pollutant	Guideline value	Comments
Sulphur dioxide (SO_2)	20 μg/m^3 24-hour mean 500 μg/m^3 10-minute mean	A SO_2 concentration of 500 μg/m^3 should not be exceeded over average periods of 10 minutes duration
Nitrogen dioxide (NO_2)	40 μg/m^3 annual mean 200 μg/m^3 1-hour mean	At short-term concentrations exceeding 200 μg/m^3, it is a toxic gas which causes inflammation of the airways.
Ozone (O_3)	100 μg/m^3 8-hour mean	The previously recommended limit, which was fixed at 120 μg/m^3 8-hour mean, has been reduced to 100 μg/m^3 based on recent conclusive associations between daily mortality and ozone levels occurring at ozone concentrations below 120 μg/m^3.
Particulate matter ($PM_{2.5}$) *	10 μg/m^3 annual mean 25 μg/m^3 24-hour mean	More harmful since, when inhaled, they may reach the peripheral regions of the bronchioles, and interfere with gas exchange inside the lungs.
Particulate matter (PM_{10})**	20 μg/m^3 annual mean 50 μg/m^3 24-hour mean	As no threshold for particulate matter has been identified below which no damage to health is observed, the recommended value should represent an acceptable and achievable objective to minimize health effects in the context of local constraints, capabilities and public health priorities.

* aerodynamic diameter < 2.5 μm

** aerodynamic diameter < 10 μm

The European Air Quality standards have been developed and an extensive body of legislation which establishes health based standards and objectives for a number of pollutants in air has been formulated by the European Union and European Commission in 2008. These standards are quite variable from the 2005 WHO Air Quality guidelines and are summarised in table 2.02. The difference is mainly new $PM_{2.5}$ objectives are based on the average exposure indicator (AEI), which is calculated from the three year annual mean concentration of $PM_{2.5}$ averaged over selected monitoring stations. These standards apply over variable time periods because the observed health impacts associated with the various pollutants occur over different exposure times.

Table 2.02 European Air Quality Standards for air pollutants

Air pollutant	**Concentration**	**Averaging period**
Fine particles ($PM_{2.5}$)	25 $\mu g/m^3$	1 year
Sulpher dioxide	350 $\mu g/m^3$ 125 $\mu g/m^3$	1 hour 24 hours
Nitrogen dioxide	200 $\mu g/m^3$ 40 $\mu g/m^3$	1 hour 1 year
PM_{10}	50 $\mu g/m^3$ 40 $\mu g/m^3$	24 hours 1 year
Lead (Pb)	0.5 $\mu g/m^3$	1 year
Carbon monoxide (CO)	10 mg/m^3	Maximum daily 8 hour mean
Benzene	5 $\mu g/m^3$	1 year
Ozone	120 $\mu g/m^3$	Maximum daily 8 hour mean
Arsenic	6 ng/m^3	1 year
Cadmium	5 ng/m^3	1 year
Nickel	20 ng/m^3	1 year
Polycyclic Aromatic hydrocarbons	1 ng/m^3	1 year

* aerodynamic diameter < 2.5 µm

** aerodynamic diameter < 10 µm

RCP and RCPCH Joint Air Pollution Report (2016), a joint report by the Royal College of Paediatrics and Child Health (RCPCH) and the Royal College of Physicians (RCP) based on recommendations drawn from evidence from assembled experts in medicine and environmental sciences suggests that neither the air pollutant concentration limits set by the UK government nor the World Health Organization's air quality guidelines, define levels of exposure that are entirely safe for the whole population. They have also reported about the damage from air pollution occurring across a lifetime with evidence pointing towards adverse effects on growth, intelligence and development of brain and coordination.

2.3.2 Types of air pollutants

2.3.2.1 Based on source. Pollutants can be classified as primary or secondary.

A Primary air pollutants

Substances that are directly emitted into the atmosphere from different sources are known as primary air pollutants. The main primary pollutants which cause harm in very high concentrations include compounds of sulphur - Hydrogen disulphide (H_2S) and Sulphur dioxide (SO_2); compounds of Nitrogen - Nitric oxide (NO), Nitrogen dioxide (NO_2 and Ammonia (NH_3) and Volatile Organic Compounds (VOC).

B Secondary air pollutants

Secondary air pollutants have no direct source of emission and are formed in the atmosphere from primary pollutants, which are precursors. The main secondary pollutants are nitrogen dioxide (NO_2) and nitric acid (HNO_3) formed from NO; Ozone (O_3) formed from photochemical reactions of nitrogen oxides and volatile organic compounds (VOCs) and sulphuric acid (H_2SO_4) droplets formed from SO_2 and Ammonium Bisulphate (NH_4HSO_4) and Nitrates formed from reactions of sulphuric acid droplets and nitric acid droplets with ammonia (NH_3), respectively.

2.3.2.2 Based on location.

Air pollutants are classified into outdoor and indoor based on their source and location.

A Outdoor air pollutants

These include motor vehicle emissions which contain volatile organic compounds (VOCs), oxides of nitrogen (NOx) especially nitrogen dioxide (NO_2) and lead, and oxides of sulphur. Outdoor secondary air pollutants, such as Ozone (O3), are produced from the action of sunlight on VOCs and NO_2 (Newman & Taylor 1995). Effects of individual pollutants may vary and is related to modifying effects of pollutants on each other (Koenig et al, 1991).

B Indoor air pollutants

These are the pollutants which are formed from sources inside homes. Distribution of indoor air pollutants varies by geographic area, socio-economic class and cultural background. Indoor air pollutants differ from outdoor air pollutants based not only on the source of pollution, but also on their concentration and composition (Allen et al, 2003; Long et al, 2000; Turpin et al, 2007; Wallace et al, 1996). The main component of indoor air pollution in industrialised countries includes products from combustion including particulate matter (PM) and oxides of nitrogen (Wallace et al, 1996), airborne allergens, pollen grains and endotoxins. Particular matter, which originates either from artificial or natural resources, is a principal component responsible for many respiratory symptoms especially among children (McCormack et al, 2008). The artificial source for particulate matter includes emissions from industries and motor vehicles and combustion by-products from incinerators and power plants, which gets trapped indoors by transport from outside sources and through ventilation sources and weather conditions including the direction of wind and proximity to industrial and traffic air pollution sources, whereas the natural sources include pollen grains, bacteria, plant and animal debris and spores. Other indoor sources include activities that re-suspend particles such as cleaning and sweeping, and suspended particles from cigarette smoking, cooking and particles from burning of biomass and wood, as well as penetration of outdoor air pollutants into the indoor environment (Wallace et al, 1996; McCormack et al, 2008). Pollutants can accumulate and reach concentrations greater than outside, if buildings are not properly ventilated, then this can result in a condition known as the ‘sick building syndrome’, a condition affecting building occupants, who claim to experience acute health effects which is linked to the overall time spent in the building (Health Canada, 1995). One of the main contributors to indoor pollution is environmental tobacco smoke.

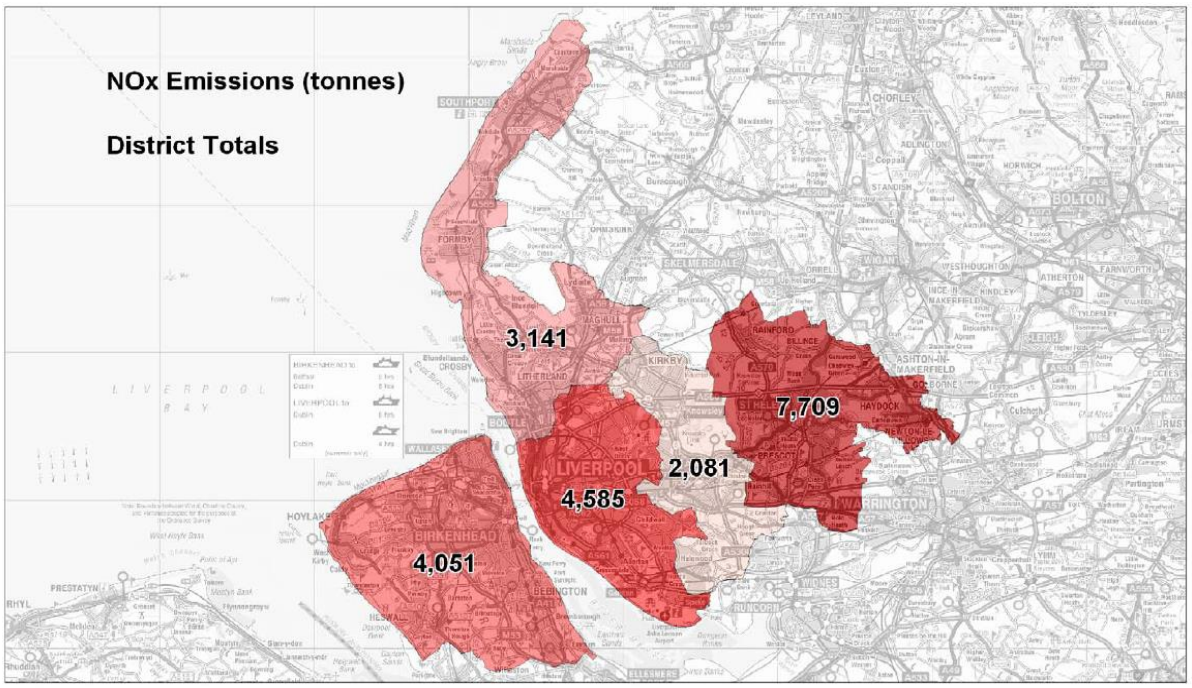
2.3.3 Sources of air pollution

Road traffic contributes to both NO_2 and PM_{10} levels causing outdoor pollution. Traffic emissions are a primary source of nitrogen oxides (NOx) which are converted to nitrogen dioxide (NO_2) (Skene, 2010). Traffic is believed to be responsible for at least half of NOx emissions and accounts for a higher proportion of exposures in urban areas (HEI, 2010). NO_2 is a precursor for several other air pollutants including nitric acid and ozone (WHO, 2003). Of particular relevance when assessing exposure to NO_2 is the observation that levels can vary over a small distance of 1500 metres (Jerrett et al, 2005; WHO, 2003).

In Sefton, Merseyside, a study on air pollution source was undertaken to investigate the concentrations of different sources, total emissions, and annual average concentrations in selected Air Quality Management Areas (AQMAs) (DEFRA, 2007). AQMA's are areas identified by the local authority by carrying out regular reviews and assessment of air quality and includes any places or even streets, which are less likely to achieve the national air objectives by relevant deadlines. The national air objectives are essential for protection of people's health and environment and based on the results, the local authority formulates a plan known as the Local Air Quality Action Plan to improve the air quality of that specific area. The results showed that the greatest contributor to annual average NO_2 concentration at the monitoring locations was from road traffic. There was an increased proportion of NOx emissions from commercial and domestic sources within Sefton, with relatively small contribution from ground level concentrations of NO_2. The concentration of annual average PM_{10} was dominated by particulates blown into Sefton from the surrounding areas from traffic and industrial sources. The other two major contributors to air pollution included emissions from industries and concentrations arising from other sources including trains, domestic and background concentrations (DEFRA, 2007).

A survey undertaken by the Environmental Health Department of Sefton Council demonstrated that the NOx emissions mainly resulted from transport (43%) and industrial and commercial activities (41%), followed by domestic (14%), and agricultural activities (2%). Major contributors of transport NO_X were road traffic (80%) and shipping (15%), followed by rail traffic (3%) and airport traffic (2%) (Merseyside Atmospheric Emissions Inventory 2006). Figure 2.01 summarises district totals for total nitrogen oxides emissions measured in tonnes per annum in 2006 for different districts in Merseyside.

Figure 2.01 Total Nitrogen Oxides (NO_X) emissions in 2006



Ref: Merseyside Atmospheric Emissions Inventory 2006

CO_2 emissions in the Sefton area were mainly a result of industrial and commercial activities (40%), followed by domestic (37%), transport (23%) and agricultural activities (0.1%). PM_{10} emissions mainly resulted from industrial and commercial activities (52%) and transport (38%), followed by domestic (7%), and agricultural activities (3%) (Merseyside Atmospheric Emissions Inventory 2006).

Table 2.03 compares road transport as a source contributing to total emissions in Merseyside compared to greater London and nationally.

Table 2.03 Road transport contribution to total emissions comparing UK, Greater London and Merseyside

Pollutant	UK	Greater London	Merseyside
Benzene	18%	51%	56%
1, 3 Butadiene	55%	91%	92%
Carbon Monoxide	43%	68%	68%
Nitrogen oxides	32%	42%	35%

Particulates, PM_{10}	21%	69%	31%
Carbon dioxide	22%	25%	22%

Source: UK Emissions of Air pollutants 1970 to 2006 (DEFRA), London Atmospheric Emissions Inventory 2004, Merseyside Atmospheric Emissions Inventory 2006

This shows that for all the pollutants the contribution to total emissions from road transport is much higher in Merseyside than the national average, however when compared to Greater London the estimates were quite comparable except for PM_{10} which was lower for Merseyside.The chemical nature of air pollutants are reviewed next.

2.3.4 Air pollutants

2.3.4.1 Chemical nature

This depends on the composition, combinations, and level of different chemicals in specific locations. Important chemical compounds formed include sulphur dioxide, oxides of nitrogen, carbon monoxide, ozone, and the volatile organic micro pollutants of benzene and butadiene.

A Sulphur dioxide (SO_2)

Sulphur compounds are present in trace amounts in fossil fuels and they are released when burnt. Most emissions are from power generation with a smaller contribution from transport sources and from shipping. Sulphur dioxide in air reacts with water in atmospheric moisture to form sulphuric acid and acid rain. Sulphuric acid and ammonium sulphate particles are the most abundant secondary particles found in air and have detrimental effects on the bronchial system (Tunnicliffe et al, 2003) presenting as bronchoconstriction in asthmatic patients after inhalation of sulphuric acid at concentrations of 1000 mg/m^3.

B Oxides of Nitrogen (NOx)

NOx is a term used to describe a mixture of nitric oxide (NO) and nitrogen dioxide (NO_2). They are inorganic gases formed by combination of oxygen with nitrogen. NO is produced in much greater quantities from traffic sources than NO_2, but is oxidised to NO_2 in the atmosphere. NO_2 causes detrimental effects to the bronchial system. Nitrogen dioxide concentrations frequently approach, and sometimes exceed air quality standards (48 µg/m3 for annual NO_2 limit value) averaged annually in many European cities (European Air Quality Standards, European Commissions 2014). NOx is emitted when fuel is being burned e.g. in power generation (70%) and transport and industrial processes (30%) (Cooper & Alley, 1986).

C Carbon Monoxide (CO)

CO is an odourless, tasteless and colourless gas produced by the incomplete burning of materials which contain carbon, including most transport fuels. CO is produced as a consequence of the incomplete combustion of hydrocarbons, is toxic in nature, and acts by reaction with haemoglobin forming carboxyhaemoglobins which reduces the capacity for oxygen transport in the blood. Major sources of CO include motor vehicle exhaust, generators, and other fuel-burning equipments; poorly maintained, poorly functioning, or unventilated heating and cooking appliances (e.g., kerosene and gas space heaters, woodstoves, fireplaces, gas stoves, boilers, and furnaces) and other occupational sources (Ernst, 1998).

D Ozone (O3)

Ground-level ozone (O_3) differs from many other pollutants in that it is not emitted directly into the atmosphere, but occurs as a secondary pollutant produced by photochemical reactions between nitrogen dioxide (NO_2), hydrocarbons and sunlight. Ozone levels are not as high in rural areas compared to urban areas where high levels of NO_2 are emitted from vehicles and power plants. Sunlight provides the energy to initiate ozone formation and consequently, high levels of ozone are generally observed during hot weather during the summer period and especially in the afternoon.

E Hydrocarbons and volatile organic compounds (VOC)

Hydrocarbons belong to a larger group of chemicals known as volatile organic compounds (VOC). The difference between hydrocarbons and volatile organic compounds is that hydrocarbons are compounds formed from hydrogen and carbon only, whereas volatile organic compounds contain other elements. Volatile organic micro pollutants are produced by incomplete combustion of hydrocarbon fuels and during evaporation processes. Sources of VOCs include gasoline, chemical industry, paints, and consumer products. There are hundreds of compounds formed from hydrocarbons displaying a wide range of properties. Some important hydrocarbons are benzene and butadiene. Benzene is a volatile organic by-product formed as a result of the combustion process of aromatics and fuels like petrol and diesel. It is also released as exhaust from petrol engine vehicles, and also from petrol refineries. 1, 3 Butadiene is a gas at normal atmospheric temperature and pressure and is similar to Benzene. It is released as a result of the combustion process of petrol and other petrochemical products. Motor vehicles are the main source of 1, 3 butadiene. But it is also an important chemical used in rubber industry as a monomer in the production of synthetic rubber (polybutadiene) by polymerisation process (Morrow et al, 1990).

F Lead

There has been a drastic reduction in the level of lead content in the atmosphere from traffic emissions with the increased use of unleaded petrol or petrochemical products with reduced lead content. This followed the implementation of the European Union Fuel Quality Standards. Secondary non-ferrous metal smelters contribute more to airborne emissions of lead in industrial areas. Lead has been most widely used in battery manufacturing industries both in its elemental as well as compound form. It is difficult to find a threshold for the effects of lead especially in children. The Centre for Disease Control (CDC) recommends public health actions to be initiated if the blood lead levels are above the reference cut off value of 5 micrograms per decilitre.

G Particulate matter (PM)

Airborne particles vary widely in physical and chemical composition, source and particle size. These particulates affect more people than any other pollutant. The major

components of PM are sulphates, nitrates, ammonia, sodium chloride, carbon, mineral dust and water. Particulates consist of a complex mixture of solid and liquid particles of organic and inorganic substances suspended in the air. They include smoke, dust, aerosols, metallic oxides, and pollen. Sources of PM include combustion, factories, construction sites, demolition areas, agricultural activities, motor vehicles, and wood burning. Inhalation of enough PM over time increases the risk of chronic respiratory disease in females (Schikowski et al, 2014) and could also result in acute coronary events including unstable angina and heart attacks especially in elderly population on chronic exposure to $PM_{2.5}$ (Guo et al 2013; Cesaroni et al, 2014).

Two types of particles are commonly cited in studies on health effects of air pollution. Black smoke refers to fine suspended particulates arising from the incomplete combustion of fossil fuels. Total suspended particulates refer to a mixture of airborne particles such as rock dust and combustion products.

The particles are identified according to their aerodynamic diameter, as either PM_{10} (particles with an aerodynamic diameter smaller than 10 μm) or $PM_{2.5}$ (aerodynamic diameter smaller than 2.5 μm). Both the particles are referred to as respirable particles as they are able to deposit in smaller airways and alveoli. However, the latter are more dangerous and are more likely to interfere with the gas exchange inside the lungs and can be absorbed into blood possibly explaining the association with heart disease. Larger particles meanwhile, are not readily inhaled, and are removed relatively efficiently from the air by sedimentation. The principal source of airborne PM_{10} and $PM_{2.5}$ matter in European cities is road traffic emissions, particularly from diesel vehicles. The limit values shown in table 2.02 are very often exceeded in European cities.

In many industrial areas there is a close relationship between particles and sulphur (HMSO, 1995). Particles greater than 2.5 micrometers are removed from the atmosphere by settling and rain, whereas very small particles (< 1 micrometer) can remain airborne for several weeks and during this time may travel large distances. Ullmann et al (2013) showed using indoor personal monitoring techniques that these samples may contain quantities of very fine particles similar to amounts as encountered outdoors. It has been estimated that life expectancy was reduced by 8.6 months in the European Union, and by 10.2 months in Germany attributable to adverse effects from levels of PM concentrations (Amann, 2005). The characteristics and health effects of different pollutants have been summarised in table 2.04.

Table 2.04 Characteristics of air pollutants

Air pollutant	Nature	Sources	Health effects
SO_2	Toxic gas with a pungent, irritating and rotten smell.	Power generators, transport and shipping fossil fuel combustion at power plants and other industrial facilities.	Adverse respiratory effects including bronchoconstriction and increased asthma symptoms.
NO_X	Colourless, non-flammable gas, with a slightly sweet odour and taste	Fossil fuels, power generators, transport combustion of coal and oil at electric power plants, and also during the combustion of gasoline in automobiles burning of fossil fuels: coal, oil and gas industries and food processing.	Contributes to the formation of photochemical smog with increased likelihood of respiratory problems like wheezing, coughing, colds, flu and bronchitis by reducing immunity to lung infections.
CO	Colourless, odourless, tasteless	Incomplete combustion of hydrocarbons, vehicle exhaust, generators, fuel burning equipment, poorly maintained and unventilated heating and cooking appliances and tobacco smoke.	Reduces capacity of oxygen transport in blood and causes headaches, dizziness, and disorientation, nausea and fatigue cause flu-like symptoms. At moderate concentrations, angina, impaired vision, and reduced brain function may result.
Ozone	Pale blue gas with a distinctively pungent smell	Secondary pollutant from photochemical reaction between Nitrogen dioxide, hydrocarbon and vehicle created in the presence of sunlight	Ground level ozone can harm lung function and irritate the respiratory system and exposure has been linked to premature death, asthma, bronchitis, heart attack, and other cardiopulmonary problems

Hydrocarbon VOC	Chemicals containing hydrogen, carbon, and possibly other elements, that evaporate easily.	Emissions from industrial facilities, electric utilities, motor vehicle exhaust, gasoline vapors, and chemical solvents and from burning of Coal, oil, and natural gas.	Carcinogenic. Long-term exposure can damage the liver, kidneys, and nervous system
Lead	Soft metal found in air	Today, metal processing is the biggest source of atmospheric lead. The highest air concentrations are found in the vicinity of ferrous and nonferrous smelters and battery manufacturers. Other sources are from burning of fuel used in motor vehicles, industrial sources, lead smelter products such as paint, gasoline, and batteries.	Affects the neurological system on chronic exposure, causing seizures, behavioral disorders and lowered IQ. May contribute to high blood pressure and heart disease and can harm the kidneys, heart and liver.
Particulate matter	Complex mixture of small particles and liquid droplets made up of number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles.	Road traffic emissions, particularly from diesel vehicles, burning of fossil fuels, power plants and various industrial processes	Once inhaled, these particles can affect the heart and lungs and cause serious health effects premature death in people with heart or lung disease can also cause decreased lung function, and aggravates asthma.

Source: Environmental Protection Agency. National Ambient Air Quality Standards (2008)

2.3.5 Methods of air pollution measurement and monitoring

Different methods have been used in various locations around the world to measure air pollution. Smoke stain resulting from air sample being drawn through a filter paper was used many for years to measure concentrations of black smoke using a standard calibration curve. A newer method is the gravimetric method which measures the mass of particulate matter obtained after a known volume of air is drawn through filter paper. A useful device is the PM10 sampling head which allows particles of 10 micrometer aerodynamic diameter or less to be captured.

Myrick et al (1996) reported air pollution data from the National Air Pollution Surveillance system (NAPS) using urban background fixed monitors in different stations in Edmonton, Canada. 'Reference methods' or 'Equivalent methods' designed by the US.

Monitoring and measurement of air quality is essential and carried out by different organisations such as local councils, environment pressure groups and research bodies as there are many towns in the UK which periodically experience unhealthy levels of pollution. Highly specialised and delicate instruments are used for measuring the range of pollutants and these are mostly called active devices as they are operated by pumps which suck air into them. There are three main methods available for measuring air pollution: passive, active and automatic sampling (Cortes et al 2014; Gaga et al 2012). Passive sampling provides a simple and inexpensive indication of average pollution levels over a period of weeks or months and is useful in highlighting hotspots where more detailed study is needed, whereas active and automatic sampling are more expensive and use sophisticated methods for producing high resolution measurements of a range of pollutants at a single point. Air pollution monitoring can be further classified into source and ambient air monitoring. Source monitoring measures emissions directly from a fixed or mobile source, whereas ambient air monitoring involves measurement of specific pollutants present in an immediate surrounding atmosphere. Automated continuous monitors are used in the UK continuously every hour of every day for measuring air quality as the UK Government is legally required to measure air pollution by the European Union in order to improve its air quality. Monitoring and measuring of air quality is also carried out by local councils and if in any area, air pollution levels above set limits are identified, it would be declared as an Air Quality Management Areas (AQMA) and placed in an Air Quality Action Plan to improve the situation.

The Environmental Protection Agency (2008) provides data on carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O3) and sulphur dioxide (SO_2). NO_2 is measured using chemiluminescence, CO using non-dispersive infrared spectrometry, O_3 using chemiluminescence / ultraviolet photometry, and SO_2 using colorimetric methods and ultraviolet fluorescence techniques. Particulate matter ($PM_{2.5}$ and PM_{10}) is measured using tapered element oscillating microbalance instruments, and concentrations estimated with a light scattering nephelometer.

Instruments used for measuring different air pollutants are summarised in table 2.05.

Table 2.05 Air pollutants and instruments used for measurement

Air pollutant	Instrument used
Nitrogen dioxide	Diffusion tube Chemiluminescence continuous analyser
$PM_{10}/PM_{2.5}$	Tapered Element Oscillating Microbalance (TEOM) Radiometric method/Light scattering Nephelometer
SO_2	Ultra violet fluorescence continuous analyser /Colorimetry
Ozone	Ultraviolet absorption photometry
CO	IR-correlation absorption spectrometry Non-dispersive infrared spectrometry
Aromatic hydrocarbons	BTX analysers and gas chromatography

Source: Environmental Protection Agency (2008)

2.3.6 Health consequences of air pollution

Air pollution is known to cause health problems irrespective of the age group exposed. Air pollutants can have immediate effects (acute) commonly seen with high levels of air pollutants or longer term effects (chronic) seen with chronic exposures to lower levels. Concentrations of air pollution over a given period of time and effects of each pollutant on health and on the environment can be used as the standard of air pollution and also as a benchmark to monitor air pollution trends.

2.3.6.1 Respiratory symptoms

Nitrogen dioxide causes inflammation of the airways of the lungs and increases the likelihood of respiratory problems including wheezing, coughing, colds, flu and bronchitis. It can also increase the frequency and intensity of asthma attacks especially in children due to reduced lung function and airway responsiveness and in older people with heart disease (Gillespie-Bennet et al, 2011). This was more commonly seem with indoor NO_2 concentration and not with outdoor NO_2. Children are more vulnerable as they breathe 50 percent more air per kg of body weight than adults.

It has been reported that the smoke that is released from combustion of biomass fuels (wood and charcoal) for cooking contains health damaging pollutants (Taylor et al, 2011). A study from Sierra Leone which investigated the prevalence of acute respiratory infection (ARI) among 520 children under 5 years of age at home exposed to smoke from burnt wood compared to charcoal stoves showed that ARI prevalence was higher among children from the first group (64%) compared to the later (44%). Intensity of smoke in this study was measured based on continuous monitoring of suspended particulate matter (SPM). Although this study highlighted the impact of smoke generated from wood and charcoal stoves on childhood ARI prevalence in a developing country it did not take into account past exposures or recent changes in cooking methods. As ARI risk is likely to be cumulative over time these combinations of factors should also be considered. Non-inclusion of other confounding factors including birth weight, growth and nutritional status, immunization history, the level of the mother's education narrows the strength of the conclusions that could be drawn. Studies over the past few years have shown the potential for toxic exposures in children due to air pollutants other than dust (Gao Y et al, 2014; Negrisoli et al, 2013; Lippmann et al, 2013; Fuertes et al, 2013; Liu et al, 2014; Macintyre et al, 2014; Schifano P et al, 2013; Jung CR et al, 2013). For example, Negrisoli et al (2013) in their ecological time-series study carried out from 2007 to 2008, used data from the public health system of Sorocaba, and reported that there was a statistically significant association between childhood pneumonia and exposure to nitrogen dioxide exposure (RR=1.016) and particulate matter (RR=1.009), using the multi–pollutant model. This significant association was noted despite a difference between the time of development of respiratory symptoms and the exposure time, with an earlier occurrence of pneumonia in relation to nitrogen dioxide exposure. Gao et al (2014), reported health outcomes in a cross sectional study among 2,203 children aged 8 to 10 years from three districts in Hong Kong in relation to airborne levels of PM10, SO2, NO2 and O3. PM10 and NO2 levels were associated with nocturnal cough (adjusted OR = 1.81 95% CI 1.71-2.78) and phlegm production in girls (adjusted OR = 3.84 95% CI 1.74-8.47), whereas only marginal significance was achieved for elevated risks for respiratory symptoms including asthma, wheezing symptoms and phlegm without cold among boys in highly air polluted areas.

Ellegerd (1996) reported mean emissions levels for PM_{10} of 1220 and 540 micrograms respectively for wood and charcoal stoves during cooking, which might explain their relative effects in terms of acute respiratory infection. Children and women are more exposed to biomass smoke in developing countries because of their frequent presence in the kitchen

environment and because of the complex mixtures of component pollutants present in biomass smoke. This comprises particulate matter, carbon monoxide, nitrogen dioxide, volatile organic compounds and aromatic hydrocarbons (Smith et al, 1993). The use of gas for cooking is associated with the production of NO_2 which may accumulate in kitchens without adequate ventilation. NO_2 has low solubility and greater than 60% of inhaled NO_2 is deposited in the lung. Melia et al, (1982 and 1985) in their series of epidemiological studies reported that respiratory symptoms in primary school children are associated with a combination of gases used for cooking, and the use of kerosene heaters, but that this association might be lost as children grow older.

The effect of air pollutants on the respiratory system depends on the depth to which the air pollutant particles penetrate the airways, which depends on airway characteristics such as size, density, shape, aerodynamic diameter and also the volume of air processed in each respiratory cycle (Parkes et al, 1994). Small dust particles of 10 micrometer size usually deposit by sedimentation, whereas small particles (2.5 micrometer) and very small particles (<1 micrometer) deposit by diffusion through Brownian movement. This is commonly seen in very small airways and alveoli where the airway surface is large and airflow velocity is very low (Parkes et al, 1994). The effect of particulate matter on the respiratory tract specifically depends on the particle size, deposited dose and its chemical and physical properties. Occupational exposures can lead to larger exposure and result in conditions like asbestosis, silicosis which are more common in adults, whereas environmental exposures are generally smaller and effects are seen both in children and adults.

Clearance mechanisms play a major role in the respiratory mechanisms and responses in relation to exposure to air pollutants. The deposited particulate matter gets cleared from the respiratory system which mainly occurs through a process which involves the combined effects of mucus, ciliary activity, phagocytosis and lymphatic drainage. This in turn depends on the size of the airways. Cilia and mucus are important for clearance in larger airways whereas in smaller airways and alveoli, phagocytosis by macrophages and lymph drainage are important. Deposited foreign particles are trapped and retained by the mucinous lining of the airways (Gilboa and Siberberg, 1976). Ciliated epithelial cells beat beneath the mucus layer and help to transport mucus into upper airways. This mucinous layer may be affected by irritation and inflammation in the diseased state resulting in defective muco-ciliary clearance (Salathe et al, 1997). Respiratory tract damage occurs when the rate of deposition exceeds the rate of removal

of particles which are highly chemically active. Deposition of large particles may cause bronchitis, whereas small particles result in alveolar disease. Exposure to air pollutants especially high concentrations of SO_2 may alter the clearance mechanism resulting in ciliary inactivity and leading to fixed deposition of more particulate matter in the airways. Two cross-sectional Dutch studies have reported higher rates of respiratory symptoms among children in schools near roadways with heavy traffic, especially truck traffic (Janssen et al, 2003; Van Vliet et al, 1997). In young children as their respiratory system is not completely developed before the age of six years, and because they breathe more air in proportion to their weight compared to adults, and often spending more time indoors, they form a special group at risk of indoor air pollution (Schwartz et al, 2004). This is especially in the first year of life which is a critical period related to exposure to air pollution, and which may lead to later respiratory health problems.

A. Asthma

The role of air pollution in the development of new-onset asthma remains controversial, and the contribution of air pollution as an environmental risk factor to this child health respiratory problem is unclear (Holguin, 2007; Eder et al, 2006; Sarnat and Holguin, 2007). Laboratory and epidemiological studies have been carried out investigating the associations between increased air pollutants and change in respiratory morbidity in asthmatic subjects.

(1) Epidemiological studies

A review by Burr (1995) concluded that air pollution episodes may exacerbate pre-existing respiratory symptoms in children. There have been numerous studies which reported increased morbidity in asthmatic children associated with particulate matter (PM) mass (Koenig et al, 2005; McConnell et al, 1999), particle number (von Klot et al, 2002), PM constituents (Delfino et al, 2003a & 2003b), nitrogen dioxide (NO_2) (Just et al, 2002; McConnell et al, 1999), carbon monoxide (Schildcrout et al, 2006), ozone (O_3) (Gent et al, 2003; Mortimer et al, 2000; Romieu et al, 2005), and sulphur dioxide (SO_2) (Segala et al, 1998).

Although there have been studies with evidence indicating increased prevalence of asthma among children living in areas near heavy traffic, some well-designed studies have reported only a decrease or no change in asthma prevalence (Heinrich and Wichmann 2004;

Oftedal et al. 2009). One of the epidemiological studies conducted in Northern and central Italy, included in a study reported by Heinrich and Wichmann (2004) found no association between traffic related exposure and increased prevalence of asthma, even though this study involved a large sample of 39275 children aged 6 to 7 and 13-14 years, although an association with increased wheeze was observed (Ciccone et al, 1998). A study conducted by Oftedal (2009) in which respiratory outcomes were assessed retrospectively among 2,871, 9 to 10 year old children in Oslo, using a parental questionnaire in relation to Nitrogen dioxide exposure, which was assessed by the EPISODE dispersion model, reported that an interquartile range (IQR) increase of NO_2 exposure before asthma onset was associated with an adjusted risk ratio of 0.82 [95% confidence interval (CI), 0.67–1.02), suggesting there was no positive association between any long-term traffic-related exposure and onset of doctor-diagnosed asthma. Brauer et al (2007) in a study conducted among 2 year old children in Netherlands, reported a non-significant association of asthma with air pollution related to NO_2 exposure from traffic (AOR 1.09, 95% CI 1.09 [0.82–1.45], which may be explained by the small sample size (n=981), and children were too young to have a reliable diagnosis of asthma, even though a significant association with wheeze (Adjusted OR 1.17, 95% CI 1.01– 1.36] was observed. Most of these epidemiological studies involve subjects exposed to air pollution mixtures generated by a variety of sources and pollutants, making it difficult to attribute the health effects uniquely to a particular pollutant. Nevertheless, in well-designed studies there are methods available to identify the most relevant sources and the most relevant pollutants.

Lung function in children has been negatively correlated with levels of total suspended particulates and black smoke (Roemer et al, 1993). A cross-sectional survey in Poland demonstrated reduced peak expiratory flow in children living in highly air polluted areas with increased exposure to dust, SO_2, NO_2 and lead compared to children from non-polluted areas (Mazur, 1995). In a study of winter type pollution (high SO_2 and moderate PM_{10} levels) on asthmatic Eastern European children, increased symptoms and reduced peak expiratory flow were reported with increased SO_2 exposures (Peters et al, 1996). Increased bronchial activity may result from inflammation caused by non-specific irritation of the airway (Landau, 1995). Indoor environmental factors known to modify asthma severity include pollutants such as PM, nitrogen oxides, second-hand smoke, and allergens from pests, pet fur, and mould (Diette et al, 2008). Mc Conell et al (2010) reported that asthma risk was increased with modelled traffic-related pollution exposure from roadways near homes (HR 1.51; 95% CI), 1.25–1.82), and near schools (HR 1.45; 95% CI, 1.06–1.98), suggesting that traffic related pollution exposure at

school and homes might contribute to the development of asthma which might increase the ventilation rate and dose of inhaled pollutants which in turn may have strong influence on the occurrence of asthma. A study conducted in North California reported that schools near busy highways had higher concentrations of oxides of nitrogen (NOx) and higher asthma prevalence (Kim et al, 2004). In a subsequent analysis of this data, the effect of school exposure was attenuated and was no longer significant after adjusting for modelled residential traffic-related exposure (Kim et al, 2008).

The mixture of pollutants in close proximity to roadways also includes transition metals and organic aerosols, which could be responsible for increasing risk of asthma. Increased incidence of respiratory symptoms with decrease in peak expiratory flow has been reported with elevated concentration of particulate matter (Ward and Ayres et al, 2004; Weinmayr et al 2010). Ward and Ayres (2004) demonstrated that for each increase of 1mg/m3 in $PM_{2.5}$, the pooled effect estimate for the PEF, based on a random effect model, was -0.144 l/min (95% CI: -0.243 - -0.044). Weinmayr et al (2010) reported that for each increase of 10 mg/m3 in PM_{10}, the authors observed a borderline significant decrease in PEF of -0.082 l/min (95% CI -0.214; 0.050).

Yamazaki et al (2007) reported that increased hourly concentration of $PM_{2.5}$ was associated with decrease in PEF among 17 hospitalised children aged 8 to 15 years who had been diagnosed with acute severe asthma and admitted at Shimoshizu National Hospital, Yotsukaido City, Japan. Liu L et al (2009) investigated the acute effects of air pollution on pulmonary function and airway oxidative stress and inflammation among 182 asthmatic children aged 9 to 14 years and demonstrated a significant decrement in small airway function and an increase in airway oxidative stress in asthmatic children in association with SO_2, NO_2, and $PM_{2.5}$ in ambient air. In this study, interquartile-range increases in 3-day average SO_2 (5.4 ppb), NO_2 (6.8 ppb), and $PM_{2.5}$ (5.4 $\mu g/m^3$) were associated with decreases in forced expiratory flow between 25% and 75% of forced vital capacity, with changes being −3.1% [95% confidence interval (CI), −5.8 to −0.3], −2.8% (95% CI, −4.8 to −0.8), and −3.0% (95% CI, −4.7 to −1.2), respectively. SO_2, NO_2, and $PM_{2.5}$ were associated with increases in thiobarbituric acid reactive substances (TBARS), a marker of oxidative stress, with changes being 36.2% (95% CI, 15.7 to 57.2), 21.8% (95% CI, 8.2 to 36.0), and 24.8% (95% CI, 10.8 to 39.4), respectively, after adjusting for co-pollutants and study periods in the model. Limiting sensitivity analyses to those that consistently demonstrated statistical significance in base

models, and considering only results showing a consistent pattern as actual effects adds further strength to this study.

Asthma medications used in the treatment of persistent asthma may modify the effects of different air pollutants on peak expiratory flow (PEF) suggesting different responses among asthmatic patients to ambient air pollutants (Qian et al, 2009; Thurston & Bates, 2003; Gent et al, 2003). This research further emphasises the need for understanding the interactions between air pollution and asthma medication interaction and its importance and role in better clinical management of asthma among children, as the scope of this problem is likely to grow in coming years. Although, contrary to this observation it was reported by Peters et al (1997) that use of medication in asthmatics was not a confounder as such, but attenuated the association between particulate air pollution and peak expiratory flow in asthmatics. An inverse relationship between PM_{10} and PEF has also been reported (Neas et al, 1995). Increased respiratory symptoms and bronchodilator usage with increased total suspended particles including PM_{10} has also been reported in preschool children (Braun- Farhlander et al, 1992; Romieu, 1996). Comparison of the effects of $PM_{2.5}$, and PM_{10} on hospitalizations of children for asthma was undertaken by two similar studies (Lin et al, 2002; Tecer et al, 2008). In both, there was significantly increased hospitalisation for asthma, after adjusting for climatic changes. However, Lin et al (2002), using bidirectional case-crossover and time-series analyses, in their study reported that coarse particulate matter ($PM_{10\text{-}2.5}$) averaged over 5-6 days was significantly associated with asthma hospitalization in both males and females. The magnitude of this effect appeared to increase with increasing number of days of exposure for most models, with the relative risk estimates stabilizing at about 6 days. Using a bidirectional case-crossover analysis, the estimated relative risks were 1.14 [95% confidence interval (CI), 1.02, 1.28] for males and 1.18 (95% CI, 1.02, 1.36) for females, for an increment of 8.4 microgram/m^3 in 6-day averages of $PM_{10\text{-}2.5}$, whereas the corresponding relative risk estimates were 1.10 and 1.18, respectively, with time-series analysis. The effect of $PM_{10\text{-}2.5}$ remained positive even after adjustment for the effects of the gaseous pollutants carbon monoxide (CO), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and ozone (O_3). Tecer et al (2008) reported that there was 18% rise in asthma admissions correlated with a 10-μg/m^3 increase in $PM_{10\text{-}2.5}$ on the same day of admissions. Burnett et al (1999) suggests that $PM_{10–2.5}$ was a better predictor for asthma hospitalizations, whereas $PM_{2.5}$ was a stronger predictor for respiratory infection.

Another study of African-American children with asthma in the Los Angeles area, observed that PM_{10} had a greater effect than $PM_{2.5}$ on daily occurrence of wheezing episodes among asthmatic children (Ostro et al, 2001). The association of asthma related increases in hospital admissions and emergency department visits with increased exposure to air pollution have been reported by many epidemiological studies (Wong et al, 2001; Stieb et al, 2000; Sunyer et al, 1997 and Villeneuve et al, 2007; Delfino et al, 2006).

Hansel et al (2008) in Baltimore estimated the effect of indoor nitrogen dioxide concentrations on asthma morbidity among 150 inner-city preschool children, while adjusting for indoor air pollutants. A care-giver completed questionnaire was used to assess asthma symptoms over the previous 2 weeks. The presence of a gas stove and the use of a space heater or oven/stove for heat were independently associated with higher NO_2 concentrations. Each 20-ppb increase in NO_2 exposure was associated with an increase in cough (RR = 1.10; 95% CI, 1.02–1.18), and nocturnal asthma symptoms (RR = 1.09; 95% CI, 1.02–1.16), after adjustment for potential confounders. NO_2 concentrations were not associated with increased visits to the outpatient department or hospital admissions. Higher indoor NO_2 concentrations were associated with increased asthma symptoms in preschool inner-city children. Interventions aimed at lowering NO_2 concentrations in inner-city homes may reduce asthma morbidity in this vulnerable population. Several pollutants have been shown to worsen asthma including particulate matter (PM), ozone, and nitrogen dioxide (Institute of Medicine Committee on the Assessment of Asthma and Indoor Air Report, 2000; Koren, 1995). NO_2 may be a particular problem in the inner city, with high indoor NO_2 concentrations in inner-city homes and where gas stoves are common and proper venting of stoves are unusual (Breysse et al, 2005; Diette et al, 2007). As preschool children spend much of their time in the home they may be especially at risk to the adverse effects of indoor NO_2 exposure (Klepeis et al. 2001; McCormack et al, 2008), although some investigators report that outdoor NO_2 increases risk of asthma in children (Belanger et al 2006; Garrett et al, 1998; Hasselblad et al, 1992; Kattan et al, 2007; Nitschke et al, 2006; Shima and Adachi, 2000; Smith et al, 2000).

Sulphur and nitrogen dioxides have been associated with decrease in lung function (Lebowitz et al, 1985), increased respiratory symptoms (Rutishauser et al, 1990) and increased bronchial reactivity in children. Devalai and colleagues (1994) demonstrated that a combination of NO_2 and SO_2, which could be encountered in heavy traffic, enhanced the airway response to inhaled allergens in adults. It has also been reported that inhalation of NO_2

and O_3 might result in airway inflammation and increased responsiveness in asthmatic adults. Frischer et al (1993) showed evidence of increased inflammation in asthmatic children exposed to increased levels of ozone. Several panel and population studies have reported negative correlations between lung function and atmospheric ozone levels in asthmatic and non-asthmatic children – decrease in PEFR with increase in ozone concentration -1.92 ± 5.77 (mL/s/ppb) (Lebowitz et al, 1985) and -2.99 ± 5.10 (mL/s/ppb) Lioy et al, 1985 (both ($p<0.05$). Increased levels of Ozone in combination with acid aerosols have also been associated with reduced lung function (Dockery et al, 1982). Ozone levels have been associated with respiratory symptoms and exacerbations of asthma (Ostro et al, 1994).

In contrast to these observations, Vedal et al (1987), Stebbing et al (1976) have demonstrated effects of O_3, SO_2, or NO_2 on lung function or on childhood respiratory symptoms. Dodge et al (1985) reported lower prevalence of wheeze and dyspnoea in a longitudinal study conducted in the period 1979 to 1982, among 343 children living in three towns of Arizona near to smelter works with increased levels of SO2 compared to those living in a non-polluted area. The annual average sulphur dioxide concentrations at two monitoring sites were 0.005 ppm and 0.04 ppm. However, increased prevalence of coughing was noted following exposure to high intermittent levels of sulphur dioxide with a peak 3 hour mean >2.5 mg/m3 (0.95 ppm) and average levels of particulates.

(2) Laboratory studies

Laboratory studies on the effects of individual pollutants have given conflicting results. Koenig et al (1990) showed SO_2 modified by O_3 can interact with pollen allergens to reduce the concentration of allergen needed to provoke an asthmatic response. This study was conducted in 8 male and 5 female patients aged 12 to 18 years and tested whether prior exposure to a low concentration of ozone (120 ppb) would condition airways in asthmatic subjects to respond to a sub-threshold concentration of sulphur dioxide (100 ppb). The pulmonary function measurements assessed were FEV_1, total respiratory resistance (RT), and maximal flow (Vmax50). Exposure to 100 ppb SO_2 after a 45-min exposure to 120 ppb O_3 caused a significant (8%) decrease in Forced Expiratory Volume (FEV1) ($p = 0.046$), a significant (19%) increase in Total Respiratory Resistance (RT) ($p = 0.048$), and a significant (15%) decrease in maximum flow (Vmax50) ($p = 0.008$) concluding that prior O_3 exposure increased bronchial hyper-responsiveness in these subjects such that they responded to an

ordinarily sub-threshold concentration of SO_2. Laboratory studies on effects of NO_2 either on its own or in combination with another pollutant, or along with other known triggers of asthma such as particulate matter have shown interesting results with regards to their association with asthma symptoms (Delfino et al, 2003; Gent et al (2003). Even though particles larger than 10 microns do not typically pass beyond the larynx, combination with other toxic air pollutants including NO_2 and hydrocarbons, along with mouth breathing may facilitate deposition of these particles in respiratory tract of children (Delfino et al, 2003). Exposure to outdoor air pollutants including ozone (O_3), particulate matter (PM) and hazardous air pollutants (HAPs), are also known risk factors for developing respiratory diseases, including asthma (Gent et al 2003). NO_2 has been reported to potentiate the effects of SO2 on respiratory function in chamber studies (Jorres and Magnussen, 1990).

B. Cough

Simoni et al (2003) assessed the effects of indoor air quality on respiratory health of school children living in Norway, Sweden, Denmark France and Italy using school based questionnaires and school environment assessment. They reported that exposure to particulate matter PM_{10} >50 $\mu g \cdot m^{-3}$ was significantly associated with dry cough at night, which was prevalent among children from poorly ventilated classrooms (OR 2.39, 95% CI 1.49–3.86). In this cross-sectional study school children exposed to CO_2 levels >1,000 ppm showed a higher risk for dry cough (OR 2.99, 95% CI 1.65–5.44). This was supported by observations from a follow up study conducted in six cities in United States, where a positive association was demonstrated between the occurrence of cough and increased levels of particulates change in daily cough reported for each 10 microgram/m3 increase in PM_{10} 8.6% (2.2% - 15.4%). (Dockery et al, 1989). Chronic cough and expectoration have been reported as common adverse respiratory symptoms with increased exposure to particulate matter (Nitta et al, 1993).

C. Breathlessness

In vitro studies of bronchial asthma have demonstrated bronchial epithelium exposure to 400 to 800 ppb NO_2 caused cell dysfunction and resulted in increased breathlessness (Devalia et al, 1994). A committee of the Environmental and Occupational Health Assembly of the American Thoracic Society (1996) concluded that indoor levels of NO_2 due to gas cooking were associated with increased risk of respiratory symptoms including breathlessness

and cough in children. Bentayeb et al (2013) investigated respiratory health effects of indoor exposures to aldehydes and VOCs among a representative sample of elderly French residents compared to other younger residents. They assessed the respiratory conditions especially breathlessness using a standardised questionnaire in 102 inhabitants aged over 15 years and twenty VOC's were objectively measured in 490 main dwellings. They reported a higher risk of breathlessness in the elderly exposed to indoor air pollution, mainly with toluene and o-xylene respectively AOR, 95% confidence interval: 3.36 (1.13, 9.98) and 2.85 (1.06, 7.68) in elderly, versus 0.91(0.59, 1.39) and 0.79 (0.47, 1.34) in the other group. There was a more pronounced effect of n-decane on breathlessness in the previous year observed in cases with poor dwelling ventilation. Increased breathlessness among school children was reported in schools located near to air pollution from traffic and industries (Nitta et al, 1993; Van et al, 1997).

D. Wheeze

A longitudinal follow up study of a cohort of 315 children with asthma aged 6-11 years in Fresno, California, using a respiratory questionnaire reported that NO_2, and PM_{10} were associated with increased risk of wheeze among asthmatic children (Mann et al, 2010). Wheeze was significantly associated with short-term exposures to NO_2 [odds ratio (OR) = 1.10 for 8.7-ppb increase; 95% confidence interval (CI), 1.02–1.20] and $PM_{10–2.5}$ (OR = 1.11 for 14.7-$\mu g/m^3$ increase; 95% CI, 1.01–1.22) and was more significant in children who were skin test positive to cat fur and common fungi, and was more prevalent among boys with mild intermittent asthma. Acute wheezing episodes were associated with increased levels of summer haze (with high SO2 and O3 content) (Buchadahl et al, 1996). Conversely, a study from Netherlands reported no relationship between reported respiratory symptoms and wide fluctuations in PM_{10} (11-135microgram/m3), or O_3 (14-114ppb) levels (Hoek and Brunekreef, 1995). Increased bronchial hyper-responsiveness was reported among children from highly polluted industrial areas compared to children from non-polluted areas in Hong Kong (Tam, 1994) which remained even after exclusion of children with a history of wheeze and diagnosed as asthma. The results demonstrated that studies on bronchial responsiveness can be used to assess the effects of air quality on the respiratory health of children and can be employed to measure impact of new air quality control measures.

E. Allergy, Allergic Rhinitis and Hay fever

Although an association of asthmatic symptoms in children has been reported in relation to air pollution, there are few studies addressing how this relates to atopy. Bjorksten et al (1980) considered month of birth was important in sensitization to birch pollen. In a study of Cedar tree pollen in Japan, hay-fever was reported by 1.7% of families living in rural mountainous areas, 5.1% living near forests with no traffic, 8.8% in areas near to forest, and 13.2% in those close to inner city roads. It was suggested that pollution from motor vehicles enhanced pollen sensitization (Ishizaki et al, 1987). A report from Barcelona (Anto et al, 1989) showed that outbreaks of asthma in the city in 1985 and 1986 coincided with the unloading of soya beans in the city harbour where the wind was blowing inland. Morgernstern et al (2008) assessed the relationship between individual-based exposure to traffic-related air pollutants and allergic disease outcomes among 2860 children aged 4 years, and 3061 children aged 6 years, in a prospective birth cohort study. Strong positive associations were found between the distance to the nearest main road and hay fever, eczema, with the highest odds ratios for children living less than 50 m from busy streets (OR, 1.41; 95% CI 1.22-1.80 for hay fever and OR 1.28; 95% CI 1.12- 1.54 for eczema). For $PM_{2.5}$ absorbance, significant positive associations were observed for hay fever (OR, 1.59; 95% CI, 1.11–2.27), and allergic pollen sensitization (OR, 1.40; 95% CI, 1.20–1.64). NO_2 exposure was associated with eczema, but there was no association with allergic sensitization (Morgernstern et al, 2008). This study does provide strong evidence for adverse effects of traffic-related air pollutants on atopic diseases as well as on allergic sensitization. However, as in most epidemiologic studies, looking at confounding factors was limited to the questionnaire derived variables and family income was not found to be a confounding factor.

Increased pollen sensitization in adults living close to major roads for more than 10 years was observed in Switzerland (Wyler 2000). Increased indices of air pollution were associated with childhood allergy in reports from Europe and Asia (Annesi-Maesano et al. 2007; Hajat et al, 2001; Hwang et al, 2006; Janssen et al, 2003; Krämer et al, 2000; Lee et al, 2003; Morgenstern et al, 2007, 2008; Pénard-Morand et al, 2005; Yu et al, 2005), although this was not found by others (Nicolai et al, 2003; Ramadour et al, 2000). Allergic symptoms secondary to air pollution could depend on seasonal variations in pollen varieties and local flora (Blaiss, 2004). Mutius et al, (1992) reported an increase in incidence of hay fever and seasonal allergic rhinitis after the reunification of Germany, and concluded that this resulted

from increased exposure to traffic pollution. The prevalence of hay fever (2.3% [34/1454] vs 5.1% [115/2252], p<0.0001) and atopic sensitisation (19.3% [252/1303] vs 26.7% [434/1624], p<0.0001) increased significantly during this period between 1991-92 and 1995-96. There was no significant change in the prevalence of asthma, asthma-related symptoms, or bronchial hyper-responsiveness.

Higher incidence of allergies (27.4%) and allergic rhinitis (20.1%) were reported with increased O_3 exposure among 6672 children aged 9-11 years recruited randomly from 108 schools in six French cities (AOR=1.34; 95% CI=1.24-1.46 and AOR 1.09 95% CI (1.00–1.19) after adjusting for exercise induced bronchial hyperactivity (EIB), lifetime allergic rhinitis (AR) and asthma, (Pénard-Morand et al, 2005). Another study by Hajat et al (2001) among children, which looked into the increase in consultations for allergic rhinitis related to air pollution between 1992- 1994 in London, United Kingdom, reported a 10th--90th percentile increase in sulphur dioxide (SO_2) levels 4 days prior to consultation (13-31 microgram/m3 was associated with a 24.5% increase in consultations (95% confidence interval: 14.6, 35.2; $p < 0.00001$); a 10th-90th percentile increase in averaged ozone (O_3) concentrations on the day of consultation and the preceding 3 days (6-29 parts per billion) was associated with a 37.6% rise (95% confidence interval: 23.3, 53.5; $p < 0.00001$). Incidence of childhood allergic rhinitis was higher in those residing in areas with heavy traffic and higher automobile exhaust emissions (Miyamoto et al, 1997). Although this study data did not distinguish between first and subsequent consultations, the increases were likely to indicate an exacerbation of symptoms by pollutants rather than an initiation of allergies. Another German study conducted among 170 school children showed that although there was an initial increase in allergic responses associated with increased levels of O3, with chronically high levels of O3 there was reduced allergic responsiveness suggesting an adaptive response (Kopp et al, 1999).

Allergic sensitisation and increased hay fever incidence and exposure to $PM_{2.5}$ was observed among young German children (Morgenstern et al, 2007). Other studies on associations between air pollution especially from traffic exposure and increased incidence of allergic problems also have been reported from Germany (Duhme et al 1996; Weiland et al, 1994), Thailand (Pothikamjorn et al, 2002), and from Taiwan (Lee et al, 2003). A study of Norwegian school children showed no associations between traffic-related pollutants, including $PM_{2.5}$, and allergen sensitization. One of the reasons considered was related to the low level of pollution in Oslo (Oftedal et al, 2007). Von Mutius et al (1998) reported reduced

risk of hay fever, atopy, and bronchial hyper responsiveness (BHR) in 9-11 year old children living in homes with wood or coal stoves and heating, compared to children from homes where gas or another fuel was used. Tunnicliffe et al (1994) observed NO_2 inhalation enhanced responses to inhaled house dust mite allergen in subjects with mild asthma. These studies along with their odds ratios related to different air pollutants have been summarised in the table 2.06.

Table 2.06 Studies reporting odds ratio for allergy in relation to different air pollutant exposures

Reference	Years	Country	Age (years)	Sample size	Air pollutant	Allergic symptom	Odds ratio (95%CI)
Morgenstern et al, 2007	1999-2000	Germany	0-2	9568	$PM_{2.5}$	Hay fever	1.16 (1,01-1.34)
Oftedal et al, 2007	2001-2002	Norway	9-10	2244	NO_2	Allergy	1.88 (1.02-3.47)
Pothikamjorn et al, 2002	1999	Thailand	15-18	290	PM_{10}	Allergy	2.21 (1.13-4.21)
Lee et al 2003	1995-1996	Taiwan	12-14	1,139,452	PM_{10}	Allergic rhinitis	1.17 (1.02-1.29)
Duhme et al 1996	1994-1995	Germany	12-15	3703	PM_{10}	Allergic rhinitis	1.71 (1.36-2.15)
Weiland et al, 1994	1991	Germany	10-12	2050	PM_{10}	Allergic rhinitis	1.67 (1.17-2.38)
Von Mutius et al, 1998	1995-1996	Germany	9-11	2664	NO_2	Hay fever	2.20 (1.51-3.32)

F. Pneumonia

Respiratory disorders including asthma and pneumonia have been reported commonly in children from schools located near to areas exposed to higher levels of air pollution from industrial and traffic sources (Rizwan et al, 2004). Prolonged exposure to increased levels of particulates, rather than nitrogen dioxide or ozone, have been associated with higher incidence of pneumonia in children (Negrisoli and Nascimento, 2013) and increased mortality in adults and with increased cough and decreased lung function in exposed individuals (Beelan et al, 2014). Beelan et al (2014) in their study as part of the multicentre European Study of Cohorts for Air Pollution Effects (ESCAPE), investigated the association between natural cause mortality and long term exposure to several air pollutants using data from 22 European cohort studies, which created a total study population of 367,251 participants. 29,076 died from a natural cause during follow-up and a significantly increased hazard ratio (HR) for $PM_{2.5}$ of 1.07 (95% CI 1.02-1.13) per 5 μg/m3 was recorded (HR quoted in relation to deaths among the participants exposed to air pollutants). HRs for $PM_{2.5}$ remained significantly raised even when participants exposed to pollutant concentrations lower than the European annual mean limit value of 25 μg/m3 (HR 1.06, 95% CI 1.00-1.12), or below 20 μg/m3 (1.07, 1.01-1.13) were only included. Long-term exposure to fine particulate air pollution was associated with natural-cause mortality; even within concentration ranges well below the present European annual mean limit value.

The correlation between wood fuel and respiratory problems has been well documented in studies from the developing world. A study conducted by the World Health Organisation has reported that pneumonia risk for young children below five years increased 80% with exposure to combustion of solid, unprocessed fuels (Duhme et al, 1996). A study on three child cohorts (N=1611) from Spain (Barcelona and Menorca), and the United Kingdom (Ashford), investigated the relationship between indoor NO_2 levels and incidence of lower respiratory tract infection (LRTIs) in the first year of life (Sunyer et al, 2004). No correlation was observed between NO_2 and lower respiratory tract infection in children suggesting that the effects observed in studies on outdoor air are probably due to other co-pollutants.

G. Bronchitis

Von Mutius et al (1998) reported that long term exposure to high levels of particulates and SO_2 in the former East German city of Lepzig was responsible for a two-fold higher childhood bronchitis prevalence compared to that observed in Munich. This was supported by data from six cities in United States which showed an association between chronic cough and bronchitis and lower particulate exposure (Dockery et al, 1989; Nitta et al, 1993). Molfino et al (1992) and Wang et al (1997) reported that asymptomatic children living in highly polluted areas from industrial sources had increased bronchial responsiveness compared to children from non-polluted areas. In contrast, Ware et al (1986) observed no association between bronchial responsiveness and pollution, even though the source of pollution was from motor traffic.

2.3.6.2 Other health problems

A. Cardiovascular

Exposure to air pollution caused by particulate matter (PM) and its association with heart disease, even at lower concentrations has been reported from a number of UK studies (Ayres, 2006; Brook et al, 2010). Patients with ischaemic heart disease (IHD) may be more susceptible to adverse health effects of PM exposure (Sacks et al, 2011). Sughis et al (2012) examined this possibility in a cross-sectional relationship of blood pressure and particulate air pollution in 8 - 12 year old school children in Lahore, Pakistan and reported that traffic related air pollution was associated with higher systemic and diastolic blood pressure. The authors considered this might be clinically relevant for adult hypertension. A study from Los Angeles suggested a role for air pollution in intima media thickening of the carotid artery (Künzli et al, 2005), and progression of intima media thickness and traffic proximity (Künzli et al, 2010). Coronary calcification has been attributed to residence near to heavy traffic movement (Hoffmann et al, 2007).

A study on the effect of long term exposure to airborne pollutants on acute coronary events including heart attack and unstable angina in 11 cohorts participating in the European Study of Cohorts for Air Pollution Effects (ESCAPE) was carried out by an international team of researchers coordinated by the University of Utrecht (Cesaroni et al 2014). This study

involved over 100,000 people with no history of heart disease enrolled from 1997 to 2007 and was followed for an average of 11.5 years. The researchers found that after taking account of several other risk factors, including other illness, smoking, and socioeconomic factors, 5 μg/m3 increase in $PM_{2.5}$ particulate matter was associated with a 13% increased risk of coronary events and a 10μg/m3 increase in PM_{10} particulate matter was associated with a 12% increased risk of coronary events. The results support lowering of the EU limits for particulate matter air pollution. In the European Union the current annual limit for particulate matter with a diameter of 2.5 micrometers (μm) or less (known as $PM_{2.5}$) is 25 μg/m3, which is far above that implemented in the United States (12 μg/m3). A 2013 study found average $PM_{2.5}$ concentrations over a five year period in Beijing were more than 10 times the World Health Organization air quality guideline value of 10 μg/m3 (Guo et al 2013).

Synergistic interaction between pollutants and aero allergens may result in enhanced passage of antigen across the epithelium when particles deposit on airways. Inflammation resulting from penetration of very fine particles could be associated with increased blood viscosity and coagulability which might influence mortality and morbidity risk from cardio vascular diseases. Irritation of the cardiovascular system induced by photochemical smog related to air pollution was reported by Cheng et al (2003). An adverse effect of traffic exhaust on risk for myocardial infarction has been reported (Peters et al, 2004; Chen and Guo 2005). Shah et al (2013) conducted a systematic review and meta-analysis and reported an association of heart failure hospitalisation, or death, with increased exposure to particulate matter ($PM_{2·5}$ 2·12% per 10 μg/m3, 95% CI 1·42—2·82; PM_{10} 1·63% per 10 μg/m3, 95% CI 1·20—2·07), with a stronger association observed on the day of exposure, and with more persistent effects for $PM_{2.5}$.

B. Neuro-behavioural

There are few studies on the neurobehavioral effects of air pollution in children. Lead exposure results in acute or chronic damage to the nervous system resulting in neuro-behavioural problems in children (Bellinger et al, 2008), with neuro-pathological changes including brain oedema, herniation and atrophy in lead intoxicated children and with white matter degeneration and volume reduction of cortical grey matter in exposed adults (Stewart et al, 2006). Effects on later intellectual development are the greatest cause of concern. Prenatal and perinatal exposure to air pollution may also be associated with impaired infant mental

development (Guxens, 2011). A study of Chinese children aged 8–10 years reported significant associations between traffic-related air pollution and neuro-behavioural function (Wang, 2007). Ultrafine particulates may directly influence the brain through lung absorption, or via the olfactory nerves (Power et al, 2011). Early life exposures to lead, mercury and polycyclic aromatic hydrocarbons (PAH) at current environmental levels can result in chronic neurotoxicity (Granjeal et al, 2006). Exposure to severe air pollution has been associated with increased levels of neural cyclo-oxygenase (CO_{X-2}) and accumulation of the 42 amino acid form of beta amyloid, an important known cause of neuronal dysfunction (Calderón-Garcidueñas et al, 2004). Associations between nitrogen dioxide levels and a range of cognitive and motor abilities were reported in 250 Spanish children (Freire et al, 2010). Mechanisms implicated include inflammation or oxidative stress (Power et al, 2011; Fonken et al, 2011). Wang et al (2009) examined traffic-related air pollution exposure and neurobehavioral function among 928 primary school children aged 8 to 10 years in Quanzhou, China. A positive association between chronic low-level traffic-related air pollution exposure and neurobehavioral function was shown.

C. Otitis Media

There is growing evidence that ear problems are linked with air pollution. Animal studies have observed impairment of ciliary function and middle ear mucous secretion with sulphur dioxide exposure (Ohashi et al, 1989b). Data from two cohorts of approximately 4350 infants from Germany and Netherlands reported significant associations between ambient air pollutants and otitis media (Brauer et al, 2006). In contrast, Heinrich and Raghuyamshi (2004) concluded it was difficult to confirm an association of air pollution with childhood otitis media. Other studies have reported a positive association (Karmaus et al 2001; Heinrich et al, 2002). The effects of air pollution on the upper respiratory tract are likely to directly impact on otitis media (Koltai et al, 1994).

These studies along with their odds ratio for otitis media related to different air pollutants are summarised in table 2.07.

Table 2.07 Studies reporting odds ratio for otitis media in relation to different air pollutant exposures

Reference	Study period	Country	Age (years)	Sample size	Air pollutant	Odds ratio (95%CI)
Brauer et al 2006	1997-1998	Germany	0-1	650	$PM_{2.5}$	1.14 (0.87–1.49)
Brauer et al 2006	1997-1998	Netherlands	0-1	3700	$PM_{2.5}$	1.13 (1.00–1.27)
Karmaus et al 2001	1997	Netherlands	0-3	340	Hydrocarbons	3.71 (1.10-12.56)
Heinrich et al 2002	1995-1996	Germany	5-11	7000	SO2	1.42 (0.94-2.15)
Jensen et al 2013	2007-2009	Greenland	4-10	223	Hydrocarbons	2.47 (1.45–4.21)
MacIntyre et al 2011	1999-2000	British Columbia	0-2	45513	NO	1.10 (1.07-1.12)
Zemek et al 2010	1992-2002	Canada	1-3	14527	O3	1.12 (1.03–1.21)

D. Dermatological problems

Increased dermatological problems associated with living in areas with high bio-aerosol exposure and with prolonged indoor storage of organic waste were found to be more pronounced in atopic children (Herr et al, 2004). Intact skin acts as a protective barrier to toxins from air pollution and microorganisms, with the outer layer epidermis shedding off contaminated surface cells. The production of collagen and elastin due to exposure to ultra violet radiation causes skin to thin reducing elasticity and increasing vulnerability to infection and possibly toxins. The role of environment contaminants in the pathophysiology of atopic dermatitis was reported in 425 children from 9 kindergartens in Korea where improvement measured by eczema area and severity index (EASI) after an intervention programme suggested that there was an association between atopic dermatitis and exposure to air pollution mainly PM_{10} and CO2 (Kim et al, 2013)

E. Ophthalmological problems

There is evidence that household air pollution is associated with major blinding and painful eye conditions in adults and children (West et al, 2013). A link between cataracts and bio-smoke condensates has been reported (Kim et al 2011). An additional effect of high ambient temperature may be implicated through denaturation of lens protein (Okuno et al, 1991). Acute macular degeneration (AMD) has been associated with increased exposure to air pollution by immune regulatory system resulting in dysregulation of the immune system and related ocular changes. (Donoso et al, 2006; Zipfel et al, 2010; Banerjee et al, 2012). Inconsistent findings on tear breakup time and dry eye symptoms were reported in relation to indoor airborne dust (Wolkoff et al 2013). Particulate matter and carbon monoxide exposures have been associated with Meibomian gland dysfunction (Malerbi et al, 2012). Exacerbation of infectious diseases such as trachoma, trichiuriasis and other corneal and conjunctival infections in areas with increased air pollution is reported (Malerbi et al, 2012; West et al, 2013).

F. Pregnancy outcomes

A decrease in mean birth weight with increasing pregnancy air pollutant exposures has been reported with the largest effects with exposure to particulate matter and other gaseous air

pollutants throughout pregnancy (Wilhelm et al, 2005; Parker et al, 2005). These associations have been related to increased $PM_{2.5}$ (Basu R, 2004), as well as carbon monoxide and ozone (Salam et al, 2005; Ezzaine, 2013). Romao et al (2013) analysed exposure to PM_{10} and low birth weight in Santo André, São Paulo State, Brazil. Babies born between 2000 and 2006 were included and data on daily PM_{10} levels obtained from the São Paulo State Environmental Agency. There was a dose response association between PM_{10} concentrations and low birth weight, which was more pronounced among babies born to mothers exposed to the highest quartile of PM_{10} (37.5 microgram per metre cube) in the third trimester. The increased risk of low birth weight was by 26% (OR: 1.26; 95% CI: 1.14-1.40) compared to that for the first quartile. Stieb et al (2012) examined the association of low birth weight (LBW) with outdoor air pollution after including sixty two studies which were identified using searches of bibliographic databases and reference list of relevant papers. Pooled estimates of decrease in birth weight ranged from 11.4 g (95% confidence interval −6.9–29.7) per 1 ppm CO to 28.1 g (11.5–44.8) per 20 ppb NO_2, and pooled odds ratios for low birth weight ranged from 1.05 (0.99–1.12) per 10 $\mu g/m^3$ $PM_{2.5}$ to 1.10 (1.05–1.15) per 20 $\mu g/m^3$ PM_{10} based on entire pregnancy exposure.

Even though many studies have investigated the association between air pollution exposure during pregnancy and preterm birth outcome, the results have been inconsistent between different studies from different countries. A study conducted in Iran by Janghorbani and Piraei (2013) reported that maternal exposure to ambient air pollution during the entire pregnancy was associated with preterm birth in Isfahani women. 4758 consecutive singleton birth records from one large referral hospital (2010-2012) in Isfahan, Iran were identified along with the cases of preterm birth and LBW, which were then combined with meteorological and air pollution monitoring data. The effect of air pollution exposure during the entire pregnancy, each trimester, and last month, and preterm birth (gestational age <37 weeks) and LBW (<2500 g) were estimated by Pollutant Standard Index (PSI) using logistic regression adjusted for gestational age, neonate gender, birth order, and mother's age. The results showed that the PSI for entire pregnancy was significantly associated with preterm birth [Odds Ratio (95% CI) = 1.26 (1.20, 1.33)], even though there was no association between maternal exposure to ambient air pollution and each trimester and the last month of pregnancy, and preterm birth or LBW.

Fleisch et al (2014) examined the association of outdoor $PM_{2.5}$ with adverse birth outcomes among 22 countries included in the World Health Organization Global Survey on

Maternal and Perinatal Health from 2004-2008. After adjusting for seasonality, $PM_{2.5}$ was not associated with preterm birth, but was associated with low birth weight [OR 1.22; 95% CI: 1.07- 1.39) with the fourth quartile of $PM_{2.5}$ (> 20.2 μg/m^3) compared with the first quartile (< 6.3 μg/m^3)]. In contrast in China, the country with the largest $PM_{2.5}$ range, preterm birth and low birth weight both were associated only with the highest quartile of $PM_{2.5}$, suggesting a possible threshold effect (OR = 2.54; CI: 1.42, 4.55 and OR = 1.99; CI: 1.06, 3.72 for preterm birth and low birth weight, respectively, for $PM_{2.5} \geq 36.5$ μg/m^3 compared with $PM_{2.5} < 12.5$ μg/m^3.

2.3.7 Mechanisms underlying adverse health outcomes from air pollution

Mechanisms underlying the effects of particulates on human healthcare are incompletely understood. NO_2 may increase permeability of bronchial mucosa to allergens lowering the threshold for sensitization (Devalia et al, 1994) leading to allergic sensitisation. There is emerging evidence that the toxic effects of new photochemical pollutants such as NO_2 are likely to be related to respiratory infection in exposed individuals (Chauhan and Sebastian, 2003). These authors propose that exposure to air pollution could act synergistically to cause respiratory illnesses especially in exacerbating symptoms in individuals with pre-existing respiratory conditions such as asthma and chronic obstructive pulmonary disease. Toxins could act directly as well as through interactions with other pollutants. Allergic rhinitis may be influenced by air pollution through different mechanisms. Nasal congestion with mast cells and lymphocytes in nasal lavage fluids occurs with SO_2 exposure, and increased nasal lavage levels of histamine, neutrophils, eosinophils and mononuclear cells can occur with increased ozone exposure, and upper airway irritation with formaldehyde. The link between otitis media may result from air pollutants disrupting muco-ciliary clearance causing Eustachian tube dysfunction (Bluestone and Klein 1983). Reduced muco-ciliary clearance may result in a greater predisposition to upper respiratory viral illnesses and OM (Heikkinen et al, 1999). Pollutants may also result in adenoidal hyperplasia, with narrowing of Eustachian tubes. Lastly, pollutants might directly cause mucosal swelling of the Eustachian tube (Ohashi et al, 1989a, 1989b). The mechanisms are summarised in table 2.08.

Table 2.08 Mechanisms of outcomes related to air pollution

Outcome	Air pollutant	Mechanism	Reference
Allergy	NO_2	Increase permeability of bronchial mucosa to allergens lowering the threshold for sensitization	Devalia et al, 1994
Respiratory infection	NO_2	Toxic effects of new photochemical pollutants	Chauhan and Sebastian, 2003
	PM_{10}	Direct and indirect effects of toxins present in particulate matter	Chauhan and Sebastian, 2003
Allergic Rhinitis	SO_2	Nasal congestion with mast cells and lymphocytes in nasal lavage fluids	Koltai et al, 1994
	Ozone	Increased nasal lavage levels of histamine, neutrophils, eosinophils and mononuclear cells	Koltai et al, 1994
URTI	Formaldehyde	Upper airway irritation by chemical reaction	Koltai et al, 1994
	PM_{10}	Reduced muco-ciliary clearance	Heikkinen et al, 1999
Otitis Media	PM_{10}	Disrupting muco-ciliary clearance causing Eustachian tube dysfunction	Bluestone and Klein, 1983
	PM_{10}	Pollutants might directly cause mucosal swelling of the Eustachian tube adenoidal hyperplasia, with narrowing of Eustachian tubes	Ohashi et al, 1989a
Cardiovascular Disease	$PM_{2.5}$	Increased oxidative stress, Increased blood viscosity, Increased coagulability	Du et al, 2016 Brook et al, 2010
	$PM_{2.5}$	Coronary calcification	Hoffman et al, 2007
	$PM_{2.5}$	Increased heart failure	Shah et al, 2013
	Traffic related PM	Increased systolic and diastolic blood pressure, intima media thickening carotid artery	Kunzli et al, 2005

2.3.8. Biomarkers and genetic effects of air pollution

These are related to characterisation of exposure risks. A dose response association has been reported between external measures of air pollution and increased levels of DNA adducts and markers of early damage, including mutagenicity (Vineis et al, 2005). Biomarker studies on mutagenicity and cytogenetic effects in humans have shown elevated urinary mutagenicity in Salmonella assay among non-smoking bus drivers exposed to polluted urban air, mainly traffic exhausts, as compared with mail carriers working in the same city (Hansen et al, 2004). Somatic HPRT (Hypoxanthine-guanine-phospho-ribosyl-transferase) mutation frequencies and aromatic DNA adducts were correlated in cord blood samples from polluted industrial areas in Poland suggesting heavy exposure to air pollutants especially in urban areas (Perera et al, 2002). Fractional exhaled nitric oxide (FeNO) has been used as a non-invasive biomarker to investigate exposure to air pollutants and airway inflammation, and asthma (Delfino et al, 2006; Mar et al, 2005; Spanier AJ, 2009). A prospective study evaluated the association of NOS polymorphisms and environmental exposures with FeNO (fractional exhaled nitric oxide) levels among 225 tobacco smoke exposed children with asthma, and observed an interaction of the NO and SO_3 polymorphism with airborne nicotine concentration and FeNO level ($p=0.01$). Sensitive individuals with asthma have higher levels of FeNO when exposed to indoor allergens (Morgan et al, 2004; Franklin et al 2003; Simpson et al 1999; Roberts et al, 2004). Reduced FeNO levels have been associated with smoking (Verleden et al, 1999; Kharitonov et al, 1995), whereas atopy was associated with increased FeNO levels (Barretto et al, 2001; Yates et al, 2001).

Several in vitro and in vivo studies using different methodologies have provided evidence for the geno-toxic effects of air pollution (Vineis et al, 2005; Peluso et al, 2008 Kawanishi et al, 2009; Coronas et al, 2008). Organic compounds, especially polycyclic aromatic hydrocarbons (PAH) present in extracts of a particulate matter, might induce oxidative damage to DNA. A systematic review of thirty five studies, with sample sizes of >10 subjects, examined whether metabolites of pyrene and DNA adducts were valid markers of low level environmental exposure to PAHs and reported that PAH metabolites, and to a lesser extent PAH–DNA adducts, correlated well at the group level with exposure to benzo-pyrene, even at low levels of air pollution (Castano Vinyals et al, 2004). A study conducted in Denmark, measured personal $PM_{2.5}$ and black smoke exposure in 50 students four times over one year and analysed biomarkers of DNA damage. It was reported that personal $PM_{2.5}$

exposure was found to predict 8-oxo-dG in lymphocyte DNA with an 11% increase in 8-oxo-dG/10 μg/m3 increase in PM exposure (P = 0.007) (Sorensen et al, 2003). There is mounting evidence from studies in several countries indicating that air pollution contributes to epigenetic variation in several genes, which in turn could contribute to disease susceptibility (Patricia et al, 2012; Herceg et al, 2011; Hou et al, 2011; Bollati et al, 2010; Weidman et al 2007; Faulk et al, 2011; Chaudari et al, 2010).

2.3.9. Air Quality Index (AQI) and Air Quality Health Index (AQHI)

Air quality is defined as a measure of the condition of air relative to the requirements of one or more biotic species, or to any human need or purpose and denotes a number used by government agencies to communicate to the public how polluted the air is currently, or how polluted it is forecast to become (Johnson et al, 1997). Computation of AQI requires an air pollutant concentration from a monitor or model. The function used to convert from air pollutant concentration to AQI varies by pollutant, and is different in different countries. Different countries have their own air quality indices which are not all consistent and different names for their indices such as Air Quality Health Index, Air Pollution Index and Pollutant Standards Index are being used (Johnson et al 1997, Zou et al 2014; Chen et al 2013). The Air Quality Health Index (AQHI) represents an aggregate measure of the outdoor air quality and measures mainly concentrations of particulate matter, ground-level O_3, SO_2, CO_2 and NO_2. The Air Quality Health Index or (AQHI) is a scale designed to help to understand the impact of air quality on health and is used for Canada and Hong Kong. It is a health protection tool used to make decisions to reduce short-term exposure to air pollution by adjusting activity levels during increased levels of air pollution and also provides a basis for advice on how to improve air quality by proposing behavioral changes to reduce the environmental footprint. The AQHI provides a number from 1 to 10+ in order to indicate the level of health risk associated with local air quality. This number might exceed 10 sometimes, when there is abnormally high air pollution and the health is at greater risk with increase in this number and health precautions need to be taken. The categories and values for this index are low risk (1-3), moderate risk (4-6), high risk (7-10 and very high risk (10+).

In comparison to the AQHI, the index used in UK is AQI. The AQI can rise (deteriorating air quality) due to several factors including: lack of dilution of air pollutants, stagnant air, often caused by temperature inversion, or low wind speeds allowing air pollution

to remain localised, with high pollutant concentrations and hazy conditions. A larger percentage of the population is likely to experience adverse health effects as the AQI increases.

Values of the Air Quality Index greater than 100 are considered to be unhealthy for vulnerable groups including those with heart or lung disease, older adults, and children, and for the total population with high values (Environmental Protection Agency 2009). The AQI will vary between different locations based on air concentrations of different pollutants and because of changing weather conditions. While both have their own respective strengths, the new index AQHI has been designed to tell us more about the health risks from air pollution on any given day. AQI provides us information of the quality of the air compared to the Canada AQHI standards and criteria for pollution. AQHI reflects the current knowledge of health effects associated with air pollution and AQI is based on the specific level of air pollution of individual air pollutamts. AQI based on the air quality standards take into consideration both the environmental and human health effects.

The most commonly used Air Quality Index in the UK is the Daily Air Quality Index recommended by the Committee on Medical Effects of Air Pollutants (COMEAP, 2015). This is a ten point system grouped into 4 bands: low, moderate, high and very high. Each band comes with advice for at-risk groups and the general population (DEFRA, 2009) (Table 2.09)

Table 2.09 Air pollution banding and health messages for at-risk individuals and general population

Pollution band	Value	Health messages for at-risk individuals	Health messages for general population
Low	1–3	Enjoy your usual outdoor activities.	Enjoy your usual outdoor activities.
Moderate	4–6	Adults and children with lung problems, and adults with heart problems, who experience symptoms, should consider reducing strenuous physical activity, particularly outdoors.	Enjoy your usual outdoor activities.
High	7–9	Adults and children with lung problems, and adults with heart problems, should reduce strenuous physical exertion, particularly outdoors, and particularly if they experience symptoms. People with asthma may find they need to use their reliever inhaler more often. Older people should also reduce physical exertion.	Anyone experiencing discomfort such as sore eyes, cough or sore throat should consider reducing activity, particularly outdoors.
Very high	10	Adults and children with lung problems, adults with heart problems, and older people, should avoid strenuous physical activity. People with asthma may find they need to use their reliever inhaler more often.	Reduce physical exertion, particularly outdoors, especially if you experience symptoms such as cough or sore throat.

The index is calculated based on the concentrations of five pollutants: Ozone, NO_2, SO_2, $PM_{2.5}$ and PM_{10}. The breakpoints between index values are defined for each pollutant separately and the overall index is defined as the maximum value of the index. Different averaging periods are used for different pollutants (DEFRA, 2014) (Table 2.10).

Table 2.10 Daily Air Quality Index recommended by the Committee on Medical Effects of Air Pollutants (COMEAP)

Index	Ozone, Running 8 hourly mean (µg/m^3)	Nitrogen Dioxide, hourly mean (µg/m^3)	Sulphur Dioxide, 15 minute mean (µg/m^3)	PM_{10} particles, 24 hourly mean (µg/m^3)	$PM_{2.5}$ particles, 24 hour mean (µg/m^3)
1	0-33	0-66	0-88	0-11	0-16
2	34-65	67-133	89-176	12-23	17-33
3	66-99	134-199	177-265	24-34	34-49
4	100-120	200-267	266-354	35-41	50-58
5	121-140	268-334	355-442	42-46	59-66
6	141-159	335-399	443-531	47-52	67-74
7	160-187	400-467	530-708	53-58	75-83
8	188-213	468-534	709-886	59-64	84-91
9	214-239	535-599	887-1063	65-69	92-99
10	≥ 240	≥ 600	≥ 1064	≥ 70	≥ 100

(Source "Review of the UK Air Quality Index". COMEAP & Air UK Website DEFRA 2014)

A study conducted by Chen et al (2013) investigated associations between the AQHI and emergency department (ED) visits for acute ischemic stroke to validate the AQHI as a predictor of risk of morbidity from stroke. This study which examined ED visits showed that NO_2 and CO had significant positive associations with the number of ED visits for ischemic stroke during April-September. The association was stronger among those 75 years of age and older. In this age range the odds ratios (95% CI) for an interquartile range increase of AQH in 1-24 hours, 25-48 hours and 1-72 hours lag periods were 1.23 (1.08-1.40), 1.15 (1.01-1.31) and 1.30 (1.10-1.54) respectively. Similar results were observed for NO_2 and CO suggesting that AQHI may be used as a valid communication tool for air pollution morbidity effects related to stroke.

2.4 UK National Air Quality Strategy

The development of a strategy to address areas of poor and declining air quality in order to reduce risks to health, and for attaining wider objectives of sustainable development in relation to air quality in United Kingdom was included as part of the Environmental Act of 1995. In March, 1997 the National Air Quality Strategy was published as a response to this act with commitments to achieve new air quality objectives throughout the UK by 2005. This was followed by a review of the Strategy which in turn led to the publication of an Air Quality Strategy for the UK in January 2000. The Air Quality Strategy provides a framework for improving air quality through a clear policy framework using realistic objectives, regulation, financial incentives, monitoring and research (DEFRA, 2000). This framework will be helpful in achieving cleaner air that will bring health and social benefits to all individuals through public awareness and motivation. COMEAP (Committee on the medical effects of air pollution in UK), is an expert Committee that provides advice to government departments and agencies, through the Department of Health's Chief Medical Officer, on all matters concerning the effects of air pollutants on health (COMEAP, 2013).

2.5 Air pollution and smoking and related toxicology

Smoking and air pollution are inter related. Smoking can contribute to air pollution by releasing tiny particles, which could contribute directly to air pollution either as particulate matter or chemical substances released into the atmosphere or both causing changes in the air composition and mixing with each other and resulting in air pollution indirectly. However,

differentiating between air pollution and smoking can be challenging due to presence of similar toxins released from different sources of smoking and air pollution, which in turn might depend on the type of cigarette smoked and also the source of air pollution including traffic and industrial sources, which makes it difficult to measure the biomarkers specific to combined air pollution and smoking. Since some of the particles from air pollution or smoking source are invisible, this may cause further difficulties, when measuring the level of air pollution and smoking. Furthermore, the pollution caused by air pollutants or smoking could further be modified by the humidity and temperature, which also depends on the climatic changes and the direction of wind.

Although there have been various guidelines, government laws and policies which have helped to reduce the exposure of the population to tobacco smoking, nearly forty percent of children all over the world are exposed to tobacco smoke in their homes leading to indoor air pollution (Oberg et al, 2011). Reports have shown that air pollution due to cigarette smoking results from specific release of toxic particles less than 2.5 micro-meters, which forms major components of cigarette smoke, and a cigarette usually emits between 7 and 23 milligrams (mg) of $PM_{2.5}$ depending on the brand of cigarette smoked and manner of smoking (James, 2007; Martin et al, 1997). Particulate matter especially $PM_{2.5}$ has been reported to be a very well established marker of air pollution resulting from environmental exposure to tobacco smoke and helps to measure it's concentration and to assess the air pollution due to second hand smoke in work places, hospitality places and residential areas (Connolly G et al, 2009; Butz et al, 2011; Hartyunyan et al, 2013; Valente et al, 2007; Eakin et al, 2014; Semple et al, 2012; Lanphear et al, 2011; Wilson et al 2013). But the disadvantage of using $PM_{2.5}$ as a biomarker is that it can be nonspecific in measuring air pollution due to tobacco smoking which could result sometimes from varied sources of $PM_{2.5}$ and the possibility of mixing of different particles from various sources including industrial and traffic sources. Interestingly, another biomarker air nicotine has been reported to be more specific indicator for tobacco smoke related air pollution and for effective measurement of its levels in the environment using passive nicotine dosimeters. Kelishadi et al (2014) in their cross sectional study conducted among 100 pre-pubescent (8-12 years) children in 2011 in Isfahan, the second largest and second most air polluted city in Iran reported that particulate matter concentration had a significant independent association with biomarkers of endothelial dysfunction and inflammation measured by C–reactive protein and nitric oxide (NO). The main finding was that regardless of passive smoking, PM_{10} concentration had a significant independent association with serum CRP and was inversely associated with NO levels.

The variation in the levels of $PM_{2.5,}$ which can be monitored using light or sound has been reported to be a promising approach for motivating behavioural changes, as it is quite acceptable and helps indirectly to differentiate the increased particulate matter caused by cooking, tobacco use or from dust (Bellettiere et al, 2014; Hovell et al, 2011; Klepesis et al 2013). Eventhough these indicators are useful, occasionally the use of $PM_{2.5}$ for continuous monitoring in home can be compromised by environmental factors such as climate and also depends on urban or rural location, whereas for air nicotine measurement, this could be influenced by logistic factors such as storage, transport conditions, shelf life and time needed to set up the monitoring system (Rosen et al, 2015).

However cigarette smoke does also contain many other particles of varying sizes that reflects light making it visible during smoking, whereas chemical gases like CO and benzene released remains invisible, even though these chemicals combine with particles contributing to varying levels and toxic nature of air pollutants. It has been estimated that tobacco smoke contains more than 7000 chemicals including 172 toxic substances, many of them regulated under existing clean air laws and nearly 70 of them cancer causing substances (U.S. Department of Health and Human Services; CDC, 2014). The toxicology of tobacco smoking and details has been previously reported in publications based on the smoking research conducted previously as part of the Merseyside Child health surveys and the Liverpool Womens Hospital study (Kelly et al, 1995; Koshy et al, 2010a, Koshy et al, 2010b; Koshy et al, 2011a, Koshy et al, 2011b; Delpisheh et al, 2006a, Delpisheh et al, 2006b; Delpisheh et al, 2006c; Delpisheh et al, 2006d; Delpisheh et al, 2007, Delpisheh et al, 2008a, Delpisheh et al, 2008b; Delpisheh et al, 2008c, Rizwan et al, 2007; Delpisheh et al, 2009, Koshy et al, 2012a, Koshy et al, 2012b). Many of the air pollutants have toxins similar to the toxins released from tobacco smoking due to tobacco smoke being one of the important sources of air pollution along with toxins released from traffic and industrial sources and due to varying combinations of chemical substances and particles which could lead to similar toxic substances released from different sources. For example, environmental tobacco smoke and diesel exhausts share many of the common chemical components including oxides of nitrogen, carbon dioxide, carbon monoxide, hydrocarbons and aldehydes and similar particulate matter emissions composed of particulate matter less than 2.5 microns (Nelson et al 1998; Cadle et al 1999).

There have been only very few studies or reports which have looked into the equivalence of air pollution in terms of detrimental health effects, when compared to that of smoking. Smoking areas or lounges can lead to extremely high levels of concentrations of toxic air pollutants indoors resulting from different brands of cigarettes containing different types of toxic substances and varying composition of inhalable particles which also depends on the number of cigarettes smoked as well as the method used for smoking tobacco. The average amount of $PM_{2.5}$ mass emitted per cigarette is equivalent to 14 mg making it a dangerous source of air pollution when compared to the average weight of a cigarette (0.9 to 1 gram) (James, 2007). The role of ETS as a major source of particulate matter pollution and increasing indoor PM concentrations up to 10-fold, when compared to the PM_{10} emissions from eco-diesel engine has been reported by Invernizzi et al (2004), which emphasises the need for producing cigarettes with low PM emissions (Nelson PR et al, 1998). However these results from the Ivernizzi study were based on a controlled experiment which was carried out in a private garage in a small mountain town based in northern Italy, well known for low levels of particulate matter air pollution. In this experiment, the results also showed that particulate matter levels from diesel engine exhaust doubled, whereas the particulate matter from environmental tobacco smoke reached levels 15 times more, when measured in the outdoor environment adjacent to the garage. Similar risks of respiratory and cardiovascular diseases from air pollution and environmental tobacco smoke (ETS) have been reported (Pope et al, 2002; Kunzli 2002). Feleszko et al (2014) in their study systematically reviewed the existing evidence of ETS exposure's impact on markers of allergic sensitisation in children, which included fourteen studies on the influence of Environmental Tobacco Smoke (ETS) on total IgE (tIgE) concentration (2603 patients) and specific IgE (sIgE) (9230 patients). In their study, they reported that there was an association between ETS exposure and allergy (t IgE) (increase in concentrations by 27.7%) in early childhood (<7years) [OR=1.20; (95% CI 1.05 to 1.38)] and the increase in risk of allergic sensitisation (sIgE) with younger children suffering more from the immuno-modulating effects of ETS exposure [OR=1.12, (95% CI 1.00-1.25)]. Interestingly, the concurrent presence of air pollution is being utilised as a protective shield by smokers in preventing them from efforts to stop smoking in reducing the health risks associated with tobacco smoke exposure (Nardini et al, 1998).

An unpublished report based on results from analysis of 26 peer reviewed papers and one unpublished dataset from 1999 onwards, being written for the Environment Science and technology journal, examined the chemical make up of indoor dust covering homes to schools across 14 states in United States (Mitro et al, 2016 in press). The results have shown that children who play frequently on floor at home are highly vulnerable to the health problems

associated with indoor household dust contaminated by dangerous and toxic chemicals from a wide variety of indoor household products which used to include phthalates, which have been commonly linked to development problems in babies and hormone disruption affecting children later in life. This is in contrast to the increase in allergies and asthma linked to extremely clean homes resulting from less exposure to various microbes. They have also reported about the need for frequent vacuuming of floors and washing the hands properly after cleaning the floor, each time before taking food and use of wet mops and damp clothes for reducing household dust levels.

Estimation of the doses of tar and nicotine from different cigarettes using a standardised smoking machine test based on the Federal Trade Commission (FTC) showed that dose of tar or fine PM since mid-1990's have been 12 mg per cigarette (National Cancer Institute 2001) and this has been supported by results from multiple data analysis from other studies as well (Djordjevic et al, 2000; Repace, 2007). It is difficult to estimate correct doses of tar or fine particulate matter from active smoking, which in turn depends on the type of cigarette, source of cigarette smoking and individual cigarette smoking patterns (National Cancer Institute 2001). Irrespective of the average daily dose of air pollution resulting from different sources, the daily inhalation rates (m3/day) and concentration of fine PM in the air plays an important role in deciding the severity of adverse health effects, which is similar for the average daily dose of fine PM to the lung from breathing second hand smoke on exposure to tobacco smoke from household members.

Even though, nicotine is the main toxic component present in cigarette smoke, there are other toxins which are harmful and form components of air pollution. For example, carbon monoxide, which is a product released as gas from car exhausts, tar present on road surfaces, acetone present in paint strippers, ammonia in cleaning agents, benzene in petrol fumes, toluene used in industrial solvents, all are toxins common to both tobacco smoke and air pollution. The nicotine released from cigarettes can be present in the atmosphere for some time resulting in indoor air pollution and could get also absorbed through the skin apart from being absorbed into the body after breathing air. It could also get deposited on indoor household items even hours after stopping smoking, which in turn could gradually find its way into the body through breathing or through getting absorbed through skin as mentioned before. This is because nicotine is a sticky molecule, which can remain attached to any material present nearby or to particles in air which could be drifted to other places, influenced by air movement (Beko et al, 2016). However, unfortunately in this study (Beko et al, 2016), the study sample was small and included only men and age was between 35 and 67 years. In order to draw final conclusions,

and for better results there is need for looking at larger study population including different age groups, women and children and also taking into consideration the other toxins present in air, which might get absorbed through skin and also considering the skin type and thickness in different individuals and studying the rate of absorption in less smoky conditions. The clothes exposed to these toxins, if worn can also result in increased levels of these toxins, emphasising the need to take exposure to second hand smoking and resulting indoor air pollution more seriously than previously thought, especially in children as this can affect their brains and causing behavioural problems in later life (Chastang et al, 2015).

Studies have reported that air pollution resulting from tobacco smoking especially from second-hand smoke exposure is caused by a mixture of more than 4000 chemical by-products released through tobacco combustion, out of which nearly 500 of these toxic chemicals are released in gaseous form, which makes it more easily diffusible and causing more health problems from easy inhalation (Repace et al, 2007; Hoffman et al 1998, Ott et al 2003). The concentration of these toxins causing indoor air pollution could also be influenced by the physical volume of the location and a relatively small location could result in excessive accumulation of these toxic substances in a short time leading to extremely high chances of being exposed to these toxins and affecting health, which also depends on the exhaled mid-stream smoke and side stream smoke entering the room. This in turn could also be influenced by the ventilation of the room and number of cigarettes smoked. Smoking tobacco not only results in indoor pollution, it can also contribute to outdoor air pollution, which however depends on the proximity to the source of tobacco smoke and the time spent near to the smoker and direction of wind, which decides the level of exposure (Klepeis et al, 2007). Outdoor air pollution can also result from the process of manufacturing cigaerettes, which releases mainly two chemicals into the air-carbon dioxide and methane. (Novotny T E & Zhao F, 1999).

CHAPTER 3

METHODOLOGY

3.1 Introduction to methodology

This chapter discusses about the different methods used in this study. This includes study design used, study location, reasons for selecting the primary study location and demographic area, study period, sample size, introduction to the two datasets from two different sources, project plan, child health survey data and the air pollution data, the data linkage process, creation of the final dataset, definition of variables used, details of research work involved with issues faced, and air pollution monitoring for air pollutants including the NOx emissions, NO2 concentrations, PM10 emissions and concentrations. The techniques and operational aspects of emissions and concentrations estimation, main outcome measures and exposure variables, optical reading of questionnaires, data analysis along with statistical methods used including the population attributable risk, Venn diagrams, spatial mapping techniques and structural equation modelling, and details of confidentiality followed by ethical considerations are also discussed.

3.2 Study design

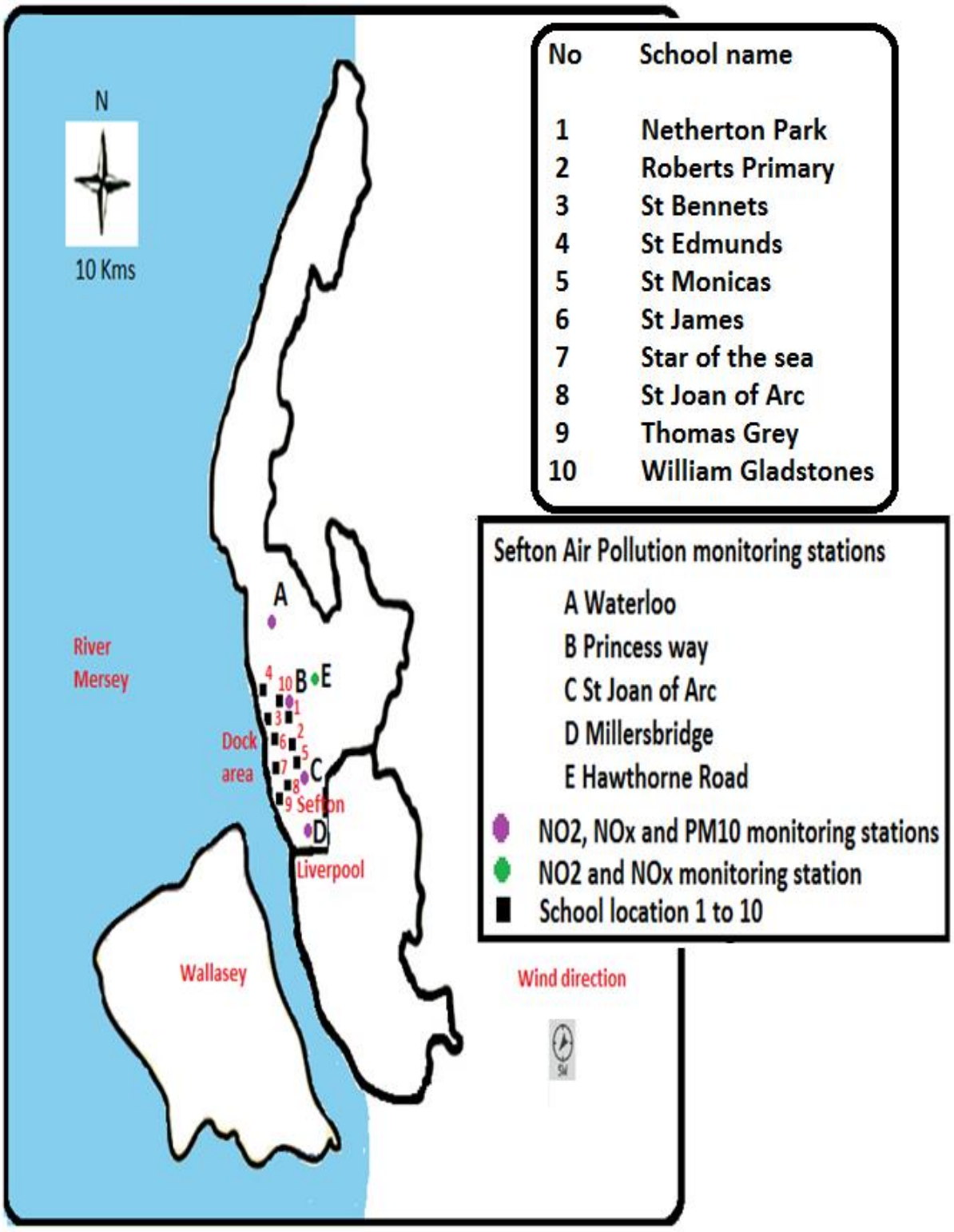
This was a retrospective analysis of a cross-sectional survey data from a dataset formed by combination of two datasets from different sources, but linked using postcode sectors and unique identification numbers. A cross-sectional survey was conducted in 2006 using a standardised respiratory child health survey questionnaire. Data relevant to the air pollution project, for ten schools out of the total 15 schools of the original survey, were selected and extracted. Concurrent air pollutant emission and concentration data for the same year were collected by Sefton Environmental Protection Department.

3.3 Study location

The smoking and air pollution data was available for 10 schools in South Sefton, Liverpool. Data on air pollution was collected within a programme of environmental monitoring and surveillance following standardised procedures and methods and was not based on pre-determined population exposures. In total 9 real time monitors including 5 NOx and 4 PM_{10} monitors were used for estimating emissions of NOx and PM_{10} emissions. The location of the 10 schools and number of air pollution monitors was based on the level of air pollution

and Air Quality Management Areas (AQMA) in Sefton area determined by the Environment protection department, Sefton council (Figure 3.01).

Figure 3.01 Map of location of ten schools in Sefton, Liverpool along with the Sefton NO_2, NOx and PM_{10} emissions and concentrations monitoring stations.



3.4 Reasons for selection of primary study location and suitable demographic area

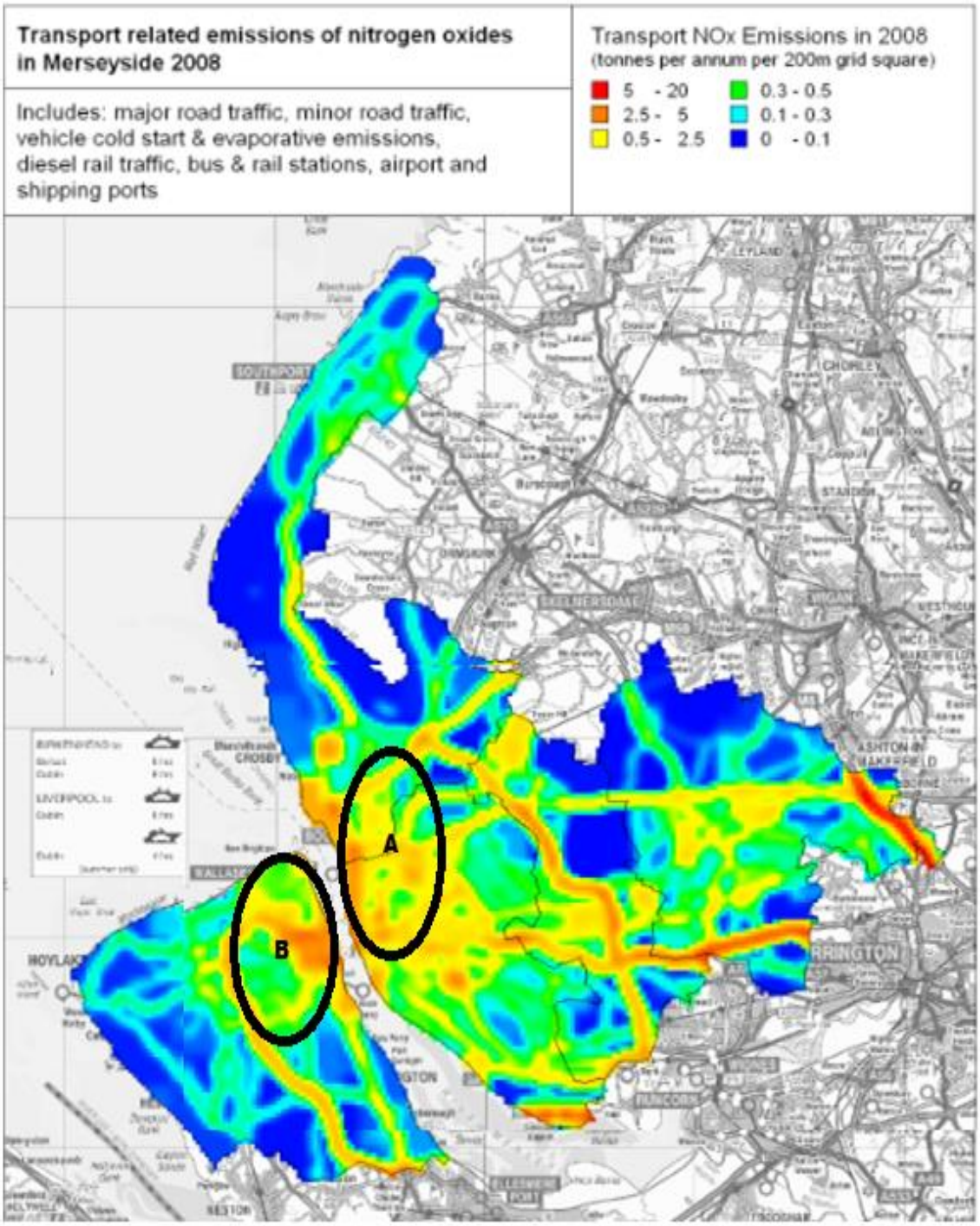
The Sefton area has a high level of dock related activity, and associated industrial traffic flow. Traffic related emissions of oxides of nitrogen for the year 2008 in Merseyside are shown in figure 3.02. Figure 3.03 shows the mild to moderate increase in traffic related emissions of NO_X and Average Annual Daily Traffic (AADT) for the year 2006 for Sefton and Wallasey areas.

The association of asthma with air pollution has been previously reported in the schools in this area. Children in this area had a high and increasing prevalence of doctor diagnosed asthma that continued to rise following reduction in coal dust emissions during the late 1990s, and the explanation for this was unclear (Milligan et al, 1994). Respiratory symptoms were previously mapped spatially in relation to airborne coal dust pollution by Milligan et al (1998) and reported that proximity to a source of dust pollution was associated with increased symptoms of cough as well as breathlessness, school absence due to respiratory symptoms, and doctor-diagnosed asthma. The symptom of excess cough was also related spatially to the average dust deposition levels occurring in the vicinity of the child's home with a dose response association observed. However, respiratory symptoms and doctor diagnosed asthma and its association with air pollutant categories for NO_x and PM_{10} emissions and NO_2 and PM_{10} concentrations havenot been assessed in the above mentioned studies.

Because previous spatial contouring had shown significant residential associations with childhood respiratory health, this raised the question, whether there were associations with other air pollutants than coal dust. The 2006 child health survey showed that there was decreased trend in the prevalence of doctor diagnosed asthma in children (29.8% in 1998 and 19.4% in 2006). If the dynamics of this had stabilised then it was of inherent interest to establish any concurrent associations with available air pollution data.

3.5 Traffic related emissions of oxides of Nitrogen for the year 2008 in Merseyside

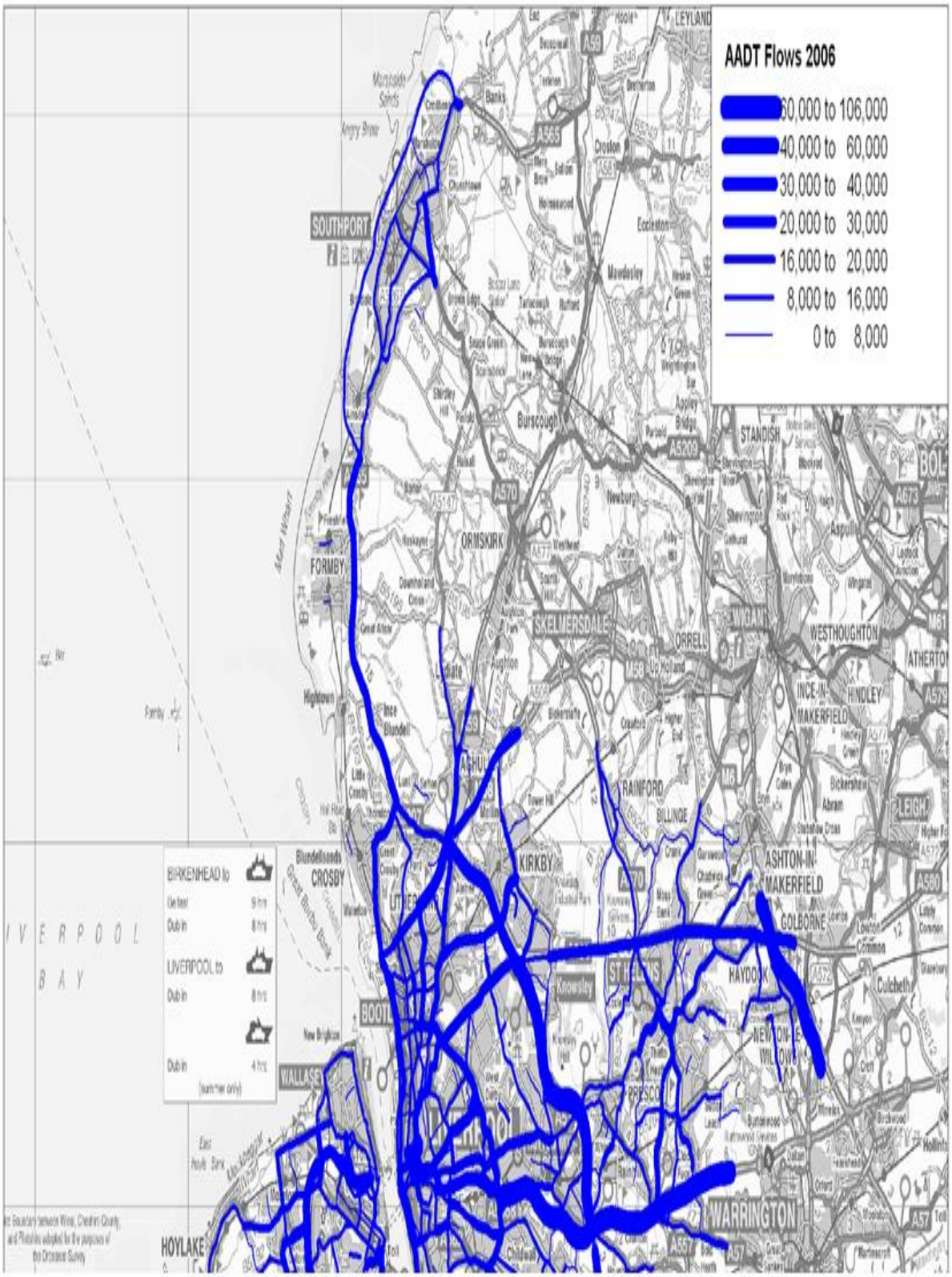
Figure 3.02 Mild to moderate increase in Traffic related emissions of NOx



A 10 Sefton schools **B 5 Wallasey schools**

Source: Sefton Council Environment Protection Department 2008

Figure 3.03 Average Annual Daily Traffic (AADT) for the year 2006 for Sefton and Wallasey areas



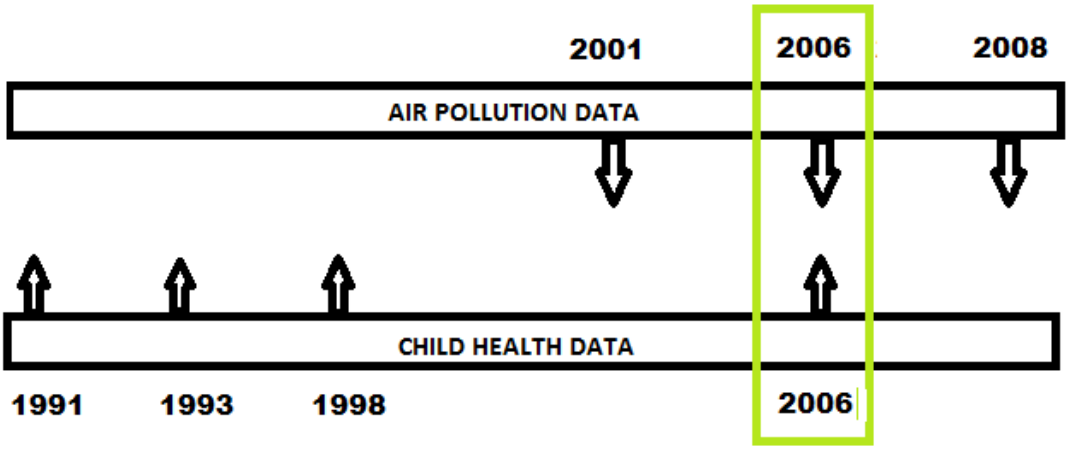
Source: Sefton Council Environment Protection Department 2006

In the 2006 survey, the schools selection was based on location in the original dust-exposed area in Liverpool (10 schools), with an additional 5 schools located upwind from the exposed area and south of the river Mersey (Wallasey). The 5 Wallasey schools originally included in the 1991 and 1993 surveys were located outside the known dispersion areas for the coal dust which had led to the original community concerns about airborne pollution. The Wallasey area is not a major industrial location with less local industrial traffic than the Sefton area and minimal dock related activities. Air pollution data was not available from Wallasey. These locations were predominantly lower socio-economic areas and had comparable Townsend Deprivation Indices based on postcode classification (Kelly et al, 1995; Kelly et al, 1996). The Townsend scores have been replaced by the indices of multiple deprivations which combine a range of indicators such as economic, social and housing issues, into a single deprivation score for each small area in England. Wirral remains the 2nd least deprived authority in the Liverpool City Region and has been ranked as 60th most deprived Local Authority (out of 326 Local Authorities in England) when ranked by average score, whereas Sefton is the only Liverpool City Region authority with a better average score (92) compared to other areas (Wirral Economic Profile, 2014). For the final combined air pollution and cigarette smoking dataset only the 10 schools in South Sefton, Liverpool were included as data on both air pollution and smoking exposure were available.

3.6 Study period

The 2006 survey was carried out from October to December to be consistent with the previous surveys as part of the follow up of the Merseyside Community Child health surveys. This was the only period for which concurrent data was available for health and air pollution exposures, which facilitated the linkage of child health data with the air pollution data for this period for analysis (Figure 3.04). The health data of 792 primary school children from 10 schools from the 2006 Merseyside Community Child health survey and corresponding air pollution data for the same period generated by the Merseyside Atmospheric Emissions Inventory were linked using postcode sectors for analysis in the present study.

Figure 3.04 Timeline of child health surveys and air pollution data concurrence



3.7 Sample size

The sample size in this study was predetermined as this was an opportunistic survey with data selected from the 2006 Child Health Survey, which was linked to the corresponding air pollution data for the same period and same area. Sample size for the 2006 survey was calculated and determined after consultation with statistician and was found to be adequate. The sample size was 792 for 10 schools for the final dataset, after data on five schools were removed from a total of 1074. The air pollution data was provided by the air pollution monitoring team, Sefton council and specific data from this dataset corresponding to the postcode sectors available with the child health data was selected. The final dataset for the study was created by linkage of the selected variables extracted from the Child Health Survey (details provided later in section) with the air pollution data, in the form of emissions for NOx and PM10 and concentrations data for NO2 and PM10 from the Sefton Council for the same period. Selection of data through this method was the best possible solution for facilitating the linkage of the two different related datasets with common postcode sectors. The sample was representative of the original 2006 Child Health Survey as the mean Townsend score scores were comparable and did not vary between the included 10 Liverpool schools (5.25 + 2.26) and the 15 schools included in the original survey (5.26 + 2.23). The selected Child Health Survey data was comparable to the original survey as it was collected during the same period of the year for the same age group of children from schools in the same location and the linked air pollution data corresponded to the same location for the same year.

3.8 Introduction to two datasets and linkage to form final dataset and variables used

3.8.1 Child health survey data

The information and data collected from primary schools in the Sefton area of Merseyside was part of the community child health survey conducted in 2006 during the months from October to December. Selected variables for 10 schools, relevant to the air pollution project were extracted from the 2006 child health survey. Details of this survey have been previously published as part of the community child health surveillance monitoring undertaken in the Merseyside area and co-ordinated by a research team from the Child and Reproductive Health Group based at the Liverpool School of Tropical Medicine, University of Liverpool (Koshy et al, 2012). More details about the study on pregnancy smoking alone and birth and child health outcomes from the child health surveys conducted from 1998 to 2006 are available as full text articles, published in peer reviewed journals over the past few years (Koshy et al, 2010a; Koshy et al, 2010b; Koshy et al, 2011a; Koshy et al, 2011b; Delpisheh et al, 2006b; Delpisheh et al, 2006c; Delpisheh, 2006d; Delpisheh et al, 2007; Delpisheh et al, 2008b; Rizwan et al, 2007; Koshy et al, 2012a; Koshy et al, 2012b; Kelly et al, 1995; Koshy et al, 2013; Reidel et al, 2014). The variables extracted from the 2006 survey to create the final dataset for the air pollution project included child's weight in kilograms, child's height in centimetres, school name, postcode, child age, sex, childhood asthma defined by doctor diagnosed asthma and symptom triad of excess cough, ever wheeze and breathlessness in the last 12 months, bronchitis, croup, allergies, pet allergy, hay fever, food allergy, eczema, household member smoking during pregnancy, maternal smoking during pregnancy, paternal smoking during pregnancy, employment status of mother and father, single parent, birthweight of child in kilograms or pounds, preterm status, breastfeeding status, damp patches on home wall, school attendance, ADHD diagnosed by doctor, and respiratory symptoms of ever wheeze, breathlessness in past twelve months, and excess cough. Questions extracted from the original 13 page parental questionnaire, which have been used in the present study are listed in Appendix B and the variables mentioned above are based on these questions. Some of the variables were recoded and computed to create new variables relevant to the project such as age group, body mass index. The three variables of child body mass index, BMI percentile and z score, child weight for age and height for age z scores were calculated for age and sex using Epi Info Nutrition software version 6 which uses the 2000 NCHS (National Centre for Health Statistics) reference growth data. The NCHS developed growth references based on national

survey data collected in the 1960s, 1970s and 1980s, and these growth charts included anthropometric measurements of weight-for height, weight-for-age, height-for-age, and head circumference-for-age. The development of these charts were based on several National Household Education Surveys (namely NHES II and NHES III). Postcode data was available up to 3 digits representing the post–code sectors. The Body mass index z-scores were used to determine the overweight and obese status of the child using cut-offs of 1.04 and 1.64 standard deviations. More details of obesity and overweight definitions based on these cut offs provided in section 3.14 and in table 3.01. Permission to undertake the study was initially obtained from parents and questionnaires were delivered to schools and distributed to children by class teachers to be taken home for completion by parents/guardians. Completed questionnaires were returned to schools. Informed consent was obtained from the parents to measure the height and weight of the primary school children. The complete details of the questionnaire for the 2006 survey based on ISAAC protocol (International Study of Asthma and Allergy in Childhood) (Asher et al 1995), school revisits for absentees, weight and height measurement and techniques used have been previously described as part of the published papers and PhD thesis (Koshy et al, 2010a; Koshy et al, 2010b; Koshy et al, 2011a; Koshy et al, 2011b; Koshy et al, 2012a; Koshy et al, 2012b; Koshy et al, 2013). The information document and consent form for the 2006 survey has been attached in text in Appendix C. Children mostly returned questionnaires during the following two week period. All the questionnaires were cross checked for any different formats of data entered (for example age entered in years or months; birthweight entered in pounds or kilograms or grams) and corrected accordingly for completeness. At the time of enrolment all these children were aged 5 to 11 years. All data were coded and entered into the SPSS version 20.

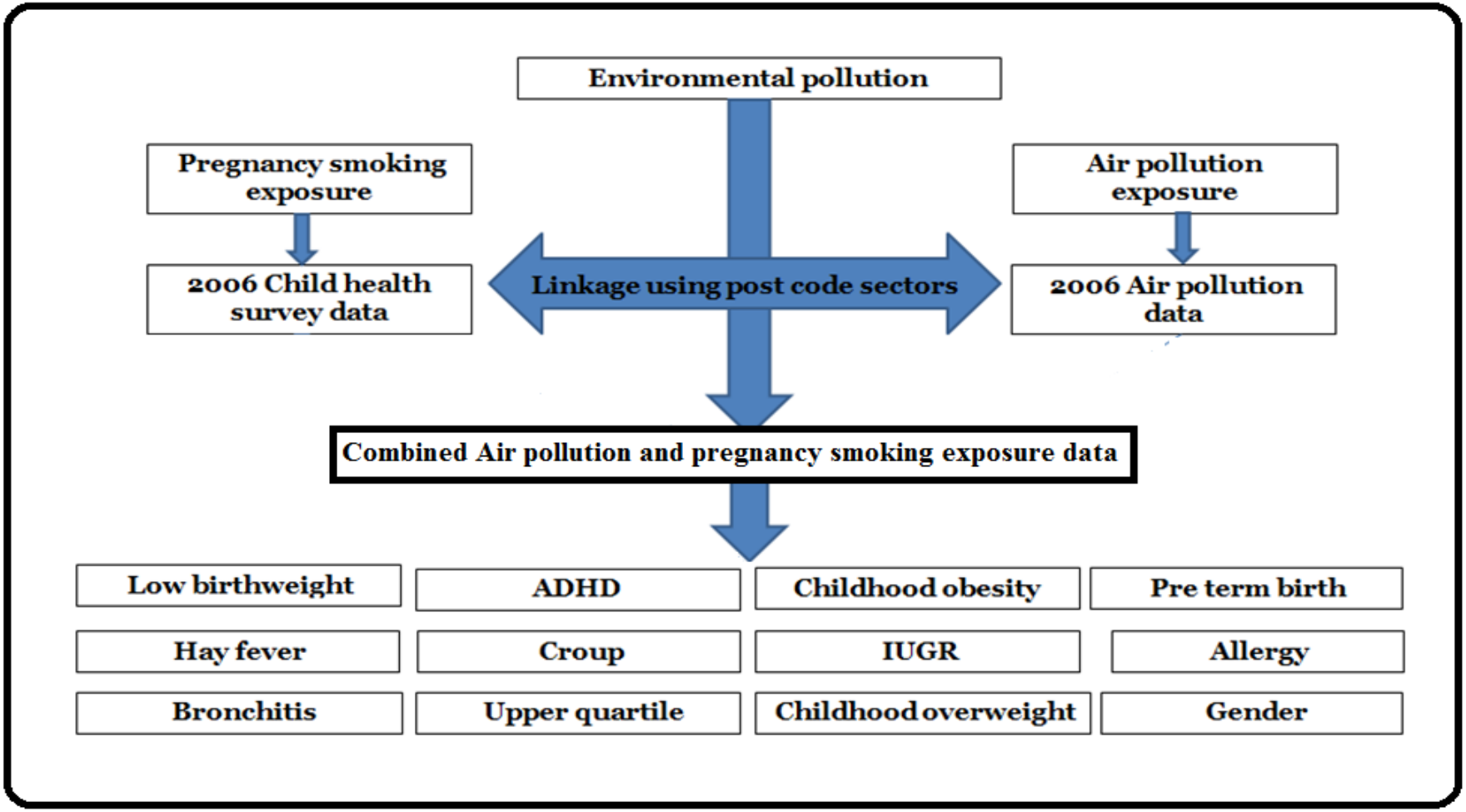
3.8.2 Air pollution data

Air pollutants data was provided by the Merseyside Atmospheric Emissions Inventory located in Sefton Council. Collection of this data was co-ordinated by Gary Mahoney, Principal Environmental Protection Officer for the Environmental Protection Department. Data on the total and average emissions of NOx and PM_{10} in tonnes per annum, and average concentrations of NO_2 and PM_{10} (particles with aerodynamic diameter <10μm (μg/m3), for the year 2006 were obtained. These were made available as excel files. Technical details are outlined below in sections 3.9, 3.10 and 3.11.

3.8.3 Linkage of child health survey data and air pollution data

The community child health data were linked to the emissions data for NOx and PM_{10}, and concentrations data for NO_2 and PM_{10} using 3 digit postcodes sectors, and unique identification numbers. The postcode data was provided by the Sefton Council along with the air pollution data. The three digit postcode sectors were used because with the details of health data available for each 6 digit postcode was limited for matching with the postcodes for the emissions data. Data based on three digit postcode sectors facilitated better matching of child health and air pollution data and better coverage of air quality management areas. Unique identification numbers are the numbers which were allocated to same postcode sectors corresponding to each dataset and this was used for linking the two datasets. Data sets were transferred to SPSS. NOx and PM_{10} data were available as total and average emissions measured in tonnes per annum and NO_2 and PM_{10} concentrations data were available in milligram per metre^3. Missing values were coded by 9 or 999. The map of the location of the ten primary schools included in the present study along with monitoring stations marked separately has been included in section 3.3 (Figure 3.01). Appendices D, E F and G show the spatial distribution of NO_X and PM_{10} emissions and NO_2 and PM_{10} concentrations for the Sefton postcode sectors for 2006. Appendix H shows the Sefton postcode sectors and emissions and concentrations values for 2006. The project plan including data linkage and analysis of relevant birth and child health outcomes in relation to air pollution and smoking and child health data using postcode sectors is outlined and summarised in the figure 3.05 below.

Figure 3.05 Project plan with linkage of air pollution data and smoking and child health data using postcode sectors



3.8.4 Final dataset

All the selected variables from the 2006 child health survey along with the recoded variables and air pollution indicator variables along with pregnancy smoking exposure categories and birth and child health outcomes were included in the final dataset. In addition to the variables included from the 2006 child health survey and from the air pollution data, the final combined dataset included some recoded new variables like categories for combined NOx-PM_{10} emissions, combined NO_2-PM_{10} concentrations, combined MSDP and NOx emissions, combined MSDP and PM_{10} emissions, combined MSDP and NOx-PM_{10} emissions, combined MSDP and NO_2 concentration, combined MSDP and PM_{10} concentration, combined MSDP and NO_2-PM_{10} concentration, combined PSDP and NOx emissions, combined PSDP and PM_{10} emissions, combined PSDP and NOx-PM_{10} emissions, combined PSDP and NO_2 concentration, combined PSDP and PM_{10} concentration, combined PSDP and NO_2-PM_{10} concentration, combined HSDP and NOx emissions, combined HSDP and PM_{10} emissions combined HSDP and NOx-PM_{10} emissions, combined HSDP and NO_2 concentration, combined HSDP and PM_{10} concentration, combined HSDP and NO_2-PM_{10} concentration. Birth and child health outcomes were also assessed in relation to air pollution categories combined with dose related smoking exposure categories for both maternal and paternal smoking categories. These categories were created for the purpose of assessing the birth and child health outcomes in relation to combined exposures of air pollution and pregnancy smoking.

The definitions of related outcome variables are shown in the table 3.01

Table 3.01 Definition of variables and related categories

Variable	Definitions
Doctor diagnosed asthma	Child diagnosed by a doctor as having asthma or bronchial asthma
Ever wheeze	Child ever had wheezing
Breathlessness	Child ever been either unexpectedly breathlessness at rest or more breathless than expected in the past 12 months

Excess cough	Child ever seemed to cough more than other children
C+W+B symptom triad	Symptom combination of excess cough, ever wheezing and breathlessness in last 12 months (cough refers to at least 12 months)
Well controlled Asthma	According to the parent, child's asthma is well controlled
Croup	Child ever been diagnosed by doctor as having croup
Allergy	Child having allergy
Hay fever	Child having hay fever
Bronchitis	Child ever been diagnosed by doctor as having bronchitis
School absenteeism	Child absent from school due to respiratory complaints for one or more than one day in the previous year
Hospital admission	Child ever admitted to hospital due to respiratory complaints for one or more than one day
Obesity	Child with BMI Z score $\geq$1.64 SD based on UK 1990 reference standard
Overweight	Child with BMI Z score $\geq$1.04 SD based on UK 1990 reference standard
Maternal smoking during pregnancy (MSDP)	Mother who smoked one or more cigarettes at any time during pregnancy
NMSDP	Mother who never smoked during pregnancy
Heavy maternal smokers	Mothers who smoked more than 10 cigarettes at any time during pregnancy period
Light maternal smokers	Mothers who smoked less than or equal to 10 cigarettes at any time during pregnancy period
Paternal smoking during pregnancy (PSDP)	Fathers who smoked at any time during his partner's or wife's pregnancy period

NPSDP	Father who never smoked at any time during his partner's or wife's pregnancy period
Heavy paternal smokers	Father who smoked more than 10 cigarettes at any time during his partner's or wife's pregnancy period
Light paternal smokers	Fathers who smoked less than or equal to 10 cigarettes at any time during pregnancy period
Household smoking (HSDP)	Presence of household member who smoked at any time during the pregnancy period
NHSDP	Household member who never smoked at any time during the pregnancy period
Fetal sex ratio	Ratio of male to female babies
Low birthweight	Birthweight < 2500 grams
High birthweight	Birthweight >4000 grams
Emissions	Amount of pollutant matter released into the atmosphere from a specific pollutant source and within a specific time interval measured in tonnes per annum
Concentrations	Amount of pollutant matter in atmosphere per volume unit representing the value of air quality measured in microgram/m3
High NOx emissions	Total NOx emissions more than 10 tonnes per annum
Low NOx emissions	Total NO_X emissions less than or equal to 10 tonnes per annum
High PM_{10} emissions	Total PM_{10} emissions more than 5 tonnes per annum
Low PM_{10} emissions category	Total PM_{10} emissions less than or equal to 5 tonnes per annum
High NO_2 concentrations	Average NO_2 concentrations more than 17.01 microgram per m^3
Low NOx concentrations	Average NO_2 concentrations less than or equal to 17.01 microgram per m^3

High PM_{10} concentrations	Average PM_{10} concentrations more than 13.91 microgram per m^3
Low PM_{10} concentrations	Average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
High NOx-PM_{10} emissions	Total NO_X emissions more than 10 tonnes per annum and total PM_{10} emissions more than 5 tonnes per annum
Low NOx-PM_{10} emissions	Total NOx emissions less than or equal to 10 tonnes per annum and total PM_{10} emissions less than or equal to 5 tonnes per annum
High NO2-PM_{10} concentrations	Average NO_2 concentrations more than 17.01 microgram per m3 and average PM_{10} concentrations more than 13.91 microgram per m^3
Low NOx-PM_{10} concentrations	Average NO_2 concentrations less than or equal to 17.01 microgram per m3 and average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
MSDP + high NOx emissions	Maternal smoking during pregnancy combined with total NOx emissions more than 10 tonnes per annum
NMSDP + low NOx emissions	Mothers not smoking during pregnancy combined with total NOx emissions less than or equal to 10 tonnes per annum
PSDP + high NOx emissions	Paternal smoking during pregnancy combined with total NOx emissions more than 10 tonnes per annum
NPSDP + low NOx emissions	Fathers not smoking during pregnancy combined with total NOx emissions less than or equal to 10 tonnes per annum
HSDP + high NOx emissions	Household smoking during pregnancy combined with total NOx emissions more than 10 tonnes per annum
NHSDP + low NOx emissions	Household members not smoking during pregnancy combined with total NOx emissions less than or equal to 10 tonnes per annum
MSDP + high PM_{10} emissions	Maternal smoking during pregnancy combined with total PM_{10} emissions more than 5 tonnes per annum

NMSDP + low PM_{10} emissions	Mothers not smoking during pregnancy combined with total PM_{10} emissions less than or equal to 5 tonnes per annum
PSDP + high PM_{10} emissions	Paternal smoking during pregnancy combined with total PM_{10} emissions more than 5 tonnes per annum
NPSDP + low PM_{10} emissions	Fathers not smoking during pregnancy period combined with total PM_{10} emissions less than or equal to 5 tonnes per annum
HSDP + high PM_{10} emissions	Household members smoking during pregnancy combined with total PM_{10} emissions more than 5 tonnes per annum
NHSDP + low PM_{10} emissions	Household members not smoking during pregnancy period combined with total PM_{10} emissions less than or equal to 5 tonnes per annum
MSDP + high NO_2 concentrations	Maternal smoking during pregnancy combined with average NO_2 concentrations more than 17.01 microgram per m^3
NMSDP + low NO_2 concentrations	Mothers who did not smoke during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3
PSDP + high NO_2 concentrations	Paternal smoking during pregnancy combined with average NO_2 concentrations more than 17.01 microgram per m^3
NPSDP + low NO_2 concentrations	Fathers who did not smoke during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3
HSDP + high NO_2 concentrations	Household member smoking during pregnancy combined with average NO_2 concentrations more than 17.01 microgram per m^3
NHSDP + low NO_2 concentrations	Household members who did not smoke during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3

MSDP + high PM_{10} concentrations	Maternal smoking during pregnancy combined with average PM_{10} concentrations more than 13.91 microgram per m3
NMSDP + low PM_{10} concentrations	Mothers who did not smoke during pregnancy combined with average PM_{10} concentrations less than or equal to 13.91 microgram per m3
PSDP + high PM_{10} concentrations	Paternal smoking during pregnancy combined with average PM_{10} concentrations more than 13.91 microgram per m^3
NPSDP + low PM_{10} concentrations	Fathers who did not smoke during pregnancy combined with average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
HSDP +high PM_{10} concentrations	Household member smoking during pregnancy combined with average PM_{10} concentrations more than 13.91 microgram per m^3
NHSDP + low PM_{10} concentrations	Household members who did not smoke during pregnancy combined with average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
MSDP + high NOx-PM_{10} emissions	Mother who smoked one or more cigarettes at any time during pregnancy combined with total NOx emissions more than 10 tonnes per annum and total PM_{10} emissions more than 5 tonnes per annum
NMSDP + low NOx-PM_{10} emissions	Mother who never smoked during pregnancy combined with total NOx emissions less than or equal to 10 tonnes per annum and total PM_{10} emissions less than or equal to 5 tonnes per annum
MSDP + high NO_2-PM_{10} concentrations	Mothers who smoked one or more cigarettes at any time during pregnancy combined with average NO_2 concentrations more than 17.01 microgram per m^3 and average PM_{10} concentrations more than 13.91 microgram per m_3
NMSDP +low NOx-PM_{10} concentrations	Mothers who never smoked during pregnancy combined with

	average NO_2 concentrations less than or equal to 17.01 microgram per m^3 and average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
PSDP + high NOx-PM_{10} emissions	Fathers who smoked at any time during his partner's or wife's pregnancy period combined with total NOx emissions more than 10 tonnes per annum and total PM_{10} emissions more than 5 tonnes per annum
NPSDP + low NOx-PM_{10} emissions	Fathers who never smoked at any time during his partner's or wife's pregnancy period combined with total NOx emissions less than or equal to 10 tonnes per annum and total PM_{10} emissions less than or equal to 5 tonnes per annum
PSDP + high NO_2-PM_{10} concentrations	Fathers who smoked at any time during his partner's or wife's pregnancy period combined with average NO_2 concentrations more than 17.01 microgram per m^3 and average PM_{10} concentrations more than 13.91 microgram per m^3
NPSDP +low NOx-PM_{10} concentrations category	Fathers who never smoked at any time during his partner's or wife's pregnancy period combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3 and average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
HSDP + high NOx-PM_{10} emissions category	Household members who smoked at any time during the pregnancy period combined with total NOx emissions more than 10 tonnes per annum and total PM_{10} emissions more than 5 tonnes per annum
NHSDP + low NOx-PM_{10} emissions category	Household members who never smoked at any time during the pregnancy period combined with total NOx emissions less than or equal to 10 tonnes per annum and total PM_{10} emissions less than or equal to 5 tonnes per annum
HSDP + high NO_2-PM_{10} concentrations category	Household members who smoked at any time during the pregnancy period combined with average NO_2 concentrations more than 17.01 microgram per m3 and

	average PM_{10} concentrations more than 13.91 microgram per m3
NHSDP + low NOx-PM_{10} concentrations category	Household members who never smoked at any time during the pregnancy period combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3 and average PM_{10} concentrations less than or equal to 13.91 microgram per m3
Heavy maternal smoking + high NOx emissions	Mothers who smoked more than 10 cigarettes at any time during pregnancy period combined with total NOx emissions more than 10 tonnes per annum
Maternal none smokers + low NOx emissions	Mothers who never smoked at any time during combined with total NOx emissions less than or equal to 10 tonnes per annum
Heavy maternal smoking + high PM_{10} emissions	Mothers who smoked more than 10 cigarettes at any time during pregnancy period combined with total PM_{10} emissions more than 5 tonnes per annum
Maternal none smokers + low PM_{10} emissions	Mothers who never smoked at any time during combined with total PM_{10} emissions less than or equal to 5 tonnes per annum
Heavy maternal smoking + high NO_2 concentrations	Mothers who smoked more than 10 cigarettes at any time during pregnancy period combined with average NO_2 concentrations more than 17.01 microgram per m3
Maternal none smokers + low NO_2 concentrations	Mothers who never smoked at any time during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m3
Heavy maternal smoking + high PM_{10} concentrations	Mothers who smoked more than 10 cigarettes at any time during pregnancy period combined with average PM_{10} concentrations more than 13.91 microgram per m3
Maternal none smokers + low PM_{10} concentrations	Mothers who never smoked at any time during pregnancy combined with average PM_{10} concentrations less than or equal to 13.91 microgram per m3
Heavy maternal smoking + high NOx-PM_{10} emissions	Mothers who smoked more than 10 cigarettes at any time during pregnancy period combined with total NOx

	emissions more than 10 tonnes per annum and total PM_{10} emissions more than 5 tonnes per annum
Maternal none smokers + low NOx-PM_{10} emissions	Mothers who never smoked at any time during pregnancy combined with total NOx emissions less than or equal to 10 tonnes per annum and total PM_{10} emissions less than or equal to 5 tonnes per annum
Heavy maternal smoking + high NO_2-PM_{10} concentrations	Mothers who smoked more than 10 cigarettes at any time during pregnancy period combined with average NO_2 concentrations more than 17.01 microgram per m3 and average PM_{10} concentrations more than 13.91 microgram per m3
Maternal none smokers + low NO_2-PM_{10} concentrations	Mothers who never smoked at any time during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m3 and average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
Heavy paternal smoking + high NOx emissions	Fathers who smoked more than 10 cigarettes at any time during pregnancy period combined with total NOx emissions more than 10 tonnes per annum
Paternal none smokers + low NOx emissions	Fathers who never smoked at any time during combined with total NOx emissions less than or equal to 10 tonnes per annum
Heavy paternal smoking + high PM_{10} emissions	Fathers who smoked more than 10 cigarettes at any time during pregnancy period combined with total PM_{10} emissions more than 5 tonnes per annum
Paternal none smokers + low PM10 emissions	Fathers who never smoked at any time during combined with total PM_{10} emissions less than or equal to 5 tonnes per annum
Heavy paternal smoking + high NO_2 concentrations	Fathers who smoked more than 10 cigarettes at any time during pregnancy period combined with average NO_2 concentrations more than 17.01 microgram per m^3

Paternal none smokers + low NO_2 concentrations	Fathers who never smoked at any time during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3
Heavy paternal smoking + high PM_{10} concentrations	Fathers who smoked more than 10 cigarettes at any time during pregnancy period combined with average PM_{10} concentrations more than 13.91 microgram per m^3
Paternal none smokers + low PM_{10} concentrations	Fathers who never smoked at any time during pregnancy combined with average PM_{10} concentrations less than or equal to 13.91 microgram per m^3
Heavy paternal smoking + high NOx-PM_{10} emissions category	Fathers who smoked more than 10 cigarettes at any time during pregnancy period combined with total NOx emissions more than 10 tonnes per annum and total PM_{10} emissions more than 5 tonnes per annum
Paternal none smokers + low NOx-PM_{10} emissions category	Fathers who never smoked at any time during pregnancy combined with total NOx emissions less than or equal to 10 tonnes per annum and total PM_{10} emissions less than or equal to 5 tonnes per annum
Heavy paternal smoking + high NO_2-PM_{10} concentrations category	Fathers who smoked more than 10 cigarettes at any time during pregnancy period combined with average NO_2 concentrations more than 17.01 microgram per m^3 and average PM_{10} concentrations more than 13.91 microgram per m^3
Paternal none smokers + low NO_2-PM_{10} concentrations category	Fathers who never smoked at any time during pregnancy combined with average NO_2 concentrations less than or equal to 17.01 microgram per m^3 and average PM_{10} concentrations less than or equal to 13.91 microgram per m^3

3.9 Research work involved in this study and issues faced

Research work included initially planning for this project and deciding on the datasets and variables to be used and finally getting the two datasets ready for linking with each other using the postcode sectors and unique identification numbers to create the combined final dataset to facilitate the analysis based on the study specific objectives to look at the birth and child health outcomes in relation to air pollution and combined air pollution and pregnancy smoking exposure.

But for this, initially regular meetings were carried out with the Sefton Council to discuss the requirements for the air pollution study and the relevant data needed specific to the period of the child health survey carried out in 2006. These meetings were carried out in the Environmental Protection Department, Sefton Council, Bootle and also with the Child and Reproductive Health Group, based in Liverpool School of Tropical Medicine. Since the air pollution data needed for this study had to be specifically selected to be linked to the child health data for the study period of the 2006 child health survey, data was obtained after few months of discussions and after approval from the Sefton Council. Special sessions and meetings were arranged to understand more about the working system of the Merseyside Atmospheric emissions inventory, the types of air pollutants estimated, the methods of collection of the emissions data for NOx and PM_{10} and concentrations data for NO_2 and PM_{10}, instruments used for estimation of these air pollutants. Regular visits to meet the Air Quality monitoring team, Sefton Council and also the Air Quality Management areas, were carried out in order to learn more about the air pollution monitoring system and also to collect more information about the real time monitors measuring emissions for NOx and PM_{10} and to assess the working of different instruments used for real time measurement and estimation of data. Air quality monitoring station and related equipment used in measurement of oxides of Nitrogen and PM_{10} emissions are illustrated in figure below showing the operating and monitoring system and accessories used in day to day working of the air pollution monitoring system (Figure 3.06).

Real time measurements of emissions were monitored from Sefton council and any system failure was rectified once it was detected. Dock areas near to air quality management areas were also visited to get an idea of the sources of air pollution including areas with high density traffic including heavy duty trucks.

Figure 3.06 Visit to the Air Quality Monitoring station

There was confusion whether to use emissions data or concentrations data or both indicators for the oxides of nitrogen and PM_{10} for the study. But, later it was decided to use both the emissions and concentrations data because it was interesting to note that these air pollution indicators, when defined in epidemiological terms, were relevant, with emissions measured in tonnes per annum, and concentrations measured in micrograms per m3, represented measurements of air pollutant indicators equivalent to the period and point prevalence respectively. The postcodes sectors for which these data were available were

examined and final data on air pollution were collected in excel format and different possible methods to be explored for the analysis of birth and child health outcomes in relation to air pollution data and pregnancy smoking data were also discussed.

Postcode sectors were used in this study as it was difficult to link the air pollution data and the child health data at the postcode level, mainly because data matching was not possible due to variability in postcodes between the two different datasets, whereas it was possible to match the data using postcode sectors, due to uniform distribution between the two datasets. For this purpose, a new variable on postcode sectors was created from the available postcodes, along with a corresponding unique identification number attached to each postcode sector, which was used to link the two datasets to form the final dataset. The cut-offs to be used for the high and low categories for NOx emissions, NO_2 concentrations, PM_{10} emissions and PM_{10} concentrations were also finalised after discussion with the Air Quality monitoring team, Environmental Protection Department, Sefton Council, which was also based on the spatial maps produced and used by them for categorising high and low emission and concentration categories for oxides of Nitrogen and PM_{10}, for their Air Quality project. Emissions data for NOx and PM_{10} for 2008 was also collected from Sefton Council to assess the trends in prevalence of these air pollutants. Corresponding concentration data for NO_2 and PM_{10} were not available for 2008. Project and subject specific training included attending online training sessions to familiarise with the Venn diagram software and to learn more about creating proportional Venn diagrams for health outcomes in relation to exposure data. Regular meetings were also carried out with staff members specialised in use of in ArcGIS software from the Liverpool School of Tropical Medicine for guidance and for getting trained in creating spatial maps based on health data and postcode sectors. Training in use of ArcGIS software was also provided by the team from the Computing Services Department, University of Liverpool. Structural Equation Modelling technique using SPSS Amos software was also tried after meetings with my second supervisor, specialised in Statistics and well trained in SEM techniques. Training on use of SPSS and logistic regression techniques and epidemiological methods was provided during the early stage of my study as part of the statistical workshops conducted by the Department of Biostatistics, University of Liverpool. Getting used to the Spatial mapping software Arc GIS, Venn diagrams software and SEM using SPSS Amos, took considerable amount of time for producing the results based on the air pollution and pregnancy smoking exposure categories and diagrammatic representation and spatial mapping of the child health outcomes in relation to these combined exposures. The issue related to creating spatial

mapping was that spatial map software did not allow using different combined air pollution-pregnancy smoking outcome category for the same postcode sector. So the mode of each combined exposure outcome category was used for each postcode sector as part of producing spatial maps after further discussion with the team.

3.10 Air pollution monitoring

The final data on NOx emissions, NO_2 concentrations, PM_{10} emissions and PM_{10} concentrations were provided by the Air Quality monitoring team, Environmental Protection Department, Sefton Council, who were the team responsible for measurement and calculation of these air pollutants following standardized techniques. Details on air pollution measurement are included in the next few sections. The Air Quality monitoring team was responsible for measurement and calculation of the NOx and PM_{10} emissions measured in tonnes per annum and NO_2 and PM_{10} concentrations measured in mg/m3 and provided the final air pollution data needed for linking with the Sefton Child health data. The NO_2 concentrations were monitored using the NO2 passive diffusion tubes not requiring any power supply and were estimated using colorimetric methods and dispersion models. The PM_{10} concentrations were monitored using the Dust scan sticky pad directional dust gauges by a passive method not requiring any power supply. The NOx and PM_{10} emissions were obtained from the Merseyside Atmospheric Emissions Inventory and were estimated using the emission factor and activity data related to source of air pollution for NOx emissions and PM_{10} emissions respectively. More details on the Merseyside Atmospheric Emissions Inventory along with the details of methods and operational aspects used for measurement of air pollutants are summarised in the next few sections.

In Merseyside, the Sefton Council currently has air pollution monitoring equipment in use which includes two stations of the same standard used by stations in the Government's air quality monitoring network (Automatic Urban Network or AUN). The different stations measure real time levels of: carbon monoxide, sulphur dioxide, oxides of nitrogen, particles, ozone, benzene, xylene and toluene levels. NOx and PM_{10} emissions and NO_2 and PM_{10} concentrations were available for 2006. A network of diffusion tubes were used to measure monthly average oxides of nitrogen concentration levels. There were three diffusion tube monitoring programs: including four tubes which formed part of the national nitrogen dioxide survey- in-house monitoring program with twenty seven tubes in Sefton and twenty four tubes

in the Community Air-watch Programme, in which members of the community have diffusion tubes at their properties. Sefton Council's monitoring network was operated and run by officers trained in all aspects of air quality monitoring, including routine site maintenance, calibration of analyzers and data ratification. Horiba 360 and 370 series analyzers were used for gaseous pollutants and Tapered Element Oscillating Microbalance (TEOM) and Beta Attenuation Monitor (BAM) analyzers were also used for particulates. Sefton Council maintains a rigorous Quality Assurance (QA) and Quality Control (QC) program which incorporates the daily screening of the monitoring and calibration process by visual examination in order to ascertain if any immediate action is necessary. They also conduct fortnightly site visits to carry out routine maintenance and calibration checks. Equipment maintenance checks are supported by six monthly servicing carried out by trained service engineers. Apart from that six monthly QA/QC audits are carried out by an external UKAS (United Kingdom Accreditation Service) accredited field auditor, who assesses the standards against internationally agreed standards and reviews data validation and verification and ratification of all datasets.

3.10.1 NOx Emissions and NO_2 concentrations estimation technique

The passive diffusion tubes were used as chemical monitors which in turn monitored the oxides of nitrogen and provided monthly average data. NOx concentrations were measured using nitrogen dioxide diffusion tubes (Figure 3.07), which is a very cheap and widely used simple device which does not require any power supply. These tubes are acrylic tubes (7cm x 1cm) with stainless steel mesh discs impregnated with triethanlomaine (TEA). Nitrogen dioxide is absorbed as nitrite, which reacts with sulphanilamide and N-1 naphthyl ethylenediamine dihydrochloride (NEDA) to form a pink coloured dye and was determined colorimetrically. The nitrogen dioxide concentration was averaged over the sampling period. Diffusion tubes exhibit under or over estimation compared with the reference chemiluminescence method (the real time monitors), which is rectified by applying a bias adjustment factor to all diffusion tube results to obtain bias corrected annual means that could be compared with the air quality standards (DEFRA, 2008).

Figure 3.07 Nitrogen dioxide diffusion Tube

3.10.2 PM_{10} emissions and concentrations estimation technique

DustScan Ltd., "sticky pad" directional dust gauges, were used as part of air quality monitoring survey for estimating particulate matter (Datson & Birch, 2007). It was developed as a novel method for directional dust monitoring in the 1980's and was used as a special adhesive dust collection system known as a "sticky pad", mounted on a replaceable cylindrical

monitoring head and this being a passive phenomenon never recquired any power supply (Figure 3.08)

Figure 3.08 Dust Scan Directional Gauge for PM_{10} monitoring

Source – DustScan Limited.

The monitoring head was attached to a post or fence and an alignment peg was used to set the gauge to magnetic north when first installed. This ensured repeatable dust sampling each time the monitoring heads were changed by monitoring and measuring the dust in that specific direction for a certain specified period. Once the protective layer was removed, the dust monitoring head samples were checked continuously every day whilst exposed, with the sampling period being typically one week, to avoid overloading. The monitoring head containing the sticky pad collects dust coming from 360^0 around the gauge, which could be

split into 15^0 sectors to ascertain the precise direction the dust originated from. At the end of the sampling period, the head was removed and placed in a protective carrying flask and a replacement head fitted to commence the next sampling period. Exposed sampling heads were send to the Dust Scan offices by courier in transport boxes for computer analysis, which was then sealed with a transparent film once it was received and then was scanned on a flatbed scanner linked to a PC. The air borne dust on the adhesive surface was then measured twice and quantified using software developed by DustScan. A rigorous quality assurance and quality control programme which involved daily screening, by visual examination, of all monitoring and calibration data was used with 6 monthly QA/QC audits carried out by an external UKAS accredited field auditor, which was followed by data validation and verification of all datasets.

3.11 Operational aspects of emissions and concentrations estimation

The emissions estimation methodologies used by the Merseyside Atmospheric emissions inventory (MAEI) (2006) were based principally on emission factors and activity data estimated or measured in the base year. Emission inventories are estimates of the amount and the type of pollutants that are emitted to the air each year from all sources including transportation, domestic dwellings and industrial and commercial premises. The MAEI is a database of geographically referenced datasets of emission sources within the Merseyside region, holding estimates of the amount and type of pollutants emitted to the air and provided a structured framework within which emissions information was stored and analysed, allowing comparisons between different emission source types, across the Merseyside local authorities and with national statistics. Emission factors used in preparing the MAEI (2006) were predominantly derived from the National Atmospheric Emissions Inventory (NAEI) online data warehouse and from the design manual for roads and bridges used by the Air Quality team, Sefton Council. The emissions factors were useful for detailed examination of the nature and size of emission sources within Merseyside. This was used to identify areas and sources of high pollution, as it was based on spatial maps and were helpful for informing decisions and investigations related to local and regional air quality issues. An emissions inventory can be used as a key input to dispersion modelling. Dispersion models were used for estimating the ambient air pollution concentration after dispersion from the emissions sources and even were useful in situations where there were different sources of air pollution (CERC Air Quality Modelling for Sefton Final Report, 2010). Concentration data was also produced using the Airviro dispersion model using emissions data from the Merseyside Atmospheric Emissions

Inventory and meteorological data from the Crosby meteorological Station. Airviro is a web based system for Air Quality Management and this uses an integrated system for time series data handling, emission inventories and dispersion modelling (Airviro system 2014, available at http://www.smhi.se/airviro).

Emissions database software was used to store manipulate and assess emissions data. Emissions factors which are representative values were used to relate the quantity of air pollutants released to the atmosphere with any activity associated with the release of that pollutant. It has been used as aspecific tool for developing local and national emissions inventories and for formulating decisions related to air quality management and emissions control strategies, which was then applied to activity data relating to a source in order to estimate emissions rate as follows:

Activity rate x Emission factor = Emission rate

The nitrogen oxides, (NO_X) and particulate matter, (PM_{10}) emissions were reported in tonnes per annum. The grid size used in the analysis was 200 metres and therefore the emissions values were measured as tonnes per 200 metre grid square and the use of grid size helps to give an idea of the composition of the atmospheric pollution based on the source in that area along with the transport and deposition of these pollutants over a certain period of time as decided by the air quality control team.

The NO_2 concentration spatial maps were created by modelling NOx figures from the Merseyside Atmospheric Emissions Inventory (MAEI) for Sefton. The NOx background figures for Sefton were obtained from the Department for Environment, Food and Rural affairs (DEFRA), one of the government bodies responsible for the protection of the environment in the United Kingdom of Great Britain and Northern Ireland and the NO_2 concentrations were calculated using DEFRA's NO_2 converter toolkit based on catalyst filled cartridges. The NO_2 concentrations for each 100m grid square in Sefton was then allocated into the appropriate postcode sector using MapInfo, a desktop based geographic information system (GIS) software followed by averaging the 100 m grid squares within each postcode sector enabling the creation of thematic maps. For PM_{10} maps, modelling of PM_{10} exhaust, brake and tyre wear figures from the MAEI for Sefton, was carried out to get the PM_{10} concentrations. The PM_{10} background figures from DEFRA were then obtained for Sefton and the total PM_{10}

concentration was derived from these figures by the Sefton Air Quality monitoring team. The PM_{10} concentrations for each 100m grid square in Sefton was then allocated into the appropriate postcode sector using MapInfo followed by averaging the 100m grid squares within each postcode sector to create thematic maps.

Emissions output values for NOx and PM_{10} were linked to health data sets using unique identification numbers, which were then allocated to residential household postcode sectors for each dataset separately. These unique identifications numbers were used for linking the child health survey data with the air pollution data based on matching postcode sectors. Individual data were checked manually in order to examine for any inconsistencies and outliers. Each entry was double checked for validation and graphs and tables were prepared to summarise the results. The site visit to the MAEI, arranged by the Sefton Council to learn more about the operational aspects of the environmental monitoring system and to get some idea on the methods used for deriving the emissions for NOx and PM_{10} and concentrations of NO_2 and PM_{10} was essential as part of the project and helped to understand the practical side of air pollution monitoring and measurement. Air Quality Index (AQI) has been usually used to inform the public about the short term monitored levels of pollutants. Since the present study used data based on modelled long term (annual) pollution levels, it was not measured in this present study.

3.12 Main outcome measures and exposure variables

The main outcome measures have been mentioned in section 3.8.1. The exposure variables were emissions of oxides of nitrogen (NO_X) and particulate matter (PM<10) and concentrations of NO_2 and PM_{10} and each emission and concentration category combined individually with categories for maternal smoking during pregnancy, paternal smoking during pregnancy and household smoking during pregnancy. The details of the air pollutant and pregnancy smoking combination categories have been included in section 3.8.4. The socio economic variables included single parent and employment status and breast feeding was also included. The definitions for outcome and exposure variables are mentioned in table 3.01.

3.13 Optical reading of questionnaires

The optical reading of the questionnaires was completed with the support of the Health Protection Agency, (Kirby) using the Read Soft software program. Reading included four

stages including the use of an optical scanner. This uses a software application which automatically captures and manages images and involves four stage - scanning, interpreting, verifying and transferring data to a target system. The major advantage compared to manual entry is that the forms were captured automatically without requiring manual data entry which was reduced by 90% or more. It was possible to make corrections when required in an efficient and user friendly manner with the option for the form to be edited directly on the screen. Once the data was ready in the word pad form it was directly transferred into a Microsoft excel table, and transferred into SPSS for further analysis after manual double checking of the complete data for any inaccuracies. Please refer Appendix C for the patient information document which accompanied the questionnaire, and Appendix B for the study specific questions extracted from the full questionnaire.

3.14 Data Analysis and statistical methods

The statistical analysis of data used SPSS software (SPSS 20) and Epi info software 2002 (Nutri software). Descriptive summary statistics were computed for study variables and comparisons were made using contingency tables (chi-square tests). Means, standard deviations (SD) or z scores were computed for anthropometric indices of weight, height and BMI using Epi - Info 2002 which was compared against the reference standard of the NCHS growth data (National Centre for Statistics Health, USA) (http://www.cdc.gov/growth charts). A z score of >1.04 and < 1.64 for body mass index (>85th centile and < 95th centile) was defined as overweight; of > 1.64 (>95th centile) as obese, (Bundred et al, 2001). Two methods were used for analysing the health data in relation to air pollution. The first method used specific cut off values for total NO_X and PM_{10} emissions and NO_2 and PM_{10} concentrations output and cross tabulation with child health and birth outcomes variables. Cut-offs used were 10 for NOx and 5 for PM_{10} (both in tonnes per annum) which separated the total emissions output data into two categories for both NOx and PM_{10}. Cut-offs used were 17.07 for NO_2 and 13.91 for PM_{10} (both in micrograms/m3) which separated the total concentrations output data into two categories for both NOx and PM_{10}. The cut off values for emissions and concentrations were finalised based on the cut off values used by the Air Quality team for categorising high and low emissions and concentrations areas in their emission and concentration maps, for the purpose of identifying Air Quality Management Areas, and also after further discussion with team members from Sefton council and Child health team. All the details of the definitions and cut off values for different air pollutants and pregnancy smoking categories and the combined

categories are summarised in table 3.01. Both these values were based on values corresponding to more than or equal to the 75th percentile for higher emissions and concentrations categories and less than or equal to 75th percentile for lower emissions and concentrations categories oxides of nitrogen and PM_{10} used specifically for Sefton air pollution data. The second method involved calculation of mean emission and concentrations outputs by postcode sector and comparison in areas with higher and lower birth and child health categories. Mean child growth indices including weight, height, body mass index and birth weight were calculated by emissions categories using these cut-off values. Similar analyses were also conducted for concentrations data. Multiple backward stepwise logistic regression methods were used to estimate odds ratios and their 95% CI for birth and child outcomes reported as significant from the univariate analysis. Data was adjusted for potential confounding factors identified in univariate analysis with a significance level of $P<0.05$. Population Attributable Risk (PAR) estimates and their 95% confidence intervals were also calculated for birth and child health outcomes, which showed significant association in relation to combined categories of pregnancy smoking exposures and air pollutants. Venn diagrams were used for the diagrammatic representation of the association between air pollution and health outcomes. Details of each method used are summarised below.

3.14.1 SPSS

The statistical analysis of the study data was performed using the SPSS (version 20) statistical package. The 2006 survey data was entered by optical reading and entered into SPSS dataset from excel file and text file created from the data read directly from the questionnaires using the optical reader. The air pollution data was entered into SPSS from an excel file and then linked to the child health survey SPSS data file using postcode sectors. The combined SPSS data file created by linking of community child health data with the emissions data for NOx and PM_{10}, and concentration data for NO_2 and PM_{10} using 3 digit postcodes sectors, and unique identification numbers was used for analysis. SPSS is a comprehensive and flexible statistical analysis software and data management system. SPSS was used for univariate analysis for cross tabulation of birth and child health outcomes in relation to air pollution and pregnancy smoking exposure indicators and for comparing means of emissions and concentrations for oxides of nitrogen and particulate matter in relation to birth and child health outcomes. Comparison of growth parameters in children in relation to air pollution and pregnancy smoking exposure and multiple logistic regression analysis by backward stepwise

regression method were also completed using SPSS. It was also used for interaction analysis, which assessed the interactions between the exposure factors and socio-economic status. Interaction analysis was needed for assessing the confounding effects of different exposure factors between them on birth and child health outcomes.

3.14.2 Structural equation modelling

Structural equation modelling (SEM) uses various types of models to depict relationships among observed variables, with the aim of providing a quantitative test of a hypothetical theoretical model. It helps to identify how sets of variables construct and how these constructs are related to each other (Schumaker and Randall E (2004). The main difference between SEM and the other statistical methods is that SEM can show the relationships among dependent variables. In SEM, more than one exogenous and endogenous variables are estimated simultaneously along with the estimation of causal relationships between endogenous variables.

The goal of SEM analysis is mainly to determine the extent to which the theoretical model is supported by sample data, and if identified, then more complex theoretical models can be hypothesised. SEM tests theoretical models using the scientific method of hypothesis testing to advance our understanding of complex relationships among constructs (Schumaker and Randall, 2004). SEM also makes use of confirmatory rather than exploratory modelling for building and testing statistical models and helps to estimate the relationships between multiple dependent, independent and latent variables. There are mainly two types of variables used for structural equational modelling which include latent and observed variables. The latent variables, constructs or factors are variables that are not directly observable or measured, whereas observed variables are directly observed. The relationships are usually based on theoretical considerations and evidence from previous studies. Structural equation modelling permits complex phenomenon with multiple variables to be modelled and tested considering measurement error, and for analysis of more advanced SEM theoretical models. It is also useful as an important statistical tool for evaluating complex relations in several research areas.

In the present study, basic structural equation models were designed to look at the complex relationships between air pollution and pregnancy smoking exposure and various birth and child health outcomes. The latent and observed variables were selected and all the SEM

models were created based on inclusion of variables which were significant from univariate analysis ($p<0.05$), and based on the exposure and outcome categories which showed significance after the logistic regression and interaction analysis. The complex interactions were first converted into a network of directional paths linking the variables and then assessed against multivariate data and the direct and indirect effects between the latent and observed variables were assessed. The underlying principles taken into consideration included justification of the model used, addressing missing data problems, using complete set of parameters with standard errors, use of correlation matrix, looking for discrepancies along with goodness of fit statistics.

There is existing data from studies on child health outcomes which have been reported previously using the SEM model. Huang et al (2017) in their study used structural equation modelling for quantifying the relationships and interactions among parental socioeconomic status, family food security level, child's food intake and certain aspects of parental feeding behaviour. However, the study was limited by its cross sectional nature. Hendrie et al (2012) in their study conducted in Adelaide, Australia among 157 primary school children used exploratory structural equation modelling to quantitatively model the complex relationships between the family environment, health behaviours and obesity.

3.14.3 Townsend score

It is not easy to measure poverty directly but it is possible to obtain measures of deprivation (Atkinson 1985a, Atkinson 1985b, Lewis and Ulph, 1988). Even though markers of poverty or of low social class are associated with many diseases and potential causes of disease, medical studies often fail to record sufficient information on the socio-economic status (Smith et al, 1998). The concept of deprivation covers the various conditions, independent of income, experienced by people who are poor, while the concept of poverty refers to a lack of income and other resources which equates to a lack of income and other resources which make those conditions at least highly likely (Townsend, 1988). Danesh et al (1999) recommended that postcode income estimates were easily available in Britain and could be useful markers of social class. The Townsend and Carstairs scores are considered useful measures of material deprivation, (Dolen et al, 1995). The level of household socio-economic status was calculated using the Townsend Deprivation Scores derived from the respondents postcodes. This utilised

MIMAS software (Manchester Information and Association Services), which converts UK postcodes directly into the deprivation scores. Townsend scores had been used in the previous survey analyses and for this reason, this socio-economic scoring method was also chosen for the 2006 survey analysis. The Townsend Deprivation Index consists of four population variables: percentage which includes the measurement of local unemployment, car ownership, overcrowding and home ownership (Gilthorpe, 1995). The Townsend Deprivation Index derived from postcodes, allowed the classification of the specific socio economic classes. The Townsend score values ranges from -6 to +12 with values below zero indicating an area of better socio- economic status. Values above zero indicate an area of lower socio-economic status. The scores are frequently divided into quartiles of -6 to -3 lower quartile (least deprived),-2 to 0 mid-lower quartile, 1-3 mid-upper quartile, 4-12 upper quartile (most deprived) (Townsend, 1988).

3.14.4 Population attributable risk estimation

Population attributable risk estimates (and 95% CI) were calculated for the birth and child outcome variables which showed significant association in relation to combined air pollution and pregnancy smoking exposure categories by backward stepwise logistic regression and was calculated using the formula:

$$PAR = P_{exp} (RR_{exp}-1) / [1 + P_{exp} (RR_{exp}-1)]$$

where P_{exp} is the prevalence of the outcome, and RR the relative risk and then multiplying it by 100 to give the percentage. Population Attributable Risk provides the incidence of a disease in a population (exposed and unexposed) which is due to exposure. In other terms it is the disease incidence in a population that would be eliminated if the exposure was removed. Adjusted odds ratios were used instead of relative risk for better results as the adjusted ratios take into consideration the effects of confounding factors, which is not the same with unadjusted odds ratios or relative risk. The decision to use adjusted odds ratio was also based on the final discussion with the statistician and study supervisors.

3.14.5 Venn diagrams

The Venn diagram was first introduced by the Hull born British philosopher and mathematician John Venn in 1881 (Venn, 1880). Venn diagram is basically just a diagram of intersecting and overlapping areas or circles demonstrating the relationships of sets based on set theory concepts and can be read easily (Henderson, 1963). Three circle Venn diagrams may be used as community tool for screening of various conditions and the relationship between three circles and elements in common can be used for comparison between two groups (Edward, 2004). There are two types of three circle Venn diagrams- proportional and non-proportional. In non-proportional Venn diagrams, these diagrams consist of circles of the same size, so that they were only symbolic of the groups they represented and do not indicate in any way their relative magnitudes (Mehdi, 2007; Soriano, 2003). Proportional Venn diagrams takes into consideration the relative magnitudes also. It can be used as a mechanism in various studies to compare and contrast two or three items. Venn diagrams are useful in showing graphically how the objects in the sets are similar, different or apparently distinct to each other and provides much more information to the researcher. Venn diagrams have been used in various studies for confirmation of various associations of medical disorders in community surveys (Viegi et al, 2004; Soriano, 2003). Tools for producing Venn diagrams include Venn diagram Plotter, SmartDraw, and ConceptDraw, Wivenn, DrawVenn, Ploticus, Venny and other softwares like Stata, GDM, DrawEuler and DrawVenn.

Venn diagram Plotter-version v1.5.4798 developed by Littlefield K and Monroe M has been used in the present study. The program includes a graphical user interface (GUI) where the user enters sizes and amount of overlap between the two (or three) lists (the overlap must be determined separately by the user). Venn diagram Plotter (2013) was available from http://ncrr.pnl.gov/ or http://www.sysbio.org/resources/staff/ accessed on 08-07-2013 Website: http://ncrr.pnl.gov/ or http://www.sysbio.org/resources/staff/. Proportional Venn diagrams were created using the above mentioned software. The proportional Venn diagrams were used in the study in order to illustrate the significant outcomes of public health importance from the final results in relation to the different combinations of air pollutants and pregnancy smoking exposures. This was also used to compare the area of outcome between each of the individual air pollutant and pregnancy smoking exposure categories and also in relation to their combined exposures.Venn diagram Plotter had been used as a standard method for generating

proportional Venn diagrams in previous studies which had been published in peer reviewed journals (Motts et al, 2014; Vladimirov et al, 2015).

3.14.6 Spatial mapping

Spatial mapping is a useful tool for studying spatial distribution of health outcomes, examination of diseases and their variations in different geographic areas. Mapping of diseases are helpful for visual representation of intricate geographic data, which in turn can be used for explanatory purposes, for presentation of disease maps to survey high risk areas and for further help with policy and resource allocation in these areas (Elliot et al 2004). GIS (Geographic Information System) software is a computerised data management system used to capture, store, manage, retrieve, analyse and display spatial maps. It uses a broad system of applications for combining digital maps and georeferenced data (Steiniger and Hunter, 2012).

3.14.6.1 Spatial mapping method

Results from the data analysis were used to create Spatial Maps. Spatial mapping was carried out using ArcGIS 10.2.2 software installed from the Computing Services Department, University of Liverpool. Spatial maps were created for the child health outcomes of public health importance in the study and which showed statistical significance after cross tabulation and logistic regression. Creating spatial maps was based on adding the postcode sector shape files for the UK and looking specifically into the Liverpool postcode sectors, which were available for downloading from the website: http://www.opendoorlogistics.com/downloads/. This was found by searching for the phrase 'Postcode sectors UK shape-file' and downloading the relevant file needed for spatial mapping. This file was imported into the ArcGIS ArcMap 10.2.2 software as a layer. Then excel data was downloaded on childhood obesity categories in relation to NO_X-PM_{10} emissions combined with maternal smoking during pregnancy (four categories for each based on 'Yes' or 'No' for each group) for the ten primary schools based in Sefton for which data on air pollution and maternal smoking during pregnancy was available. Data with missing postcode sectors were removed, and postcode sectors with multiple data points were summarised, based on selecting the mode of the smoking and air pollution and combining data for each postcode sector. This facilitated matching of the excel data with the postcode sector shape data for creating spatial maps of childhood obesity categories in relation to NO_X-PM_{10} emissions when combined with maternal smoking during pregnancy.The spatial

maps were created to illustrate the post code sector areas corresponding to the different combined air pollutant–pregnancy smoking categories of public health importance and colour coding these areas to differentiate them from each other.

3.15 Specific endpoints selected for analysis

All the birth and child health outcomes available were assessed in relation to individual air pollutant exposure categories and combined air pollutant and pregnancy smoking exposure categories for any significant association as part of the univariate analysis. The mean emissions for NOx and PM_{10} and the mean concentrations for NO_2 and PM_{10} in areas with high and low prevalence of all these birth and child health outcomes were also assessed. The outcomes which showed significance after the univariate analysis were included in the backward stepwise logistic regression analysis. For the population attributable risks and for structural equation modelling, relevant outcomes which showed significant association after the logistic regression analysis were included after discussion with the child health team. The outcomes which were significant in relation to the combined air pollution and pregnancy smoking exposure categories were used as the main endpoints in the final analysis including Venn diagram illustration and spatial mapping.

3.16 Confidentiality

Data collected on air pollution and selected child heath data for the present study was stored with personal password protection using encrypted USB storage. Data was available to myself, Professor Brabin and Dr Brian Faragher. The analysis was anonymised and confidentiality was strictly maintained using study codes alone and no individual names were included in the dataset. The data were kept in a secure place in the investigators personal locker. All data collected from the database was anonymised with no linkage to participant records. All computerised data was password protected.

3.17 Ethical considerations

Permission was obtained from the Ethical Committees of the Royal Liverpool Children's Hospital for the 2006 child health survey. Only 3 digit postcode identification was approved for statistical analysis. Permission to use the air pollution emission and concentration

data was obtained from the Environmental Protection Department of Sefton Borough Council. The Ethical approval letters from Royal Liverpool Childrens Hospital Ethical Committee and Sefton Council are included in Appendices J and K.

CHAPTER 4

RESULTS

4.0 Introduction to results

In this chapter on results, the population characteristics, questionnaire compliance, information on missing values, child and birth characteristics, demographic, anthropometric, respiratory characteristics and parental smoking patterns, study characteristics of Sefton air pollution data, univariate analysis of air pollution exposure and combined air pollution and pregnancy smoking exposure including combination with smoking dose categories as risk factors for birth and child health outcomes along with summary tables are summarised. This chapter also includes interaction analysis, comparing means analysis, growth parameter patterns comparison in relation to air pollutant categories and combined categories of air pollution and pregnancy smoking exposure followed by logistic regression, population attributable risk, Venn diagrams, Structural equation modelling and spatial mapping.

4.1 Population characteristics

4.1.1 Questionnaire compliance

Table 4.01 summarises parental compliance for school questionnaire completion for 10 Liverpool schools included in the present air pollution study. Compliance ranged between 18.9% - 55.3% for individual schools, with an average compliance of 32.9 % (792/2405). Approximately 3% of the questionnaires were not distributed due to school absenteeism.

Table 4.01 Parental questionnaire compliance

School *	Name	Compliance	(%)
1	Netherton Park	36/170	21.2
2	Roberts Primary	46/200	23.0
3	St Bennets	70/300	23.3
4	St Edmunds	127/310	41.0
5	St Monicas	249/450	55.3
6	St James	51/200	25.5
7	Star of the sea	48/150	32.0
8	St Joan of Arc	35/185	18.9
9	Thomas Grey	85/290	29.3
10	William Gladstone	45/150	30.0
	All Schools	792/2405	32.9

Brackets = percentage; * school code

4.1.2 Missing values (non-responders to specific questions)

Missing responses were highest for questions on food allergy (64.65%), pet allergy (64.39%), hay fever (62.75%), eczema (60.86%), paternal smoking during pregnancy (28.66%) and for computed variables CWB triad (40.12%), childhood obesity (27.15%) and childhood overweight (27.15%). Computed variables indicating overweight and obesity were computed using BMI z scores adjusted for age and sex, so the quantity of missing observations for these variables were affected by the numbers of missing values for height, weight age and/or sex. The lowest number of missing values were for the child's sex (0%) followed by child age (8.7%).

The proportion of individuals responding was lower for the 2006 survey than for previous surveys. Parental questionnaire compliance was 92% (1872/2035) in 1991, 87.4% (3746/4288) in 1993, and 78.1% (1964/2514) in 1998; these figures compare to 30.3% (1074/3540) for all 15 schools surveyed in 2006 and 32.9% (792/2405) for the 10 schools surveyed in 2006 that were included in this dissertation. The missing and valid numbers for each variable in the table below (Table 4.02) are out of a total of 792.

Table 4.02 Missing values for the study data

Variable	Obtained	Missing	Percentage
Gender	792	0	0
Child age	723	69	8.71
NO_X total emissions	706	86	10.86
PM_{10} total emissions	706	86	10.86
Average NO_2 concentrations	706	86	10.86
Average PM_{10} concentrations	706	86	10.86
MSDP	704	88	11.11
ADHD	703	89	11.24
DDA	699	93	11.74
Bronchitis	699	93	11.74
HSDP	698	94	11.87
Breast feeding	697	95	11.99
Croup	692	100	12.62
Single parent	691	101	12.75
GMSDP	689	103	13.01

Maternal asthma	689	103	13.01
Child height	688	104	13.13
Preterm	686	106	13.38
Well controlled asthma	108	17	13.60
Child weight	677	115	14.52
Postcode sectors	668	124	15.66
Mean NOx emissions	668	124	15.66
Mean PM_{10} emissions	668	124	15.66
Excess cough	663	129	16.29
Breathlessness last 12months	663	129	16.29
Ever wheeze	663	129	16.29
GFSDP	660	132	16.67
Stunting	651	141	17.80
Height for age z score	651	141	17.80
Weight for age z score	642	150	18.94
Allergy	635	157	19.82
IUGR	627	165	20.83
Birthweight	616	176	22.22
LBW	616	176	22.22
Paternal asthma	612	180	22.73
Townsend score	606	186	23.48
Lower socio economic status	606	186	23.48
BMI z score	577	215	27.15
Overweight	577	215	27.15
Obesity	577	215	27.15
PSDP	565	227	28.66
CWB triad	397	266	40.12
Eczema	310	482	60.86
Hay fever	295	497	62.75
Pet allergy	282	510	64.39
Food allergy	280	512	64.65

MSDP: Maternal smoking during pregnancy
PSDP: Paternal smoking during mother's pregnancy
HSDP: Household member smoking during mother's pregnancy
GMSDP: Grandmother smoked during pregnancy period
GFSDP: Grandfather smoked during pregnancy period
BMI z-score: Body Mass Index z-score

NOx: Oxides of Nitrogen
NO_2: Nitrogen dioxide
PM_{10}: Particulate matter less than 10 microns
LBW: Low birth weight
DDA: Doctor diagnosed asthma
IUGR: Intrauterine Growth Restriction
ADHD: Attention Deficit Hyperactivity Disorder
CWB triad: symptom triad of excess cough, ever wheeze and breathlessness in the last 12 months
Lower Socio economic status: Upper Quartile: Townsend score >4)

4.1.3 Child and birth characteristics

4.1.3.1 Age and sex distribution

Median age was 84.0 (SD 22.8) months. The number of females was 403 (50.9%).

4.1.3.2 Preterm birth and low birthweight

Of the 616 children with reported birthweights

- 70 (11.4%) were LBW, 69 (11.2%) were high birthweight and the remaining 477 (77.4%) were normal birthweight;
- 28 (9.2%) out of 305 males and 42 (13.5%) out of 311 females were LBW.

Of the 686 children for whom gestational age at birth was reported:

- in total, 105 (15.3 %) were preterm;

50 (14.8%) out of 337 males and 55 (15.8%) out of 349 females were preterm.

4.1.4. Demographic characteristics

Of the 606 children for whom a Townsend score of socio economic status was

available:

- 556 (91.7%) were classified in the upper quartile - this corresponds to a lower socio-economic class category (+4 to +12 based on Townsend quartiles);
- no children were classified in the upper class category (-6 to -3);
- 34 (5.6%) belonged to the mid-lower (-2 to 0) quartile;
- 16 (2.7%) were placed in the mid-upper (+1 to +3) socio-economic category.

There were no significant differences in age and anthropometric measurements (weight, height and birthweight) between those children for whom socio-economic status data were and were not available. Other socio-economic variables recorded included: percentage with a single

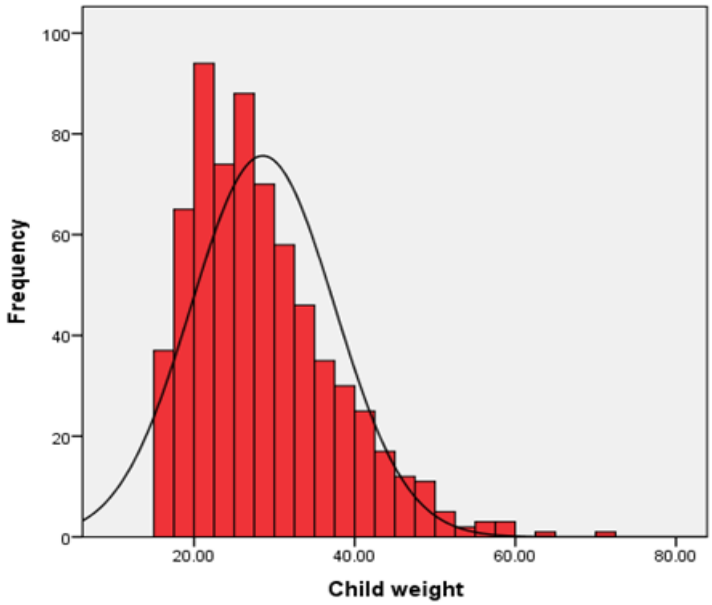
parent of (36.8%; 254/691), maternal (51.3%; 354/691) and paternal employment (61.7%; 371/601).

4.1.5 Anthropometric characteristics

4.1.5.1 Weight:

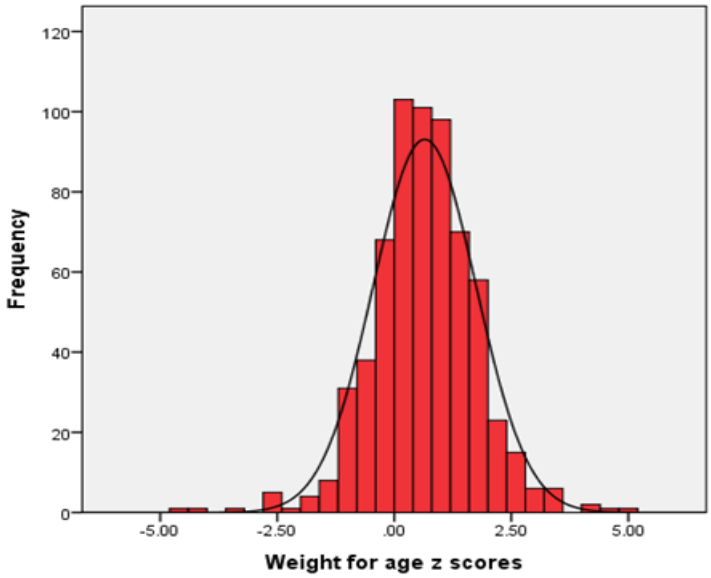
Median weight was 27.30 kg (IQR 21.65-33.40) and mean weight for age z-score was 0.65 (1.10). Figures 4.01 and 4.02 show the distribution of child weight, and weight for age z-scores.

Figure 4.01 Weight distribution



Median 27.30; Interquartile range: 21.65-33.40

Figure 4.02 Distribution of weight for age z scores

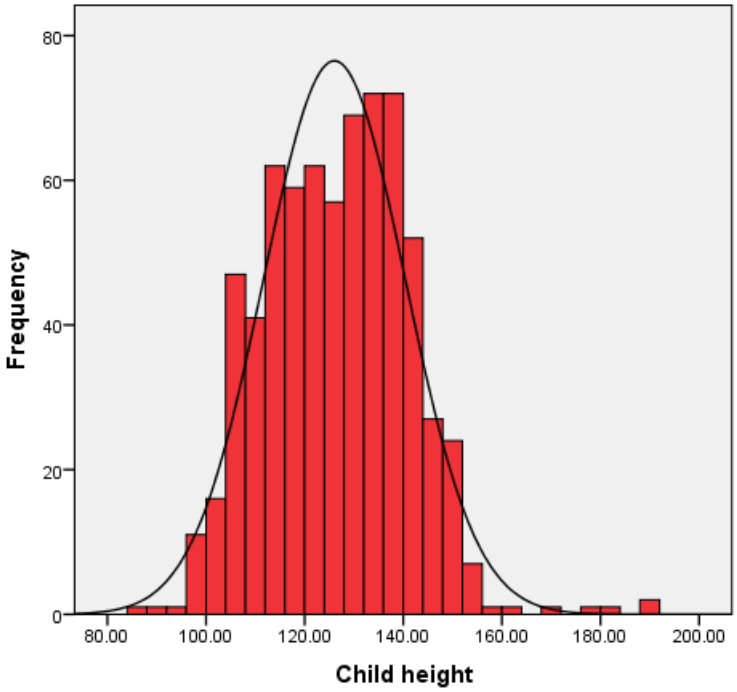


Mean 0.65; SD 1.10

4.1.5.2 Height:

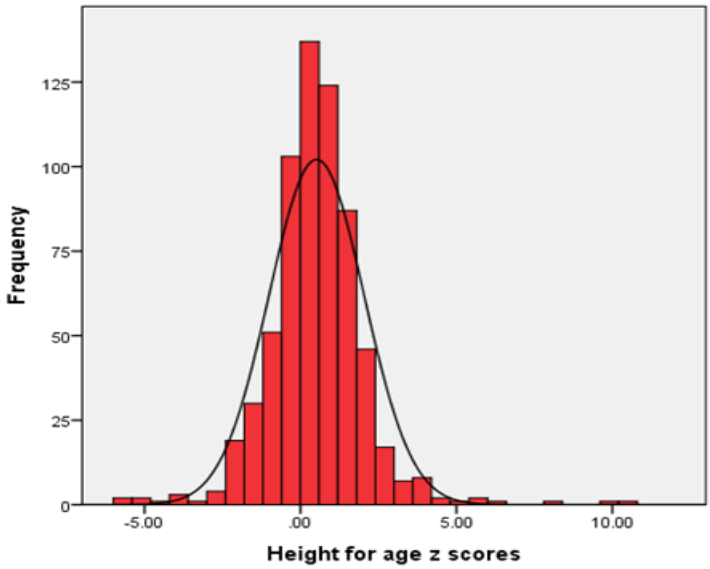
Mean actual height was 126.03 (SD 14.35) cms and mean height for age z score was 0.52 (SD 1.53). Figures 4.03 and 4.04 show the distribution of child height and height for age z scores.

Figure 4.03 Distribution of child height



Mean: 126.03; SD: 14.35

Figure 4.04 Distribution of child height for age z scores

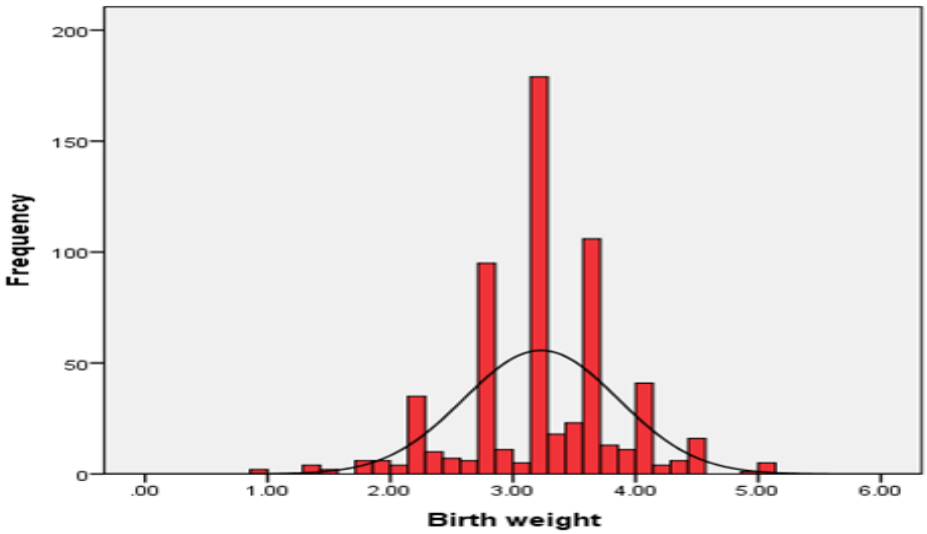


Mean: 0.52; SD: 1.53

4.1.5.3 Birthweight:

Mean reported birthweight was 3.23 Kgs (SD 0.63; range 0.91 to 5.09). The distribution of recorded birthweights is shown in figure 4.05; the peaks in this figure are a consequence of rounding in the original birthweight units (6, 6.5, 7, 7.5, 8, 8.5 lbs etc.).

Figure 4.05 Distribution of birthweights

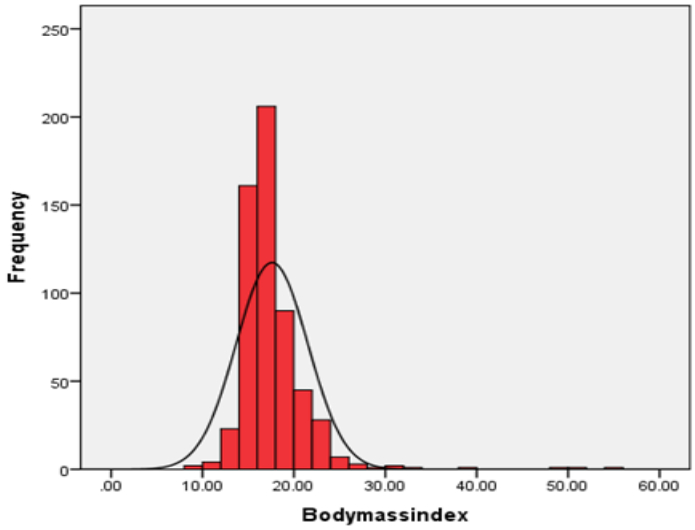


Mean 3.23; SD 0.63

4.1.5.4 Body mass index (BMI):

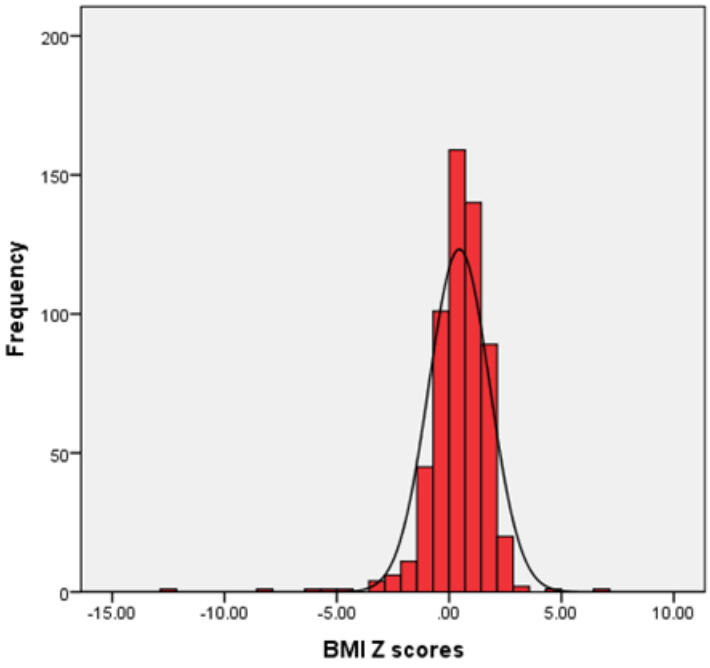
Mean actual BMI was 17.60 (SD 3.92) kg/m^2 and mean BMI z -score was 0.45 (SD 1.35). Figures 4.06 and 4.07 show the distributions of BMI and BMI z-scores.

Figure 4.06 Distribution of Body mass indices



Mean 17.60; SD 3.92

Figure 4.07 Distribution of BMI for age z-scores



Mean 0.45; SD 1.35

Prevalence estimates for obesity and overweight were 13.9% (80/577) and 29.5 % (170/577) respectively. Based on a BMI z score < -1.04, 8.2 % (48/584) of participants were underweight. Table 4.03 summarises the anthropometric characteristics.

Table 4.03 Anthropometric characteristics of study children

Characteristic	Valid	Missing	Mean (SD)	Range
Weight (Kg)	677	115	27.65*	21.65 – 33.40^
Weight / age z -score	642	150	0.65 (1.10)	-4.51 - + 5.18
Height (cms)	688	104	126.03 (14.35)	84 - 191
Height /age z -score	651	141	0.52 (1.53)	- 5.71 - + 10.21
Birth weight (kg)	616	176	3.23 (0.63)	0.91 – 5.09
Body Mass Index(BMI) (kg/m2)	577	215	17.60 (3.92)	8.64 – 55.88
BMI z score	577	215	0.45 (1.35)	-12.69 -+7.10

***Median used ^ Interquartile range**

4.1.6 Respiratory characteristics based on symptom profiles

Table 4.04 Prevalence of respiratory characteristics

Characteristics	Prevalence	% (95% CI)†
Doctor diagnosed asthma	125 / 699	17.9 (15.1 - 20.9)
Wheezing	128 / 663	19.3 (16.4 - 22.5)
Excess cough	124 / 663	18.7 (15.8 - 21.9)
Breathlessness	48 / 663	7.2 (5.4 - 9.5)
C+W+B+	37 / 397	9.3 (6.6 - 12.6)
Asthma well controlled*	57 / 108	52.7 (44.9 – 71.4)
Allergies**	129 / 635	20.3 (17.3 - 23.7)
Croup	67 / 691	9.7 (7.6 - 12.2)
Maternal asthma	91 / 689	13.2 (10.8 - 16.0)
Paternal asthma	58 / 612	9.5 (7.3 - 12.1)
Hay fever	81 / 295	27.5 (25.2 -29.8)

C+W+B+: Cough, wheezing and breathlessness symptom triad

*: Proportion of those with asthma in whom it was well controlled

**: Does your child have any allergies?

†: exact binomial 95% confidence intervals

A total of 91 / 689 (13.2%) of mothers reported themselves as asthmatic compared with 58 / 612 (9.5%) of fathers. Table 4.04 summarises the prevalence estimates for childhood respiratory symptoms including doctor diagnosed asthma, and parental asthma. Prevalence estimates for specific respiratory symptoms were 19.3% (128/663), 18.7% (124/663), and 7.2% (48/663) respectively for ever wheeze, excess cough and breathlessness. Prevalence of asthma in children, defined by the symptom triad of excess cough, wheezing and breathlessness was 9.3% (37/397).

4.1.7 Parental smoking patterns

Table 4.05 Prevalence of parental smoking during pregnancy

Smoking status*	Prevalence	% (95% CI)†
Maternal	179/704	25.4 (22.2 - 28.8)
Heavy maternal	19/278	6.8 (4.2 - 10.5)
Paternal	202/565	35.8 (31.8 - 39.9)
Heavy paternal	20/269	7.4 (4.6 - 11.2)
Grandmother	280/689	40.6 (36.9 - 44.4)
Grandfather	205/660	31.1 (27.5 - 34.7)
Any household smoker	305/698	43.7 (40.0 - 47.5)

†: exact binomial 95% confidence intervals

Parental smoking exposure prevalence during pregnancy is shown in table 4.05. The prevalence of paternal smoking was higher than that for maternal smoking (35.8% versus 25.4%, p <0.001); 43.7% of household had a smoker during the period of mother's pregnancy. A higher proportion of grandmothers (40.6%) than grandfathers (31.1%) smoked during their daughter's pregnancy (p<0.001).

4.2 Sefton air pollution data-study characteristics

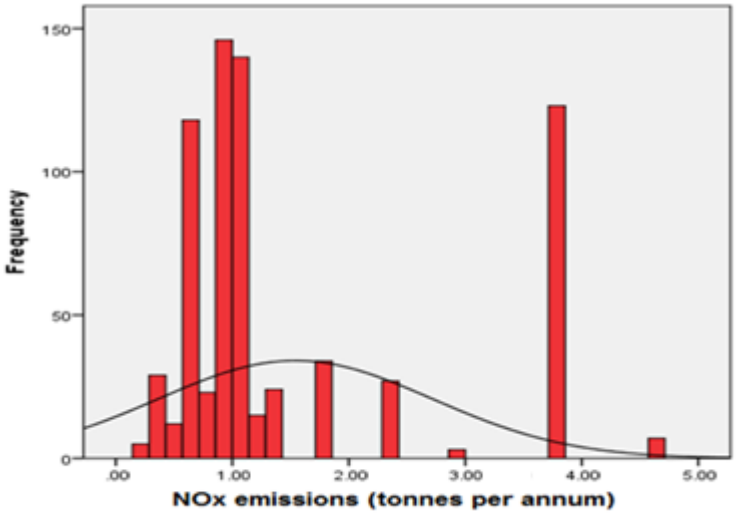
The study characteristics of the air pollution data from the Environment Protection Department, Sefton Council are summarised below. The air pollution data includes the emissions data for PM_{10} and NOx and the concentrations data for PM_{10} and NO_2.

4.2.1 Emissions for oxides of nitrogen (NO_x) and PM_{10}

The distribution of the emissions for oxides of nitrogen (NOx) and PM_{10} are summarised below.

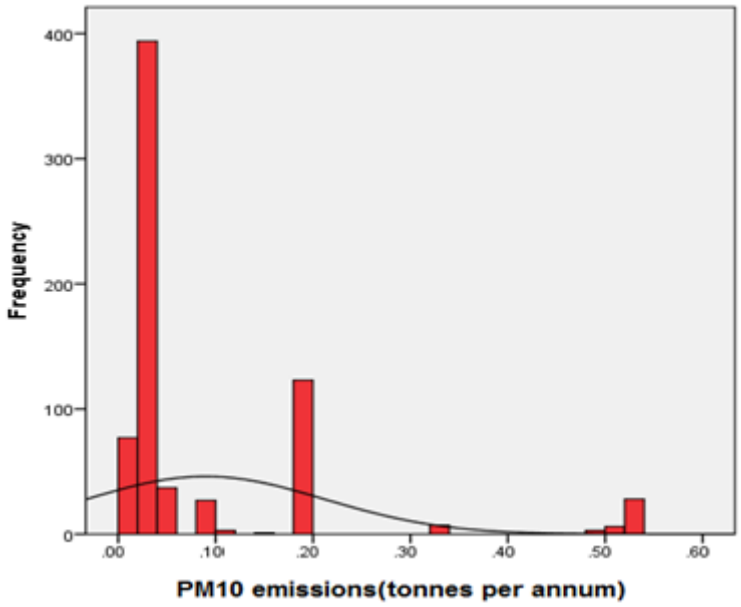
4.2.1.1 Distribution in NOx and PM_{10} emissions

Figure 4.08 Distribution of NOx emissions



Median NO_X emissions : 1.08 (IQR 0.76-1.84)

Figure 4.09 Distribution of PM_{10} emissions



Median PM_{10} emissions : 0.04 (IQR 0.03-0.05)

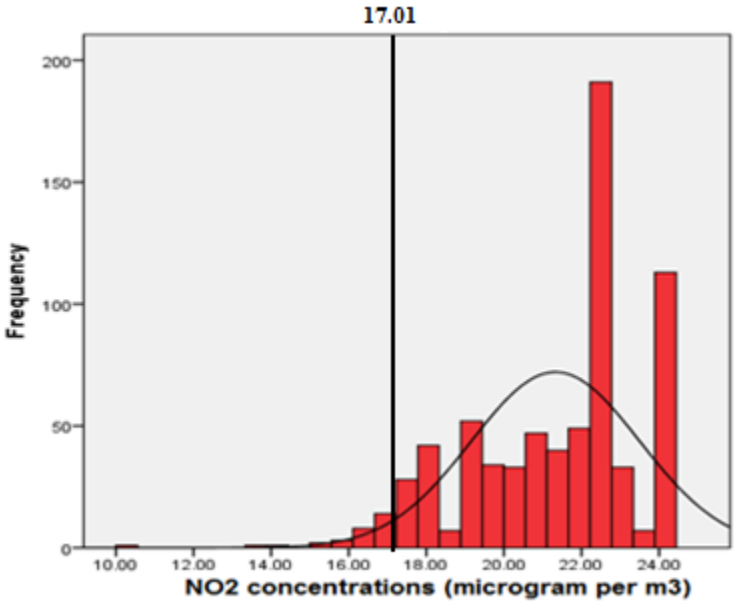
Median NO_X emissions were 1.08 (IQR 0.76-1.84) tonnes per annum and median PM_{10} emissions were 0.04 (IQR 0.03-0.05) tonnes per annum (Figures 4.08 and 4.09); both distributions were positively skewed. Highest median emissions for NOx and PM_{10} emissions were reported in the L20 1 post code sector.

4.2.2 Concentrations of oxides of nitrogen (NO_2) and PM_{10}

The distribution of the average concentrations for nitrogen dioxide (NO_2) and PM_{10} are summarised below.

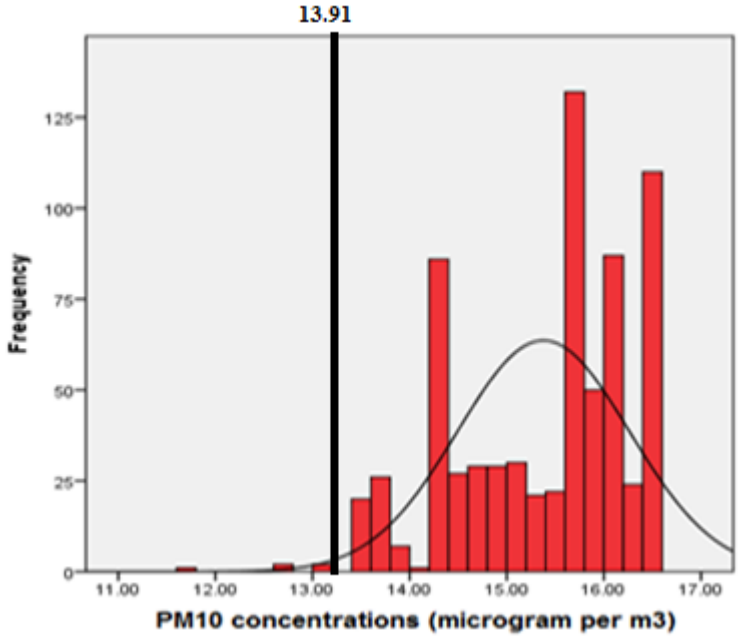
4.2.2.1 Distribution in NO_2 and PM_{10} concentrations

Figure 4.10 Distribution of NO_2 concentrations



Median NO_2 concentrations: 22.15 (IQR 19.73-22.77)

Figure 4.11 Distribution of PM_{10} concentrations



Median PM_{10} concentrations: 15.65 (IQR 14.66-16.03)

Median NO_2 concentrations were 22.15 (IQR 19.73-22.77) micrograms per m^3 and median PM_{10} concentrations were 15.65 (IQR 14.66-16.03) micrograms per m^3 (Figures 4.10 and 4.11); both distributions were negatively skewed. Higher median concentrations for NO_x and PM_{10} were reported in the L20 8 and L20 9 postcode sector respectively.

Table 4.06 Summary of median values for air pollution indicators

Air pollution indicator	Median (interquartile range)
NO_x emissions	1.08 (0.76-1.84) tonnes per annum
PM_{10} emissions	0.04 (0.03-0.05) tonnes per annum
NO_2 concentration	22.15 (19.73-22.77) micrograms per m^3
PM_{10} concentration	15.65 (14.66-16.03) micrograms per m^3

The median values for air pollution indicators are summarised in table 4.06. Figures illustrating the spatial maps of NO_x and PM_{10} emissions and NO_2 and PM_{10} concentrations (showing highest and lowest ranges) across the study area are shown by post code sectors in Appendices D, E F and G (Figures A,B,C,D,E). The location area of the schools as an overlay has been illustrated in the previous Methodology chapter and in figure 3.01. The black outlined rectangle area in these maps show the areas of the 10 Liverpool primary schools included in this study.

4.2.3 Prevalence of air pollution

The prevalence of air pollution categories and their combinations are shown in table 4.07.

Table 4.07 Prevalence of air pollution

Air pollution category	Prevalence (2006)	Prevalence (2008)
High NO_x emissions	458/668 (68.6)	449/617 (72.8)
High PM_{10} emissions	142/668 (21.3)	139/609 (22.8)
High NO_x concentrations	686/706 (97.2)	Data not available
High PM_{10} concentrations	648/706 (91.8)	Data not available
High NO_x-PM_{10} emissions	139/668 (20.8)	136/609 (22.3)
High NO_2-PM_{10} concentrations	646/706 (91.5)	Data not available

High NOx emissions: NOx emissions > 10 (in tonnes per annum)
High PM_{10} emissions: PM_{10} emissions > 5 (in tonnes per annum)
High NOx-PM_{10} emissions: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum)
High NO_2 concentrations NO_2 concentrations > 17.01 in microgram per m3
High PM_{10} concentrations: PM_{10} concentrations > 13.91in microgram per m3
High NO_2-PM_{10} concentrations NO_2 concentrations > 17.01 + PM_{10} concentrations > 13.91in microgram per m3
Brackets: Percentage
1:2006; 2:2008

The prevalence of high NO_x emissions was greater than that for high PM_{10} emissions (68.6% versus 21.3%, p <0.001). The corresponding prevalence levels for high NO_x concentrations and high PM_{10} concentrations were 97.2% and 91.8% respectively. For the combined categories, the prevalence levels were 21.3% and 91.5% for high NO_x-PM_{10} emissions and high NO_2-PM_{10} concentrations respectively. For the year 2008, two years after the study, the air pollution data showed increases in the prevalence levels for high NO_x emissions, high PM_{10} emissions and high NO_x-PM_{10} emissions when compared to 2006, although the changes were not statistically significant; comparative data was not available for high NO_2 concentrations, high PM_{10} concentrations and high NO_x-PM_{10} concentrations.

4.2.4 Prevalence of combined air pollution and pregnancy smoking exposure

The combined prevalence levels for air pollution and pregnancy smoking exposure categories are detailed in table 4.08

Table 4.08 Prevalence of combined air pollution and pregnancy smoking levels

Emissions / concentrations	Smoking	Emissions level / smoking category: n (%)				Totals
		H / H	H / L	L / H	L / L	
NO_x emissions	MSDP	112 (25.7)	324 (74.3)	46 (22.9)	155 (77.1)	637
PM_{10} emissions	MSDP	47 (34.1)	91 (65.9)	111 (22.2)	388 (77.8)	637
NO_2 concentrations	MSDP	169 (25.9)	483 (74.1)	3 (15.8)	16 (84.2)	671
PM_{10} concentrations	MSDP	164 (26.7)	451 (73.3)	8 (14.3)	48 (85.7)	671
NO_x emissions	PSDP	119 (34.6)	225 (65.4)	59 (35.5)	107 (64.5)	510
PM_{10} emissions	PSDP	49 (47.1)	55 (52.9)	129 (31.8)	277 (68.2)	510
NO_2 concentrations	PSDP	191 (36.2)	336 (63.8)	3 (21.4)	11 (78.6)	541
PM_{10} concentrations	PSDP	183 (37.0)	311 (63.0)	11 (23.4)	36 (76.6)	541
NO_x emissions	HSDP	183 (42.2)	251 (57.8)	87 (43.3)	114 (56.7)	635
PM_{10} emissions	HSDP	68 (50.4)	67 (49.6)	202 (40.4)	298 (59.6)	635
NO_2 concentrations	HSDP	291 (44.6)	361 (55.4)	4 (21.1)	15 (78.9)	671
PM_{10} concentrations	HSDP	280 (45.4)	337 (54.6)	15 (27.8)	39 (72.2)	671

High NO_x emissions: NO_x emissions > 10 (in tonnes per annum)
High PM_{10} emissions: PM_{10} emissions > 5 (in tonnes per annum)
High NO_x-PM_{10} emissions: NO_x emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum)
High NOx concentrations NOx concentrations > 17.01 in microgram per m3
High PM_{10} concentrations: PM_{10} concentrations > 13.91in microgram per m3
High NOx-PM_{10} concentrations NO_x concentrations > 17.01 + PM_{10} concentrations > 13.91in microgram per m3
MSDP: Maternal smoking during pregnancy
PSDP: Paternal smoking during pregnancy

HSDP: Household member smoking during pregnancy
Brackets: Percentage; H: High; L: Low

The prevalence of high NOx emissions combined with HSDP were higher (42.2) when compared to high NOx emissions combined with PSDP (34.6) and MSDP (25.7) respectively. The corresponding prevalence of high PM_{10} emissions were showing similar trends, when combined with different pregnancy smoking exposure categories 50.4 %, 47.1 % and 34.1 % respectively for HSDP, PSDP and MSDP respectively and for combined NO_2 concentration pregnancy smoking categories it was 44.6, 36.2, and 25.9 respectively. For combined PM_{10} concentration category, it was 45.4, 37.0 and 26.7 respectively for HSDP, PSDP and MSDP.

4.2.5 Prevalence of combined air pollution and pregnancy smoking dose exposure

The prevalence of combined air pollution and pregnancy smoking dose exposure categories and their combinations are shown in table 4.09

Table 4.09 Prevalence of combined air pollution and pregnancy smoking dose levels

Emissions / concentrations	Smoking†	Emissions level / smoking category: n (%)				Totals
		H / H	H / L	L / H	L / L	
NO_x emissions	MSDP	8 (5.3)	142 (94.7)	8 (9.8)	74 (90.2)	232
PM_{10} emissions	MSDP	2 (4.3)	45 (95.7)	14 (7.6)	171 (92.4)	232
NO_x concentrations	MSDP	15 (6.3)	226 (93.8)	1 (11.1)	8 (88.9)	250
PM_{10} concentrations	MSDP	14 (6.2)	213 (93.8)	2 (8.7)	21 (91.3)	250
NO_x emissions	PSDP	8 (5.4)	140 (94.6)	6 (7.4)	75 (92.6)	229
PM_{10} emissions	PSDP	4 (7.7)	48 (92.3)	10 (5.6)	167 (94.4)	229
NO_x concentrations	PSDP	13 (5.5)	222 (94.5)	3 (30.0)	7 (70.0)	245
PM_{10} concentrations	PSDP	9 (4.1)	211 (95.9)	7 (28.0)	18 (72.0)	245

High NO_x emissions: NO_x emissions > 10 (in tonnes per annum)
High PM_{10} emissions: PM_{10} emissions > 5 (in tonnes per annum)
High NO_x-PM_{10} emissions: NO_x emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum)
High NO_x concentrations NO_x concentrations > 17.01 in microgram per m3
High PM_{10} concentrations: PM_{10} concentrations > 13.91in microgram per m3
High NO_x-PM_{10} concentrations NOx concentrations > 17.01 + PM10 concentrations > 13.91in microgram per m3
Heavy maternal smoking: Maternal smoking more than 10 cigarettes/ day;
Heavy paternal smoking: Paternal smoking more than 10 cigarettes/ day;
Brackets: Percentage
†: Smoking dose H = heavy; L = not heavy

The prevalence of high NOx emissions combined with heavy paternal smoking was slightly higher (5.4) when compared to high NOx emissions combined with heavy maternal smoking (5.3), whereas the prevalence of high PM_{10} emissions combined with heavy maternal smoking was lower (4.3) when compared to high PM_{10} emissions combined with heavy paternal smoking (7.7). For the concentrations data, the prevalence of high NO_2 concentrations

and high PM_{10} concentrations separately combined with heavy maternal smoking were higher when compared with the heavy paternal smoking (6.3 versus 5.5) and (6.2 versus 4.1) respectively.

4.3 Univariate analysis and estimation of odds ratios for birth and child health outcomes in relation to air pollution indicators and combined air pollution and pregnancy smoking exposure indicators

The relationships between the pollution emission / concentration measures, clinical measures and demographic measures should, ideally, be examined in a small number of multivariate analyses involving all (or, at least, a substantial subset) of these measures in appropriate combinations.

Unfortunately, however, the considerable numbers of missing observations (see section 4.1.2 above) makes such an analysis unreliable. Complex multivariate analyses involving many potential covariates / factors would be based inevitably on only a small subset of respondents – and these subsets would almost certainly have average characteristics somewhat different from those of the whole study cohort.

While a small number of such analyses were completed and are reported later, it was considered more sensible to, initially, obtain an overview of the associations between the pollution, clinical and demographic measures using a series of carefully selected sub-group analyses. It is these that are presented in this section.

Because of the possible exploratory nature of these subgroup analyses, statistical significance was set at the 10% level ($p<0.10$). Associations significant at the conventional 5% level are shown in bold, while associations with p-values between 5% and 10% are shown in italics.

4.3.01 High NOx emissions

Table 4.10 Associations between NOx emissions categories and birth and child health outcomes

Outcome	↑NOx E	↓ NOx E	p value	Unadjusted OR (95% CI)
Hay Fever	**46/195 (23.6)**	**30/73 (41.1)**	**0.006**	**0.443 (0.250-0.784)**
Croup	**32/438 (7.3)**	**26/196 (13.3)**	**0.024**	**0.515 (0.298-0.891)**
M: F ratio (%)	46.5:53.5	52.4:47.6	0.182	1.265 (0.912-1.754)[fb]
Eczema	63/208 (30.3)	27/707 (38.6)	0.237	0.692 (0.393-1.217)
Obesity	51/355 (14.4)	18/169 (10.7)	0.271	1.407 (0.795-2.493)
IUGR	15/390 (3.8)	10/170 (5.9)	0.275	0.640 (0.282-1.455)
Ever wheeze	84/416 (20.2)	32/186 (17.2)	0.435	1.218 (0.777-1.909)
Breathlessness	31/416 (7.5)	11/186 (5.9)	0.604	1.281 (0.629-2.607)
Bronchitis	33/435 (7.6)	14/202 (6.9)	0.871	1.102 (0.576-2.109)
Stunting	15/383 (3.9)	8/178 (4.5)	0.820	0.866 (0.360-2.082)
Allergy	84/405 (20.7)	39/173 (22.5)	0.658	0.899 (0.585-1.382)
CWB triad	20/230 (8.7)	7/105 (6.7)	0.667	1.333 (0.546-3.258)
Overweight	104/355 (29.3)	49/169 (29.0)	1.000	1.015 (0.678-1.519)
Excess cough	78/416 (18.8)	32/186 (17.2)	0.732	1.111 (0.706-1.748)
DDA	75/433 (17.3)	36/203 (17.7)	0.911	0.972 (0.627-1.506)
Preterm birth	70/431 (16.2)	31/194(16.0)	1.000	1.020 (0.643-1.618)
LBW	32/435 (7.4)	15/192 (7.8)	0.870	0.937 (0.495-1.774)
ADHD	12/432 (2.8)	6/200 (3.0)	1.000	0.924 (0.342-2.498)

OR: Odds Ratio
M: F Male: Female
LBW: Low birthweight: Birthweight < 2.5kg)
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
High (↑) NOx emissions: NOx emissions >10 tonnes per annum
Low (↓) NOx emissions: NOx emissions ≤10 tonnes per annum
Preterm: Reported as preterm by mothers in questionnaire
E: Emissions
Fb-for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to high versus those with exposure to low NOx emissions. Significantly decreased odds for hay fever and croup were observed in the high NOx emissions group.

4.3.02 High NO_2 concentrations

Table 4.11 Associations between NO_2 concentrations categories and birth and child health outcomes

Outcome	↑ NO_2 C	↓ NO_2 C	p value	Unadjusted OR 95% CI
Preterm birth	**95/640 (14.8)**	**7/20 (35.0)**	**0.024**	**0.324 (0.126-0.832)**
Bronchitis	51/654 (7.8)	3/19 (15.8)	0.190	0.451 (0.127-1.600)
Obesity	73/539 (13.5)	0/14 (0)	0.234	Cannot be calculated
Breathlessness	45/621 (7.2)	0/18 (0)	0.630	Cannot be calculated
Stunting	27/576 (4.2)	0/17 (0)	1.000	Cannot be calculated
CWB triad	26/345 (7.5)	1/10 (10)	0.551	0.734 (0.089-6.016)
IUGR	25/578 (4.3)	1/17 (5.9)	0.537	0.723 (0.092-5.673)
Overweight	159/539 (29.5)	3/14 (21.4)	0.767	1.534 (0.422-5.573)
LBW	50/645 (7.8)	2/18 (11.1)	0.645	0.672 (0.150-3.007)
M: F ratio (%)	48.4 : 51.6	40.0 : 60.0	0.503	0.711 (0.287-1.761)[fb]
Excess cough	116/621 (18.7)	4/18 (22.2)	0.758	0.804 (0.260-2.487)
Ever wheeze	119/621 (19.2)	4/18 (22.2)	0.762	0.830 (0.268-2.566)
Allergy	124/592 (20.9)	3/18 (16.7)	1.000	1.325 (0.378-4.641)
Croup	60/650 (9.2)	1/18 (5.6)	1.000	1.729 (0.226-13.219)
Hay fever	77/275 (28.0)	2/9 (22.2)	1.000	1.361 (0.277-6.697)
DDA	119/654 (18.2)	2/19 (10.5)	0.551	1.891 (0.431-8.294)
ADHD	18/649 (2.8)	0/19 (0)	1.000	Cannot be calculated
Eczema	92/287 (32.1)	3/9 (33.3)	1.000	0.944 (0.231-3.857)

OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg)
M: F Male: Female
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m3
Low (↓) NO_2 C: NO_2 concentrations $\leq$ 17.07 microgram per m3
Preterm: Reported as preterm by mothers in questionnaire
C: Concentration
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to high versus those with exposure to low NO_2 concentrations. A significantly decreased odds of being preterm was observed in the high NO_2 concentrations group.

4.3.03 High PM_{10} emissions

Table 4.12 Associations between PM_{10} emissions categories and birth and child healh outcomes

Outcome	↑ PM_{10} E (%)	↓PM_{10} E (%)	p value	Unadjusted OR(95% CI)
Ever wheeze	**36/133 (27.1)**	**80/469 (17.1)**	**0.013**	**1.805 (1.149-2.836)**
Croup	*7/138 (5.1)*	*51/496 (10.3)*	*0.066*	*0.466 (0.207-1.052)*
Breathlessness	*14/133 (10.5)*	*28/469 (6.0)*	*0.082*	*1.853 (0.946-3.631)*
Bronchitis	14/138 (10.1)	33/466 (6.6)	0.196	1.594 (0.828-3.672)
M: F ratio (%)	42.3 : 57.7	50.0 : 50.0	0.108	1.366 (0.940-1.988)[fb]
IUGR	3/128 (2.3)	22/432 (5.1)	0.229	0.447 (0.132-1.519)
Obesity	18/109 (16.5)	51/415 (12.3)	0.265	1.412 (0.787-2.533)
Preterm birth	18/134 (13.4)	83/491 (16.9)	0.358	0.763 (0.440-1.322)
CWB triad	8/80 (10)	19/255 (7.5)	0.482	1.380 (0.580-3.285)
Overweight	29/109 (26.6)	124/415 (29.9)	0.555	0.851 (0.530-1.367)
LBW	8/131 (6.1)	39/496 (7.9)	0.579	0.762 (0.347-1.673)
ADHD	3/137 (2.2)	15/495 (3.0)	0.776	0.716 (0.204-2.511)
DDA	24/130 (18.5)	87/506 (17.2)	0.796	1.090 (0.662-1.797)
Hay fever	16/63 (25.4)	60/205 (29.3)	0.633	0.823 (0.433-1.564)
Eczema	20/65 (30.8)	70/213 (32.9)	0.880	0.908 (0.499-1.653)
Excess cough	25/133 (18.8)	85/469 (18.1)	0.899	1.046 (0.638-1.715)
Stunting	4/119 (3.4)	19/442 (4.3)	0.798	0.774 (0.258-2.321)

OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg
M: F: Male: Female
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
High (↑) PM_{10} emissions: PM_{10} emissions >5 tonnes per annum
Low (↓) PM_{10} emissions: PM_{10} emissions $\leq$ 5 tonnes per annum
$PM1_0$: Particulate matter <10 microns
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to high versus those with exposure to low PM_{10} emissions. Increased odds for ever wheeze and breathlessness, and decreased odds for croup, were observed in the high PM_{10} emissions group.

4.3.04 High PM_{10} concentrations

Table 4.13 Associations between PM_{10} concentrations categories and birth and child health outcomes

Outcome	↑ PM_{10} C (%)	↓ PM_{10} C (%)	p value	Unadjusted OR (95% CI)
Obesity	**71/504 (14.0)**	**2/49 (4.1)**	**0.047**	**3.853 (0.916-16.218)**
LBW	*44/612 (7.2)*	*8/51 (15.7)*	*0.080*	*0.416 (0.184-0.940)*
Preterm	93/603 (15.4)	9/57 (15.8)	1.000	0.973 (0.461-2.050)
M: F ratio (%)	47.4 : 52.6	56.9 : 43.1	0.173	1.466 (0.853-2.519)[fb]
Eczema	87/280 (31.1)	8/16 (50.0)	0.166	0.451 (0.164-1.240)
Allergy	117/556 (21.0)	10/54 (18.5)	0.729	1.173 (0.573-2.400)
Breathlessness	44/587 (7.5)	1/52 (1.9)	0.164	4.133 (0.558-30.621)
DDA	114/618 (18.4)	7/55 (12.7)	0.361	1.551 (0.684-3.517)
ADHD	18/612 (2.9)	0/56 (0)	0.388	Cannot be calculated
Hay fever	73/269 (27.1)	6/15 (40.0)	0.373	0.559 (0.192-1.624)
CWB triad	24/327 (7.34)	3/28 (10.7)	0.460	0.660 (0.186-2.345)
Ever wheeze	115/587 (19.6)	8/52 (15.4)	0.583	1.340 (0.614-2.924)
IUGR	25/547 (4.6)	1/48 (2.1)	0.713	2.251 (0.298-16.984)
Overweight	149/504 (29.6)	13/49 (26.5)	0.744	1.162 (0.599-2.254)
Stunting	22/540 (4.1)	2/53 (3.8)	1.000	1.083 (0.248-4.796)
Croup	56/614 (9.1)	5/54 (9.3)	1.000	0.984 (0.376-2.569)
Excess cough	110/587 (18.7)	10/52 (19.2)	1.000	0.969 (0.471-1.990)
Bronchitis	50/618 (8.1)	4/55 (7.3)	1.000	1.122 (0.390-3.233)

OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
PM_{10}: Particulate matter <10 microns;
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m3
Low (↓) PM_{10} C: PM_{10} concentrations $\leq$ 13.91 microgram per m3
C: Concentration
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to high versus those with exposure to low PM_{10} concentrations. Increased odds for obesity and decreased odds for being born LBW were observed in the high PM_{10} concentrations group.

4.3.05 Combined high NOx and PM_{10} emissions

Table 4.14 Associations between combined NO_X - PM_{10} emissions categories and birth and child health outcomes

Outcome	↑ (NO_X + PM_{10}) E (%)	↓ (NO_X + PM_{10}) E (%)	p value	Unadjusted OR (95% CI)
Croup	**7/138 (5.1)**	**26/196 (13.3)**	**0.015**	**0.349 (0.147-0.830)**
Ever wheeze	**36/133 (27.1)**	**32/186 (17.2)**	**0.038**	**1.786 (1.041-3.064)**
Hay fever	*16/63 (25.4)*	*30/73 (41.1)*	*0.069*	*0.488 (0.234-1.017)*
M: F ratio (%)	*42.3:57.7*	*52.4 : 47.6*	*0.066*	*1.504 (0.978-2.309)*[fb]
Breathlessness	14/133 (10.5)	11/186 (5.9)	0.143	1.872 (0.822-4.264)
IUGR	3/128 (2.3)	10/170 (5.9)	0.163	0.384 (0.103-1.425)
Obesity	18/109 (16.5)	18/169 (10.7)	0.200	1.659 (0.821-3.352)
Bronchitis	14/138 (10.1)	14/202 (6.9)	0.319	1.516 (0.699-3.290)
Eczema	20/65 (30.8)	27/70 (36.9)	0.370	0.708 (0.347-1.445)
CWB triad	8/80 (10.0)	7/105 (6.7)	0.429	1.556 (0.539-4.485)
Allergy	24/124 (19.4)	39/173 (22.5)	0.566	0.825 (0.466-1.459)
Preterm birth	18/134 (13.4)	31/194 (16.0)	0.637	0.816 (0.436-1.528)
LBW	8/131 (6.1)	15/192 (7.8)	0.662	0.767 (0.316-1.866)
Overweight	29/109 (26.6)	49/169 (29.0)	0.684	0.888 (0.518-1.522)
ADHD	3/137 (2.2)	6/200 (3.0)	0.743	0.724 (0.178-2.945)
Excess cough	25/133 (18.8)	32/186 (17.2)	0.768	1.144 (0.625-1.986)
Stunting	4/119 (3.4)	8/178 (4.5)	0.768	0.739 (0.217-2.512)
DDA	24/130 (18.5)	36/203 (17.2)	0.885	1.050 (0.593-1.859)

OR: Odds Ratio
M: F: Male: Female ratio
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
High (↑) NOx-PM_{10} Emissions: NOx emissions >10 and PM_{10} emissions > 5 tonnes per annum
Low (↓) NOx-PM_{10} Emissions: NOx emissions $\leq$10 and PM_{10} emissions $\leq$5 tonnes per annum
PM10: Particulate matter <10 microns;
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_X ***and*** high PM_{10} emissions versus those with exposure to low NO_X ***and*** high PM_{10} emissions. Increased odds for ever wheeze, and decreased odds for croup and hay fever, were observed in the high joint emissions group. Females were more likely than males to be exposed to high levels of both emissions.

4.3.06 Combined high NO_2 and PM_{10} concentrations

Table 4.15 Associations between combined NO_2 - PM_{10} concentrations categories and birth and child health outcomes

Outcome	↑(NO_2 + PM_{10}) C (%)	↓ (NO_2 + PM_{10}) C (%)	P value	Unadjusted OR(95% CI)
Preterm birth	**93/601 (15.5)**	**7/18 (38.9)**	**0.016**	**0.288 (0.109-0.761)**
Obesity	71/503 (14.1)	0/13 (0)	0.232	Cannot be calculated
LBW	44/610 (7.2)	2/16 (12.5)	0.331	0.544 (0.120-2.471)
Eczema	86/279 (30.8)	2/8 (25.0)	1.000	1.337 (0.264-6.757)
Breathlessness	44/585 (7.5)	0/16 (0)	0.621	Cannot be calculated
M: F ratio (%)	47.5 : 52.5	44.4. : 55.6	0.816	0.883 (0.344-2.268)[fb]
Ever wheeze	115/585 (7.5)	4/16 (25.0)	0.535	0.734 (0.232-2.318)
DDA	114/616 (18.5)	2/17 (11.8)	0.751	1.703 (0.384-7.552)
Hay fever	73/268 (27.2)	2/8 (25.0)	1.000	1.123 (0.222-5.691)
ADHD	18/610 (3.0)	0/17 (0)	1.000	Cannot be calculated
CWB triad	24/326 (7.4)	1/9 (11.1)	0.507	0.636 (0.076—5.297)
Allergy	117/554 (21.1)	3/16 (18.8)	1.000	1.160 (0.325-4.139)
Overweight	149/503 (29.6)	3/13 (23.1)	0.764	1.403 (0.381-5.170)
IUGR	25/545 (4.6)	1/15 (6.7)	0.514	0.673 (0.085-5.324)
Bronchitis	50/616 (8.1)	3/17 (17.6)	0.163	0.412 (0.115-1.483)
Stunting	22/538 (4.1)	0/15 (0)	1.000	Cannot be calculated
Excess cough	110/585 (18.8)	4/16 (25.0)	0.520	0.695 (0.220-2.195)
Croup	56/612 (9.2)	1/16 6.3)	1.000	1.511 (0.196-11.652)

OR: Odds Ratio; M: F: Male: Female
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
C: Concentrations
High NO_2-PM_{10} concentration: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m3
Low NO_2-PM_{10} concentration: NO_2 concentration $\leq$ 17.01 & PM_{10} concentration $\leq$ 13.91 microgram per m3
PM_{10}: Particulate matter <10 microns
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_X ***and*** high PM_{10} concentrations versus those with exposure to low NO_X ***and*** high PM_{10} concentrations. Decreased odds for being born preterm were observed in the high joint NO_2 and PM10 concentrations group.

4.3.07 Combined high NO_X emissions and maternal smoking during pregnancy

Table 4.16 Associations between combined NO_X emissions – MSDP categories and birth and child health outcomes

Outcome	↑ NO_X E + MSDP	↓NO_X E+ NMSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	**29/104 (27.9)**	**20/137 (14.6)**	**0.015**	**2.262 (1.194-4.286)**
Obesity	**20/87 (23.0)**	**10/125 (8.0)**	**0.003**	**3.433 (1.517-7.768)**
M: F ratio (%)	**33.9 :66.1**	**56.8 : 43.2**	**<0.001**	**2.567 (1.545-4.251)[fb]**
Croup	*5/110 (4.5)*	*17/147 (11.6)*	*0.069*	*0.364 (0.130-1.020)*
Hay fever	*13/53 (24.5)*	*22/54 (40.7)*	*0.099*	*0.473 (0.206-1.082)*
Breathlessness	8/104 (7.7)	7/137 (5.1)	0.432	1.548 (0.543-4.414)
Bronchitis	12/109 (11.0)	10/151 (6.6)	0.260	1.744 (0.725-4.198)
IUGR	3/99 (3.0)	7/126 (5.6)	0.519	0.531 (0.134-2.109)
Preterm birth	13/105 (12.4)	23/144 (16.0)	0.470	0.743 (0.357-1.546)
CWB triad	6/59 (10.2)	6/80 (7.5)	0.761	1.396 (0.427-4.568)
Overweight	25/87 (28.7)	35/125 (28.0)	1.000	1.037 (0.565-1.902)
LBW	11/105 (10.5)	11/141 (7.8)	0.504	1.383 (0.575-3.324)
Allergy	20/105 (19.0)	28/129 (21.7)	0.630	0.849 (0.447-1.613)
ADHD	5/108 (4.6)	3/146 (2.1)	0.291	2.314 (0.541-9.900)
DDA	21/107 (19.6)	24/150 (16.0)	0.507	1.282 (0.672-2.447)
Eczema	15/53 (28.3)	21/54 (38.9)	0.307	0.620 (0.276-1.395)
Excess cough	14/104 (13.5)	22/137 (16.1)	0.716	0.813 (0.394-1.678)
Stunting	4/91 (4.4)	8/129 (6.2)	0.765	0.695 (0.203-2.383)

OR: Odds Ratio
Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
Preterm: Reported as preterm by mothers in questionnaire;
MSDP: Maternal smoking during pregnancy; NMSDP Mothers who did not smoke during pregnancy
E: Emissions
High NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
Low NOx Emissions + NMSDP: NOx emissions $\leq$ 10 (in tonnes per annum) and mothers who did not smoke during pregnancy

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NOx emissions ***and*** MSDP versus those with exposure to low NOX emissions and ***no*** MSDP. Increased odds for ever wheeze and obesity, and decreased odds for croup and hay fever were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.08 Combined high NOx emissions and paternal smoking during pregnancy

Table 4.17 Associations between combined NO_X emissions - PSDP categories and birth and child health outcomes

Outcome	↑ NO_X E + PSDP	↓ NO_X E + NPSDP	p value	Unadjusted OR (95% CI)
Eczema	**8/150 (16.0)**	**17/34 (50.0)**	**0.001**	**0.190 (0.069-0.524)**
Ever wheeze	**32/108 (29.6)**	**14/92 (15.2)**	**0.018**	**2.346 (1.161-4.738)**
Obesity	**14/95 (14.7)**	**5/91 (5.5)**	**0.050**	**2.973 (1.025-8.626)**
Croup	**6/113 (5.3)**	**14/104 (13.5)**	**0.050**	**0.360 (0.133-0.977)**
Bronchitis	*12/113 (10.6)*	*4/103 (3.9)*	*0.071*	*2.941 (0.917-9.428)*
Hay fever	*12/51 (23.5)*	*14/33 (42.4)*	*0.091*	*0.418 (0.162-1.076)*
M: F ratio (%)	47.1:52.9	52.3: 47.7	0.505	1.234 (0.732-2.083)[fb]
Breathlessness	10/108 (9.3)	3/92 (3.3)	0.148	3.027 (0.807-11.352)
IUGR	6/100 (6.0)	6/89 (6.7)	1.000	0.883 (0.274-2.843)
Preterm birth	21/110 (19.1)	13/102 (12.7)	0.262	1.615 (0.762-3.425)
CWB triad	5/58 (8.6)	1/58 (1.7)	0.206	5.377 (0.608-47.539)
Overweight	24/94 (25.5)	22/91 (24.2)	0.866	1.075 (0.552-2.096)
LBW	10/111 (9.0)	9/99 (9.1)	1.000	0.990 (0.385-2.546)
Allergy	18/109 (16.5)	20/90 (22.2)	0.366	0.692 (0.341-1.407)
ADHD	1/111 (0.9)	2/103 (1.9)	0.610	0.459 (0.041-5.140)
DDA	21/111 (18.9)	18/103 (17.5)	0.860	1.120 (0.549-2.210)
Excess cough	22/108 (20.4)	15/92 (16.3)	0.584	1.313 (0.636-2.711)
Stunting	2/97 (2.1)	2/90 (2.2)	1.000	0.926 (0.128-6.718)

OR: Odds Ratio
Upper quartile: Townsend score > 4; LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male : Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
Preterm: Reported as preterm by mothers in questionnaire
PSDP: Fathers who smoked during his partner's pregnancy period
NPSDP: Fathers who did not smoke during his partner's pregnancy period
E: Emissions; High NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
Low NOx emissions + NPSDP: NOx emissions < 10 (in tonnes per annum) and fathers who did not smoke during their partner's pregnancy period
fb :female birth

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NOx emissions ***and*** PSDP versus those with exposure to low NOX emissions and ***no*** PSDP. Increased odds for ever wheeze, obesity and bronchitis, and decreased odds for eczema, croup and hay fever were observed in the high joint exposure group.

4.3.09 Combined high NOx emissions and household member smoking during pregnancy

Table 4.18 Associations between combined NOx emissions - HSDP and birth and child health outcomes

Outcome	↑ NO_X E+ HSDP	↓NO_X E + NHSDP	p value	Unadjusted OR (95% CI)
M: F ratio (%)	**42.1:57.9**	**58.8 :41.2**	**0.006**	**1.961 (1.221-3.155)[fb]**
Croup	**8/178 (4.5)**	**17/107 (15.9)**	**0.002**	**0.249 (0.104-0.600)**
Obesity	**28/143 (19.6)**	**8/91 (8.8)**	**0.027**	**2.526 (1.096-5.822)**
Ever wheeze	*41/164 (25.0)*	*16/100 (16.0)*	*0.092*	*1.750 (0.922-3.322)*
Breathlessness	*15/164 (9.1)*	*3/100 (3.0)*	*0.076*	*3.235 (0.918-11.541)*
Hay fever	***20/81 (24.7)***	***17/33 (51.5)***	***0.008***	***0.309 (0.132-0.721)***
Eczema	*23/87 (26.4)*	*12/27 (44.4)*	*0.096*	*0.449 (0.183-1.101)*
Bronchitis	18/175 (10.3)	8/110 (7.3)	0.527	1.462 (0.613-3.487)
IUGR	6/156 (3.8)	5/92 (5.4)	0.542	0.696 (0.206-2.340)
Preterm birth	30/174 (17.2)	17/107 (15.9)	0.870	1.103 (0.575-2.114)
CWB triad	7/92 (7.6)	2/54 (3.7)	0.485	2.141 (0.428-10.700)
Overweight	49/142 (34.5)	28/91 (30.8)	0.572	1.185 (0.675-2.083)
LBW	16/170 (9.4)	9/104 (8.7)	1.000	1.097 (0.466-2.581)
Allergy	35/163 (21.5)	20/98 (20.4)	0.877	1.066 (0.575-1.977)
ADHD	7/176 (4.0)	1/108 (0.9)	0.162	4.432 (0.538-36.528)
DDA	30/173 (17.3)	15/110 (13.6)	0.505	1.329 (0.679-2.601)
Excess cough	35/164 (21.3)	18/100 (18.0)	0.532	1.236 (0.657-2.326)
Stunting	7/151 (4.6)	5/97 (5.2)	1.000	0.894 (0.276-2.902)

OR: Odds Ratio; Upper quartile: (Townsend score > 4)
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile) ; Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female; CWB triad: Symptom triad of cough, wheeze and breathlessness
Preterm: Reported as preterm by mothers in questionnaire
HSDP: Household members who smoked during pregnancy period
NHSDP: Household members who did not smoke during pregnancy period
E: Emissions; High NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
Low NOx emissions + NHSDP: NOx emissions < 10 (in tonnes per annum) and household member who did not smoke during their partner's pregnancy period; fb :female birth

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NOx emissions ***and*** HSDP versus those with exposure to low NOx emissions and ***no*** HSDP. Increased odds for obesity, ever wheeze and breathlessness, and decreased odds for croup, hay fever and eczema were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.10 Combined high PM_{10} emissions and maternal smoking during pregnancy

Table 4.19 Associations between combined PM_{10} emissions - MSDP categories and birth and child health outcomes

Outcome	↑ PM_{10} E+ MSDP	↓PM_{10} E + NMSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	**14/44 (31.8)**	**53/348 (15.2)**	**0.010**	**2.597 (1.292-5.233)**
Obesity	**9/37 (24.3)**	**32/309 (10.4)**	**0.026**	**2.782 (1.207-6.416)**
M: F ratio (%)	**38.3 : 61.7**	**54.6 : 45.4**	**0.044**	**1.942 (1.043-3.610)[fb]**
Bronchitis	*7/46 (15.2)*	*24/370 (6.5)*	*0.065*	*2.588 (1.047-6.394)*
Croup	1/46 (2.2)	37/368 (10.1)	0.103	0.199 (0.027-1.484)
Breathlessness	3/44 (6.8)	19/348 (5.5)	0.725	1.267 (0.359-4.468)
IUGR	1/43 (2.3)	16/320 (5.0)	0.705	0.452 (0.058-3.500)
Preterm birth	4/44 (9.1)	66/366 (18.0)	0.201	0.455 (0.157-1.314)
CWB	3/25 (10.7)	15/190 (7.9)	0.710	1.400 (0.378-5.181)
Overweight	13/37 (35.1)	94/309 (30.4)	0.575	1.239 (0.605-2.538)
LBW	3/41 (7.3)	26/363 (7.2)	1.000	1.023 (0.296-3.541)
Allergy	8/43 (18.6)	74/335 (22.1)	0.697	0.806 (0.359-1.813)
Hay fever	5/19 (26.3)	44/145 (30.3)	0.797	0.820 (0.278-2.416)
ADHD	1/46 (2.2)	8/370 (2.2)	1.000	1.006 (0.123-8.226)
DDA	6/42 (14.3)	59/372 (15.9)	1.000	0.884 (0.357-2.192)
Eczema	6/19 (31.0)	53/153 (34.6)	1.000	0.871 (0.313-2.422)
Excess cough	6/44 (13.6)	67/348 (19.3)	0.419	0.662 (0.269-1.631)
Stunting	3/39 (7.7)	18/323 (5.6)	0.484	1.412 (0.396-5.029)

OR: Odds Ratio; LBW: Low birthweight: Birthweight <2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions; PM_{10}: Particulate matter <10 microns
High PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
Low PM_{10} emissions + NMSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and mothers who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} emissions ***and*** MSDP versus those with exposure to low PM_{10} emissions and ***no*** MSDP. Increased odds for obesity, ever wheeze and bronchitis were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.11 Combined high PM_{10} emissions and paternal smoking during pregnancy

Table 4.20 Associations between combined PM_{10} emissions - PSDP categories and birth and child health outcomes

Outcome	↑PM_{10} E + PSDP	↓PM_{10} E + NPSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	**14/46 (30.4)**	**37/244 (15.2)**	**0.019**	**2.448 (1.193-5.023)**
Bronchitis	**7/48 (14.6)**	**13/268 (4.9)**	**0.020**	**3.349 (1.262-8.890)**
Obesity	7/45 (15.6)	24/224 (10.7)	0.441	1.535 (0.618-3.816)
M: F ratio (%)	55.1:44.9	52.0 : 48.0	0.757	0.881 (0.479-1.623)[fb]
Croup	*1/48 (2.1)*	*30/270 (11.1)*	*0.062*	*0.170 (0.023-1.279)*
Breathlessness	4/46 (8.7)	10/224 (4.1)	0.249	2.229 (0.668-7.437)
IUGR	1/44 (2.3)	9/232 (3.9)	1.000	0.576 (0.071-4.666)
Preterm birth	8/46 (17.4)	41/267 (15.4)	0.667	1.160 (0.505-2.666)
CWB triad	2/29 (6.9)	7/144 (4.9)	0.648	1.450 (0.286-7.361)
Overweight	14/45 (31.1)	67/225(29.8)	0.860	1.065 (0.533-2.129)
LBW	4/45 (8.9)	22/263 (8.4)	1.000	1.069 (0.350-3.261)
Allergy	7/45 (15.6)	56/248 (22.6)	0.331	0.632 (0.267-1.492)
Hay fever	4/20 (20.0)	33/111 (29.7)	0.433	0.591 (0.184-1.902)
ADHD	0/48 (0)	9/267 (3.4)	0.364	1.035 (1.012-1.058)
DDA	7/44 (15.9)	44/268 (16.4)	1.000	0.963 (0.403-2.299)
Eczema	4/21 (19.0)	44/118 (37.3)	0.137	0.396 (0.125-1.251)
Excess cough	10/46 (21.7)	48/244 (19.7)	0.841	1.134 (0.526-2.446)
Stunting	0/41 (0)	10/234 (4.3)	0.367	Cannot be calculated

OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
PM_{10}: Particulate matter <10 microns;
High PM_{10} emissions + PSDP: PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
Low PM_{10} Emissions + NPSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy period
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} emissions ***and*** PSDP versus those with exposure to low PM_{10} emissions and ***no*** PSDP. Increased odds for ever wheeze and bronchitis, and decreased odds of croup, were observed in the high joint exposure group.

4.3.12 Combined high PM_{10} emissions and household smoking during pregnancy

Table 4.21 Associations between combined PM_{10} emissions - HSDP categories and birth and child health outcomes

Outcome	↑ PM_{10} E + HSDP	↓PM_{10} E + NHSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	**19/64 (29.7)**	**41/268 (15.3)**	**0.011**	**2.338 (1.244-4.394)**
Obesity	**12/52 (23.1)**	**24/231 (10.4)**	**0.020**	**2.588 (1.197-5.595)**
Croup	**2/67(3.0)**	**35/283 (12.4)**	**0.025**	**0.218 (0.051-0.930)**
Breathlessness	*7/64 (10.9)*	*12/268 (4.5)*	*0.067*	*2.620 (0.988-6.948)*
Bronchitis	*9/67 (13.4)*	*17/286 (5.9)*	*0.064*	*2.455 (1.043-5.781)*
M: F ratio (%)	45.6 :54.4	57.0 :43.0	0.105	1.585 (0.934-2.688)[fb]
IUGR	1/60 (1.7)	11/243 (4.5)	0.471	0.357 (0.045-2.824)
Preterm birth	6/65 (9.2)	42/283 (14.8)	0.319	0.584 (0.237-1.437)
CWB triad	3/39 (7.7)	9/141 (6.4)	0.724	1.222 (0.314-4.751)
Overweight	20/52 (38.5)	69/232 (29.7)	0.248	1.476 (0.790-2.760)
LBW	5/61 (8.2)	20/282 (7.1)	0.786	1.170 (0.421-3.249)
Allergy	14/59 (23.7)	57/263 (21.7)	0.730	1.124 (0.577-2.192)
Hay fever	7/29 (24.1)	33/105 (31.4)	0.501	0.640 (0.262-1.563)
ADHD	1/67 (1.5)	4/280 (1.4)	1.000	1.045 (0.115-9.508)
DDA	12/62 (19.4)	47/288 (16.3)	0.576	1.231 (0.609-2.486)
Eczema	8/32 (25.0)	37/108 (34.3)	0.392	0.640 (0.262-1.563)
Excess cough	14/64 (21.9)	47/268 (17.5)	0.472	1.317 (0.673-2.576)
Stunting	3/57 (5.3)	11/250 (4.4)	0.729	1.207 (0.326-4.475)

OR: Odds Ratio
LBW: Low birthweight: Birthweight<2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile) ; Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
PM_{10}: Particulate matter <10 microns;
High PM_{10} emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum)
and household member smoking during pregnancy
Low PM_{10} emissions + NHSDP: PM_{10} emissions < 5 (in tonnes per annum)
and household member who did not smoke during pregnancy period
Preterm: Reported as preterm by mothers in questionnaire ; fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} emissions ***and*** HSDP versus those with exposure to low PM_{10} emissions and ***no*** HSDP. Increased odds for ever wheeze, obesity, breathlessness and bronchitis, and decreased odds of croup, were observed in the high joint exposure group.

4.3.13 Combined high NO_2 concentrations and maternal smoking during pregnancy

Table 4.22 Associations between combined NO_2 concentrations - MSDP categories and birth and child health outcomes

Outcome	↑ NO_2 C + MSDP	↓NO_2 C + NMSDP	p value	Unadjusted OR (95% CI)
Preterm birth	**19/158 (12.0)**	**6/16 (37.5)**	**0.014**	**0.228 (0.074-0.698)**
Ever wheeze	41/156 (26.3)	3/14 (21.4)	1.000	1.307 (0.347-4.920)
Obesity	28/135 (20.7)	0/12 (0)	0.123	Cannot be calculated
M: F ratio (%)	36.1 : 63.9	37.5 : 62.5	1.000	1.062 (0.368-3.067)[fb]
Croup	14/162 (8.6)	1/15 (6.7)	1.000	1.324 (0.162-10.831)
Breathlessness	13/156 (8.3)	0/14 (0)	0.604	Cannot be calculated
Bronchitis	16/162 (9.9)	2/15 (13.3)	0.653	0.712 (0.147-3.443)
IUGR	6/148 (4.1)	1/13 (7.7)	0.452	0.507 (0.056-4.565)
CWB triad	7/86 (8.1)	1/8 (12.5)	0.523	0.620 (0.066-5.787)
Overweight	41/135 (30.4)	3/12 (25.0)	1.000	1.309 (0.337-5.089)
LBW	17/159 (10.7)	2/15 (13.3)	0.670	0.778 (0.162-3.746)
Allergy	28/150 (18.7)	2/15 (13.3)	1.000	1.492 (0.318-6.989)
Hay fever	19/70 (27.1)	2/7 (28.6)	1.000	0.931 (0.166-5.214)
ADHD	8/164 (4.9)	0/15 (0)	1.000	Cannot be calculated
DDA	34/164 (20.7)	1/15 (6.7)	0.309	3.662 (0.465-28.851)
Eczema	24/73 (32.9)	3/7 (42.9)	0.683	0.653 (0.135-3.153
Excess cough	24/156 (15.4)	4/14 (28.6)	0.252	0.455 (0.132-1.568)
Stunting	4/141 (2.8)	0/13 (0)	1.000	Cannot be calculated

OR: Odds Ratio
MSDP: Mothers who smoked during his partner's pregnancy period
NMSDP: Mothers who did not smoke during his partner's pregnancy period
LBW: Low birthweight: Birthweight < 2.5 kgs
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M:F : Male : Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: concentration
Combined high NO_2 concentration and MSDP: NO_2 concentration >17.01 microgram per m3 and maternal smoking during pregnancy
Combined low NO_2 concentration and NMSDP: NO_2 concentration $\leq$ 17.01 microgram per m3 and mothers who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb : for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_2 concentrations ***and*** MSDP versus those with exposure to low NO_2 concentrations and ***no*** MSDP. Decreased odds for being born preterm were observed in the high joint exposure group.

4.3.14 Combined high NO_2 concentrations and paternal smoking during pregnancy

Table 4.23 Associations between combined NO_2 concentrations - PSDP categories and birth and child health outcomes

Outcome	↑ NO_X C + PSDP	↓NO_X C + NPSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	40/175 (22.9)	3/11 (27.3)	0.718	0.790 (0.200-3.119)
Obesity	22/155 (14.2)	0/9 (0)	0.610	Cannot be calculated
M:F ratio (%)	48.2 :51.8	27.3 :72.7	0.223	0.404 (0.103-1.567)[fb]
Croup	16/184 (8.7)	1/10 (10)	1.000	0.857 (0.102-7.203)
Breathlessness	13/175 (7.4)	0/11 (0)	1.000	Cannot be calculated
Bronchitis	19/185 (10.3)	2/11 (18.2)	0.334	0.515 (0.104-2.561)
IUGR	11/162 (6.8)	1/9 (11.1)	0.489	0.583 (0.067-5.089)
Preterm birth	*27/177(15.3)*	*4/11 (36.4)*	*0.086*	*0.315 (0.086-1.150)*
CWB triad	10/91 (11.0)	1/6 (16.7)	0.524	0.617 (0.065-5.829)
Overweight	43/154 (27.9)	1/9 (11.1)	0.446	3.099 (0.376-25.523)
LBW	13/178 (7.3)	2/10 (20.0)	0.184	0.315 (0.086-1.150)
Allergy	33/172 (19.2)	2/10 (20.0)	1.000	0.950 (0.193-4.682)
Hay fever	23/81 (28.4)	2/4 (50.0)	0.577	0.397 (0.053-2.985
ADHD	2/180 (1.1)	0/11 (0)	1.000	Cannot be calculated
DDA	32/182 (17.6)	1/10 (10.0)	1.000	1.920 (0.235-15.694)
Eczema	16/75 (21.3)	2/4 (50.0)	0.222	0.271 (0.035-2.078)
Excess cough	34/175 (19.4)	4/11(36.4)	0.239	0.422 (0.117-1.524)
Stunting	4/155 (2.6)	0/8 (0)	1.000	Cannot be calculated

OR: Odds Ratio
PSDP: Father who smoked during his partner's pregnancy period
NPSDP: Fathers who did not smoke during his partner's pregnancy period
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: concentration
Combined high NO_2 concentration and PSDP: NO_2 concentration >17.01 microgram per m3 and paternal smoking during pregnancy
Combined low NO_2 concentration and NPSDP: NO_2 concentration $\leq$ 17.01 microgram per m3 and fathers who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb: for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_2 concentrations ***and*** PSDP versus those with exposure to low NO_2 concentrations and ***no*** PSDP. Decreased odds for being born preterm were observed in the high joint exposure group.

4.3.15 Combined high NO_2 concentrations and household smoking during pregnancy

Table 4.24 Associations between combined NO_2 concentrations - HSDP categories and birth and child health outcomes

Outcome	↑ NO_2 C+ HSDP	↓NO_2 C + NHSDP	p value	Unadjusted OR 95% CI
Preterm birth	*40/274 (14.6)*	*5/15 (33.3)*	*0.065*	*0.342 (0.111-1.053)*
Ever wheeze	60/263 (22.8)	3/13 (23.1)	1.000	0.985 (0.263-3.695)
Obesity	39/231 (16.9)	0/12 (0)	0.223	Cannot be calculated
M: F ratio (%)	42.3 : 57.7	40.0 : 60.0	1.000	0.911 (0.316-2.625)[fb]
Croup	19/282 (6.7)	1/14 (7.1)	1.000	0.939 (0.117-7.567)
Breathlessness	24/263 (9.1)	0/13 (0)	0.613	0.909 (0.875-0.944)
Bronchitis	26/279 (9.3)	2/14 (14.3)	0.631	0.617 (0.131-2.906)
IUGR	11/248 (4.4)	1/12 (8.3)	0.440	0.511 (0.060-4.316)
CWB triad	12/152(7.9)	1/7 (14.3)	0.456	0.514 (0.057-4.650)
Overweight	73/230 (31.7)	3/12 (25)	0.758	1.395 (0.367-5.305)
LBW	26/269 (9.7)	2/14 (14.3)	0.637	0.642 (0.136-3.027)
Allergy	51/251 (20.3)	1/14 (7.1)	0.316	3.315 (0.424-25.933)
Hay fever	32/128 (25.0)	1/6 (16.7)	1.000	1.667 (0.188-14.803)
ADHD	11/281 (3.9)	0/14 (0)	1.000	Cannot be calculated
DDA	55/279 (19.7)	1/14 (7.1)	0.483	3.192 (0.409-24.925)
Eczema	39/137 (28.5)	2/6 (33.3)	1.000	0.796 (0.140-4.523)
Excess cough	54/263 (20.5)	3/13 (23.1)	0.735	0.861(0.229-3.238)
Stunting	11/242 (4.5)	0/12 (0)	1.000	Cannot be calculated

OR: Odds Ratio
LBW: Low birthweight: Birthweight<2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
IUGR: Intrauterine growth restriction
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: Concentration
Combined high NO_2 concentration and HSDP: NO_2 concentration >17.01 microgram per m3 and household member smoking during pregnancy
Combined low NO_2 concentration and NHSDP: NO_2 concentration $\leq$ 17.01 microgram per m3 and household members who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_2 concentrations ***and*** HSDP versus those with exposure to low NO_2 concentrations and ***no*** HSDP. Decreased odds for being born preterm were observed in the high joint exposure group.

4.3.16 Combined high PM_{10} concentrations and maternal smoking during pregnancy

Table 4.25 Associations between combined PM_{10} concentrations - MSDP categories and birth and child health outcomes

Outcome	↑ PM_{10} C + MSDP	↓PM_{10} C + NMSDP	p value	Unadjusted OR (95% CI)
Obesity	**27/130 (20.8)**	**1/41 (2.4)**	**0.003**	**10.485 (1.578-79.762)**
M: F ratio (%)	**35.4 : 64.6**	**56.3 : 43.8**	**0.012**	**2.347 (1.222-4.525)[fb]**
Ever wheeze	41/151(27.2)	7/42 (16.7)	0.226	1.864 (0.767-4.526)
Croup	14/158 (8.9)	5/46 (10.9)	0.773	0.797 (0.271-2.344)
Breathlessness	13/151 (8.6)	1/42 (2.4)	0.310	3.862 (0.490-30.413)
Bronchitis	16/157 (10.2)	3/45 (6.7)	0.576	1.589 (0.442-5.716)
IUGR	6/144 (4.2)	1/40 (2.5)	1.000	1.696 (0.198-14.508)
Preterm birth	19/154 (12.3)	8/48 (16.7)	0.469	0.704 (0.287-1.728)
CWB triad	7/84 (8.3)	3/23 (13.0)	0.445	0.606 (0.144-2.556)
Overweight	38/130 (27.2)	9/41 (22.0)	0.426	1.469 (0.640-3.370)
LBW	16/154 (10.4)	7/42 (16.7)	0.282	0.580 (0.721-1.518)
Allergy	27/146 (18.5)	8/46 (17.4)	1.000	1.078 (0.452-2.571)
Hay fever	18/69 (26.1)	5/12 (41.7)	0.307	0.494 (0.139-1.755)
ADHD	8/159 (5.0)	0/46 (0)	0.203	Cannot be calculated
DDA	34/159 (21.4)	6/45 (13.3)	0.290	1.768 (0.691-4.523)
Eczema	23/72 (31.9)	7/13 (53.8)	0.205	0.402 (0.121-1.333)
Excess cough	24/151 (15.9)	10/42 (23.8)	0.255	0.605 (0.263-1.392)
Single parent	52/146 (35.6)	20/43 (46.5)	0.214	0.636 (0.310-1.266)
Stunting	4/136 (2.9)	2/43 (4.7)	0.631	0.621 (0.110-3.515)

OR: Odds ratio
LBW: Low birthweight: Birthweight <2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness; C: Concentration
Combined high PM_{10} concentration and MSDP: PM_{10} concentration >13.91 microgram per m3 and maternal smoking during pregnancy
Combined low PM_{10} concentration and NMSDP: PM_{10} concentration $\leq$ 13.91 microgram per m3 and mothers who did not smoke during pregnancy
PM10: Particulate matter <10 microns; Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} concentrations ***and*** MSDP versus those with exposure to low PM_{10} concentrations and ***no*** MSDP. Increased odds for obesity were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.17 Combined high PM_{10} concentrations and paternal smoking during pregnancy

Table 4.26 Association between combined PM_{10} concentrations - PSDP categories and birth and child health outcomes

Outcome	↑ PM_{10} C + PSDP	↓PM_{10} C + NPSDP	p value	Unadjusted OR (95% CI)
LBW	**11/170 (6.5)**	**6/31 (19.4)**	**0.029**	**0.288 (0.098-0.849)**
Obesity	**21/148 (14.2)**	**0/32 (0)**	**0.028**	**Cannot be calculated**
Hay fever	*23/81 (28.4)*	*6/11 (54.5)*	*0.094*	*0.330 (0.092-1.190)*
Ever wheeze	40/157 (24.0)	4/32 (12.5)	0.172	2.205 (0.729-6.665)
M: F ratio (%)	48.1: 51.9	55.6: 44.4	0.468	1.350 (0.658-2.770)[fb]
Croup	16/176 (9.1)	3/35 (8.6)	1.000	1.067 (0.294-3.876)
Breathlessness	13/167 (9.1)	0/32 (0)	0.134	Cannot be calculated
Bronchitis	19/177 (10.7)	2/35 (5.7)	0.539	1.984 (0.441-8.933)
IUGR	11/155 (7.1)	1/30 (3.3)	0.694	2.215 (0.275-17.832)
Preterm birth	27/169 (16.0)	6/36 (16.7)	1.000	0.951 (0.361-2.504)
CWB triad	9/88 (10.2)	2/19 (10.5)	1.000	0.968 (0.192-4.890)
Overweight	40/147 (27.2)	6/32 (18.8)	0.379	1.620 (0.621-4.227)
Allergy	33/164 (20.1)	8/34 (23.5)	0.646	0.879 (0.340-1.973)
ADHD	2/172 (1.2)	0/35 (0)	1.000	Cannot be calculated
DDA	32/174 (18.4)	4/33 (12.1)	0.462	1.634 (0.537-4.975)
Eczema	16/75 (21.3)	7/11 (63.6)	0.007	0.155 (0.040-0.596)
Excess cough	33/167 (19.8)	6/32 (18.8)	1.000	1.067 (0.406-2.804)
Stunting	3/148 (2.0)	0/32 (0)	1.000	Cannot be calculated

OR: Odds Ratio
LBW: Low birthweight: Birthweight<2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
IUGR: Intrauterine growth restriction
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: Concentration
Combined high PM_{10} Concentration and PSDP: PM_{10} concentration >13.91 microgram per m3 and paternal smoking during pregnancy
Combined low PM_{10} Concentration and NPSDP: PM_{10} Concentration $\leq$ 13.91 microgram per m3 and fathers who did not smoke during pregnancy
PM_{10}: Particulate matter <10 microns; Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} concentrations ***and*** PSDP versus those with exposure to low PM_{10} concentrations and ***no*** PSDP. Increased odds for obesity and decreased odds for hay fever and for being born LBW were observed in the high joint exposure group.

4.3.18 Combined high PM_{10} concentrations and household smoking during pregnancy

Table 4.27 Associations between combined PM_{10} concentrations - HSDP categories and birth and child health outcomes

Outcome	↑ PM_{10} C+ HSDP	↓PM_{10} C + NHSDP	p value	Unadjusted OR (95% CI)
Obesity	**37/221 (16.7)**	**0/34 (0)**	**0.007**	**Cannot be calculated**
M: F ratio (%)	**41.8 :58.2**	**61.5: 38.5**	**0.025**	**2.227 (1.121-4.425)[fb]**
Ever wheeze	60/253 (23.7)	7/35 (20.0)	0.831	1.244 (0.517-2.990)
Croup	19/272 (7.0)	5/37 (13.5)	0.185	0.481 (0.168-1.376)
Breathlessness	24/253 (9.5)	1/35 (2.9)	0.333	3.563 (0.467-27.201)
Bronchitis	26/269 (9.7)	3/36 (8.3)	1.000	1.182 (0.339-4.122)
IUGR	11/239 (4.6)	1/32 (3.1)	1.000	1.496 (0.187-11.986)
Preterm birth	40/264 (15.2)	6/39 (15.4)	1.000	0.982 (0.386-2.496)
CWB triad	11/146(7.5)	1/17 (5.9)	1.000	1.304 (0.158-10.771)
Overweight	69/220 (31.4)	8/34 (23.5)	0.426	1.455 (0.640-3.447)
LBW	23/259(8.9)	5/34 (14.7)	0.346	0.565(0.200-1.601)
Allergy	49/241 (20.3)	5/37(13.5)	0.382	1.633(0.605-4.410)
Hay fever	31/127 (24.4)	4/10 (40.0)	0.277	0.489 (0.128-1.828)
ADHD	11/271 (4.1)	0/38 (0)	0.371	Cannot be calculated
DDA	54/268 (20.1)	5/38 (13.2)	0.384	1.665 (0.621-4.468)
Eczema	37/135 (27.4)	4/10 (40.0)	0.469	0.566 (0.151-2.121)
Excess cough	53/253 (20.9)	8/35 (22.9)	0.826	0.894 (0.384-2.082)
Stunting	10/232 (4.3)	1/35 (2.9)	1.000	1.532 (0.190-12.346)

OR: Odds Ratio
LBW: Low birthweight: Birthweight <2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile) ; Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
IUGR: Intrauterine growth restriction
CWB triad: Symptom triad of cough, wheeze and breathlessness; C: Concentration
Combined high PM_{10} concentration and HSDP: PM_{10} concentration >13.91 microgram per m3 and household member smoking during pregnancy
Combined low PM_{10} concentration and NHSDP: PM_{10} concentration $\leq$ 13.91 microgram per m3 and household member who did not smoke during pregnancy
PM_{10}: Particulate matter <10 microns
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} concentrations ***and*** HSDP versus those with exposure to low PM_{10} concentrations and ***no*** HSDP. Increased odds for obesity were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.19 Combined high NO_X and PM_{10} emissions and maternal smoking during pregnancy

Table 4.28 Associations between combined NO_X + PM_{10} emissions - MSDP and birth and child health outcomes

Outcome	↑ (NO_X + PM_{10}) E + MSDP	↓(NO_X + PM_{10}) E + NMSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	**14/44 (31.8)**	**20/137 (14.6)**	**0.015**	**2.730 (1.236-6.028)**
Obesity	**9/37 (24.3)**	**10/125 (8.0)**	**0.016**	**3.696 (1.372-9.955)**
M:F ratio (%)	**38.3:61.7**	**56.8:43.2**	**0.031**	**2.144 (1.085-4.132)[fb]**
Bronchitis	*7/46 (15.2)*	*10/151 (6.6)*	*0.078*	*2.531 (0.904-7.081)*
Croup	1/46 (2.2)	17/147 (11.6)	0.078	0.170 (0.022-1.314)
Breathlessness	3/44 (6.8)	7/137 (5.1)	0.707	1.359 (0.336-5.946)
IUGR	1/43 (2.3)	7/126 (5.6)	0.681	0.405 (0.048-3.388)
Preterm birth	4/44 (9.1)	23/144 (16.0)	0.330	0.526 (0.172-1.613)
CWB	3/28 (10.7)	6/80 (7.5)	0.693	1.480 (0.344-6.361)
Overweight	13/37 (35.1)	35/125 (28.0)	0.418	1.393 (0.639-3.038)
LBW	3/41 (7.3)	11/141 (7.8)	1.000	0.933 (0.248-3.516)
Allergy	8/43 (18.6)	28/129 (21.7)	0.829	0.824 (0.344-1.977)
Hay fever	5/19 (26.3)	22/54 (40.7)	0.408	0.519 (0.163-1.651)
ADHD	1/46 (2.2)	3/146 (2.1)	1.000	1.059 (0.108-10.437)
DDA	6/42 (14.3)	24/150 (16.0)	1.000	0.875 (0.332-2.304)
Eczema	6/19 (31.6)	21/54 (38.9)	0.783	0.725 (0.239-2.204)
Excess cough	6/44 (13.6)	22/137 (16.1)	0.814	0.825 (0.312-2.187)
Stunting	3/39 (7.7)	8/129 (6.2)	0.719	1.260 (0.318-5.000)

OR: Odds Ratio; LBW: Low birthweight: Birthweight <2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female; CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions; NO_X: Oxides of Nitrogen; PM_{10}: Particulate matter <10 microns
MSDP: Maternal smoking during pregnancy; NMSDP: Mothers who did not smoke during pregnancy
Combined high NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
Combined low NOx-PM_{10} emissions + NMSDP: NOx emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and mothers who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire; fb: for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x+ high PM_{10} emissions ***and*** MSDP vs. those with exposure to low NO_x + low PM_{10} emissions and ***no*** MSDP. Increased odds for wheeze, obesity and bronchitis were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.20 Combined high NO_X and PM_{10} concentrations and maternal smoking during pregnancy

Table 4.29 Associations between combined NO_X + PM_{10} concentrations - MSDP categories and birth / child health outcomes

Outcome	↑ (NO_2 + PM_{10}) C + MSDP	↓ (NO_X + PM_{10}) C + NMSDP	p value	Unadjusted OR (95% CI)
Preterm birth	**19/154 (12.3)**	**6/14 (42.9)**	**0.008**	**0.188 (0.059-0.600)**
Obesity	27/130 (20.8)	0/11 (0)	0.124	Cannot be calculated
M: F ratio (%)	35.4 : 64.6	42.9 : 57.1	0.574	1.370 (0.454-4.149)[fb]
LBW	16/154 (10.4)	2/13 (15.4)	0.635	0.638 (0.130-3.137)
Eczema	23/72 (31.9)	2/6 (33.3)	1.000	0.939 (0.160-5.501)
breathlessness	13/151 (8.6)	0/12 (0)	0.601	Cannot be calculated
DDA	34/159 (21.4)	1/13 (7.7)	0.471	3.264 (0.410-25.994)
Hay fever	18/69 (26.1)	2/6 (33.3)	0.654	0.706 (0.119-4.187)
ADHD	8/159 (5.0)	0/13(0)	1.000	Cannot be calculated
CWB triad	7/84 (8.3)	1/7 (14.3)	0.487	0.545 (0.057-5.196)
Allergy	27/146 (18.5)	2/13 (15.4)	1.000	1.248 (0.261-5.959)
Overweight	38/130 (29.2)	3/11 (27.3)	1.000	1.101 (0.277-4.377)
IUGR	6/144 (4.2)	1/11 (9.1)	0.409	0.435 (0.048-3.972)
Bronchitis	16/157 (10.2)	2/13 (15.4)	0.632	0.624 (0.127-3.069)
Stunting	4/136 (2.9)	0/11 (0)	1.000	Cannot be calculated
Excess cough	24/151 (15.9)	4/12 (33.3)	0.128	0.378 (0.105-1.355)
Croup	14/158 (8.9)	1/13 (7.7)	1.000	1.167 (0.141-9.647)
Ever wheeze	41/151 (27.2)	3/12 (25.0)	1.000	1.118 (0.288-4.335)

OR: Odds Ratio
LBW: Low birthweight: Birthweight <2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness; C: Concentration
Combined high NO_2-PM_{10} concentration and MSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m3 and maternal smoking during pregnancy
Combined low NO_2-PM_{10} concentration and NMSDP: NO_2 concentration $\leq$ 17.01 & PM_{10} concentration $\leq$ 13.91 microgram per m3 and mothers who did not smoke during pregnancy
PM10: Particulate matter <10 microns
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x + high PM_{10} concentrations ***and*** MSDP vs. those with exposure to low NO_2 + low PM_{10} concentrations and ***no*** MSDP. Decreased odds for being born preterm were observed in the high joint exposure group.

4.3.21 Combined high NO_X and PM_{10} emissions and paternal smoking during pregnancy

Table 4.30 Associations between combined NO_X + PM_{10} emissions - PSDP and birth and child health outcomes

Outcome	↑(NO_X + PM_{10}) E + PSDP	↓ (NO_X + PM_{10}) E + NPSDP	p value	Unadjusted OR (95% CI)
Ever wheeze	**14/46 (30.4)**	**14/92 (15.2)**	**0.045**	**2.438 (1.045-5.688)**
Eczema	**4/21 (19.0)**	**17/34 (50.0)**	**0.026**	**0.235 (0.065-0.846)**
Croup	**1/48 (2.1)**	**14/104 (13.5)**	**0.038**	**0.137 (0.017-1.072)**
Bronchitis	**7/48 (14.6)**	**4/103 (3.9)**	**0.037**	**4.226 (1.173-15.217)**
Obesity	*7/45 (15.6)*	*5/91 (5.5)*	*0.062*	*3.168 (0.945-10.620)*
M: F (%)	55.1:44.9	52.3:47.7	0.863	0.894 (0.454-1.764)[fb]
Breathlessness	4/46 (8.7)	3/92 (3.3)	0.222	2825 (0.605-13.196)
IUGR	1/44 (2.3)	6/89 (6.7)	0.424	0.322 (0.038-2.758)
Preterm birth	8/46 (17.4)	13/102 (12.7)	0.455	1.441 (0.552-3.761)
CWB triad	2/29 (6.9)	1/58 (1.7)	0.257	4.222 (0.367-48.623)
Overweight	14/45 (31.1)	22/91 (24.2)	0.414	1.416 (0.641-3.130)
LBW	4/45 (8.9)	9/99 (9.1)	1.000	0.976 (0.284-3.352)
Allergy	7/45 (15.6)	20/90 (22.2)	0.494	0.645 (0.250-1.662)
Hay fever	4/20 (20.0)	14/33 (42.4)	0.137	0.339 (0.093-1.239)
ADHD	0/48 (0)	2/103 (1.9)	1.000	Cannot be calculated
DDA	7/44 (15.9)	18/103 (17.5)	1.000	0.893 (0.344-2.321)
Excess cough	10/46 (21.7)	15/92 (16.3)	0.485	1.426 (0.584-3.482)
Stunting	0/41 (0)	2/90 (2.2)	1.000	Cannot be calculated

OR: Odds Ratio
PSDP: Paternal smoking during pregnancy; NPSDP: Fathers who did not smoke during pregnancy
LBW: Low birthweight: Birthweight<2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female; CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions; NO_X: Oxides of Nitrogen; PM_{10}: Particulate matter <10 microns
Combined high NOx-PM_{10} Emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
Combined low NOx-PM_{10} Emissions + NPSDP: NOx emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire ; fb: for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x + high PM_{10} emissions ***and*** PSDP versus those with exposure to low NO_x + low PM_{10} emissions and ***no*** PSDP. Increased odds for ever wheeze, bronchitis and obesity, and decreased odds for eczema and croup, were observed in the high joint exposure group.

4.3.22 Combined high NO_2 and PM_{10} concentrations and paternal smoking during pregnancy

Table 4.31 Associations between combined NO_2 + PM_{10} concentrations - PSDP categories and birth and child health outcomes

Outcome	↑(NO_2 + PM_{10}) C +PSDP	↓(NO_2 + PM_{10}) C +PSDP	p value	Unadjusted OR (95% CI)
Preterm birth	*27/169 (16.0)*	*4/10 (40.0)*	*0.073*	*0.285 (0.075-1.079)*
Eczema	16/75 (21.3)	2/4 (50.0)	0.222	0.271 (0.035-2.078)
Obesity	21/148 (14.2)	0/8 (0)	0.599	Cannot be calculated
LBW	11/170 (6.5)	2/9 (22.2)	0.131	0.242 (0.045-1.307)
Breathlessness	13/167 (7.8)	0/10 (0)	1.000	Cannot be calculated
M: F ratio (%)	48.1:51.9	30.0:70.0	0.339	0.463 (0.116-1.845)[fb]
DDA	32/174 (18.4)	1/9 (11.1)	1.000	1.803 (0.218-14.929)
Hay fever	23/81 (28.4)	2/4 (50.0)	0.577	0.397 (0.053-2.985)
ADHD	2/172 (1.2)	0/10 (0)	1.000	Cannot be calculated
CWB triad	9/88 (10.2)	1/6 (16.7)	0.501	0.570 (0.060-5.432)
Allergy	33/164 (20.1)	2/9 (22.2)	1.000	0.882 (0.175-4.443)
Overweight	40/147 (27.2)	1/8 (12.5)	0.682	2.617 (0.312-21.944)
IUGR	11/155 (7.1)	1/8 (12.5)	0.465	0.535 (0.060-4.745)
Bronchitis	19/177 (10.7)	2/10 (20.0)	0.312	0.481 (0.095-2.433)
Stunting	3/148 (2.0)	0/7 (0)	1.000	Cannot be calculated
Excess cough	33/167 (19.8)	4/10 (40.0)	0.221	0.369 (0.099-1.385)
Croup	16/176 (9.1)	1/9 (11.1)	0.588	0.800 (0.094-6.809)
Ever wheeze	40/167 (24.0)	3/10 (30.0)	0.707	0.735 (0.182-2.975)

OR: Odds Ratio
LBW: Low birthweight: Birthweight<2.5kg
ADHD: Attention deficit hyperactivity disorder ; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile) ; Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
IUGR: Intrauterine growth restriction
CWB triad: Symptom triad of cough, wheeze and breathlessness; C: Concentration
Combined high NO_2-PM_{10} concentration and PSDP: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m3 and paternal smoking during pregnancy
Combined low NO_2-PM_{10} concentration and NPSDP: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration < 13.91 microgram per m3 and fathers who did not smoke during pregnancy
PM_{10}: Particulate matter <10 microns; Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_2 + high PM_{10} concentrations ***and*** PSDP versus those with exposure to low NO_2 + low PM_{10} concentrations and ***no*** PSDP. Decreased odds for being born preterm were observed in the high joint exposure group.

4.3.23 Combined high NO_X and PM_{10} emissions and household member smoking during pregnancy

Table 4.32 Associations between combined NO_X + PM_{10} emissions - HSDP categories and birth and child health outcomes

Outcome	↑ (NO_X + PM_{10}) E + HSDP	↓(NO_X + PM_{10}) E + HSDP	p value	Unadjusted OR (95% CI)
Croup	**2/67 (3.0)**	**17/107 (15.9)**	**0.011**	**0.163 (0.036-0.730)**
Hay fever	**7/29 (24.1)**	**17/33 (51.5)**	**0.037**	**0.299 (0.101-0.891)**
Obesity	**12/52 (23.1)**	**8/91 (8.8)**	**0.034**	**3.133 (1.179-8.218)**
Ever wheeze	**19/64 (29.7)**	**16/100 (16.0)**	**0.043**	**2.217 (1.040-4.727)**
Breathlessness	*7/64 (10.9)*	*3/100 (3.0)*	*0.052*	*3.971 (0.988-15.966)*
M: F ratio (%)	*45.6 : 54.4*	*58.8 : 41.2*	*0.093*	*1.701 (0.929 -3.115)[fb]*
Bronchitis	9/67 (13.4)	8/110 (7.3)	0.196	1.978 (0.724-5.407)
IUGR	1/60 (1.7)	5/92 (5.4)	0.404	0.295 (0.034-2.589)
Preterm birth	6/65 (9.2)	17/107 (15.9)	0.254	0.538 (0.201-1.444)
CWB triad	3/39 (7.7)	2/54 (3.7)	0.646	2.167 (0.344-13.628)
Overweight	20/52 (38.5)	28/91 (30.8)	0.363	1.406 (0.688-2.873)
LBW	5/61 (8.2)	9/104 (8.7)	1.000	0.942 (0.301-2.953)
Allergy	14/59 (23.7)	20/98 (20.4)	0.691	1.213 (0.559-2.634)
ADHD	1/67 (1.5)	1/108 (0.9)	1.000	1.621 (0.100-26.362)
DDA	12/62 (19.4)	15/110 (13.6)	0.384	1.520 (0.661-3.495)
Eczema	8/32 (25.0)	12/27 (44.4)	0.168	0.417 (0.138-1.255)
Excess cough	14/64 (21.9)	18/100 (18.0)	0.551	1.276 (0.584-2.788)
Stunting	3/57 (5.3)	5/97 (5.2)	1.000	1.022 (0.235-4.447)

OR: Odds Ratio
HSDP: Household member smoking during pregnancy; NHSDP: Household member who did not smoke during pregnancy
LBW: Low birthweight: Birthweight < 2.5 kgs
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations; M: F: Male: Female
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions; NO_X: Oxides of Nitrogen; PM_{10}: Particulate matter <10 microns
Combined high NOx-PM10 emissions + HSDP: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
Combined low NOx-PM10 emissions + NHSDP: NO_X emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and household members who did not smoke during pregnancy
Preterm: Reported as preterm by mothers in questionnaire; fb: for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x + high PM_{10} emissions ***and*** HSDP versus those with exposure to low NO_x + low PM_{10} emissions and ***no*** HSDP. Increased odds of obesity, ever wheeze and breathlessness, and decreased odds for croup and hay fever were observed in the high joint exposure group. Females were more likely than males to be in the high joint exposure group.

4.3.24 Combined high NO_2 and PM_{10} concentrations and household member smoking during pregnancy

Table 4.33 Associations between combined NO_2 + PM_{10} concentrations - HSDP categories and birth and child health outcomes

Outcome	↑(NO_2 + PM_{10}) C + HSDP	↓(NO_2 + PM_{10}) C + NHSDP	p value	Unadjusted OR (95% CI)
Preterm birth	**40/264 (15.2)**	**5/13 (38.5)**	**0.042**	**0.286 (0.089-0.918)**
Obesity	37/221 (16.7)	0/11 (0)	0.220	Cannot be calculated
LBW	23/259 (8.9)	2/12 (16.7)	0.305	0.487 (0.101-2.360)
Eczema	37/135 (27.4)	1/5 (20.0)	1.000	1.510 (0.163-13.957)
Breathlessness	24/253 (9.5)	0/11 (0)	0.606	Cannot be calculated
M: F ratio (%)	41.8:58.2	46.2 :53.8	0.780	1.195 (0.391-3.650)[fb]
DDA	54/268 (20.1)	1/12 (8.3)	0.471	2.766 (0.551-21.970)
Hay fever	31/127 (24.4)	1/5 (20.0)	1.000	1.292 (0.139-11.993))
ADHD	11/271 (4.1)	0/12 (0)	1.000	Cannot be calculated
CWB triad	11/146 (7.5)	1/6 (16.7)	0.595	0.407 (0.044-3.801)
Allergy	49/241 (20.3)	1/12 (8.3)	0.470	2.807 (0.354-22.270)
Overweight	69/220 (31.4)	3/11 (27.3)	1.000	1.219 (0.314-4.734)
IUGR	11/239 (4.6)	1/10 (10.0)	0.395	0.434 (0.050-3.738)
Bronchitis	26/268 (9.7)	2/12 (16.7)	0.342	0.547 (0.112-2.585)
Stunting	10/232 (4.3)	0/10 (2.7)	1.000	Cannot be calculated
Excess cough	53/253 (20.9)	3/11 (27.3)	0.705	0.707 (0.181-2.756)
Croup	19/272 (7.0)	1/12 (8.3)	0.591	0.826 (0.101-6.743)
Ever wheeze	60/253 (23.7)	3/11 (27.3)	0.727	0.829 (0.213-3.224)

OR: Odds Ratio
HSDP: Household member smoking during pregnancy; NHSDP: Household member who did not smoke during pregnancy
LBW: Low birthweight: Birthweight < 2.5 kgs
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: Concentration
Combined high NO_2-PM_{10} concentration and HSDP: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m3 and household member smoking during pregnancy
Combined low NO_2-PM_{10} concentration and NHSDP: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration $\leq$ 13.91 microgram per m3 and household member who did not smoke during pregnancy
PM_{10}: Particulate matter <10 microns
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth/child health outcomes were compared between those respondents with exposure to both high NO_2 + high PM_{10} concentrations ***and*** HSDP vs. those with exposure to low NO_2 + low PM_{10} concentrations and ***no*** HSDP. Decreased odds of being born preterm were observed in the high joint exposure group.

4.3.25 Combined high NOx emissions and heavy maternal smoking during pregnancy

Table 4.34 Associations between combined NO_X emissions - maternal smoking dose categories and birth and child health outcomes

Outcome	↑ NO_X E + heavy maternal smoking	↓NO_X E + none maternal smoking	p value	Unadjusted OR* (95% CI)
Ever wheeze	**4/8 (50)**	**12/70 (17.1)**	**0.050**	**4.833 (1.088-22.075)**
DDA	**4/8 (50)**	**12/73 (16.4)**	**0.045**	**5.083 (1.114-23.192)**
Obesity	*3/8 (37.5)*	*7/59 (11.9)*	*0.091*	*4.457 (0.869-22.850)*
M : F ratio (%)	25.0 :75.0	50.0 : 50.0	0.269	3.003 (0.568-15.873)[fb]
Croup	0/8 (0)	10/70 (14.3)	0.587	Cannot be calculated
Breathlessness	1/8 (12.5)	3/70 (4.3)	0.357	3.190 (0.291-34.936)
Bronchitis	1/8 (12.5)	4/72 (5.6)	0.418	2.429 (0.237-24.844)
IUGR	1/8 (12.5)	3/64 (4.7)	0.382	2.905 (0.265-31.845)
Preterm birth	2/7 (28.6)	10/68 (14.7)	0.310	2.320 (0.394-13.645)
CWB triad	0/3 (0)	4/41 (9.8)	1.000	Cannot be calculated
Overweight	2/7 (28.6)	16/59 (27.1)	1.000	1.075 (0.189-6.109)
LBW	0/6 (0)	6/68 (8.8)	1.000	Cannot be calculated
Allergy	1/7 (14.3)	16/61 (26.2)	0.670	0.469 (0.052-4.199)
Hay fever	1/4 (25.0)	17/31 (54.8)	0.338	0.275 (0.026-2.440)
ADHD	0/8 (0)	3/70 (4.3)	1.000	Cannot be calculated
Eczema	1/4 (25.0)	11/26 (42.3)	0.632	0.455 (0.042-4.976)
Excess cough	0/8 (0)	11/70 (15.7)	0.592	Cannot be calculated
Stunting	0/7 (0)	5/64 (7.8)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
High NOx emissions and heavy maternal smoking: NOx emissions >10 and > 10 cigarettes smoked by mother during pregnancy
Low NOx and none maternal smoking: NOx emissions $\leq$ 10 and mothers who never smoked at any time during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x emissions ***and*** heavy MSDP versus those with exposure to low NO_x emissions and ***no*** MSDP. Increased odds of ever wheeze, doctor diagnosed asthma and obesity were observed in the high joint exposure group.

4.3.26 Combined high PM_{10} emissions and heavy maternal smoking during pregnancy

Table 4.35 Associations between combined PM_{10} emissions - maternal smoking dose categories and birth and child health outcomes

Outcome	↑ PM_{10} E + heavy maternal smoking	↓ PM_{10} E + none maternal smoking	p value	Unadjusted OR (95% CI)
Ever wheeze	1/2 (50.0)	27/159 (17.0)	0.318	4.889 (0.297-80.601)
Obesity	1/2 (50.0)	17/136 (12.5)	0.245	7.000 (0.418-117.204)
M: F ratio (%)	50.0 : 50.0	47.4 : 52.6	1.000	0.900 (0.055-14.075)[fb]
Croup	0/2 (0)	19/162 (11.7)	1.000	Cannot be calculated
Breathlessness	0/2 (0)	11/148 (74.3)	1.000	Cannot be calculated
Bronchitis	0/2 (0)	11/153 (71.9)	1.000	Cannot be calculated
IUGR	0/2 (0)	7/146 (4.8)	1.000	Cannot be calculated
Preterm birth	1/2 (50)	26/159 (16.4)	0.308	5.115 (0.310-84.415)
CWB triad	0/2 (0)	8/94 (8.5)	1.000	Cannot be calculated
Overweight	1/2 (50.0)	39/136 (28.7)	0.497	2.487 (0.152-40.762)
LBW	0/1 (0)	12/163 (7.4)	1.000	Cannot be calculated
Allergy	0/1 (0)	40/145 (27.6)	1.000	Cannot be calculated
Hay fever	1/1 (100)	31/69 (44.9)	0.457	Cannot be calculated
ADHD	0/2 (0)	5/163 (3.1)	1.000	Cannot be calculated
DDA	1/2 (50)	28/165 (17.0)	0.318	4.093 (0.287-80.578)
Eczema	0	29/67 (43.3)	1.000	Cannot be calculated
Excess cough	0/2 (0)	32/159 (20.1)	1.000	Cannot be calculated
Stunting	0/2 (0)	9/146 (6.2)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5 kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile) ; Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions
High PM_{10} emissions and heavy maternal smoking: PM_{10} emissions >5 and >10 cigarettes smoked by mother during pregnancy
Low PM_{10} emissions and none maternal smoking: PM_{10} emissions $\leq$ 5 and mothers who never smoked at any time during pregnancy
PM_{10}: Particulate matter < 10 microns
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} emissions ***and*** heavy MSDP versus those with exposure to low PM_{10} emissions and ***no*** MSDP. Because of the small numbers in the high exposure group, no statistically significant associations were observed.

4.3.27 Combined high NO_2 concentrations and heavy maternal smoking during pregnancy

Table 4.36 Associations between combined NO_2 concentrations - maternal smoking dose categories and birth and child health outcomes

Outcome	↑ NO_2 C + heavy maternal smokers	↓ NO_2 C + none maternal smokers	p value	Unadjusted OR (95% CI)
Ever wheeze	7/15 (46.7)	3/7 (42.9)	1.000	1.167 (0.191-7.116)
Obesity	5/12 (41.7)	0/5 (0)	0.245	Cannot be calculated
M: F ratio (%)	40.0 :60.0	75.0 : 25.0	0.657	0.050 (0.074-3.356)[fb]
Croup	1/13 (7.7)	0/8 (0)	1.000	Cannot be calculated
Breathlessness	2/15 (13.3)	0/7 (0)	1.000	Cannot be calculated
Bronchitis	2/14 (14.3)	2/8 (25.0)	0.602	0.500 (0.056-4.473)
IUGR	2/13 (15.4)	1/6 (16.7)	1.000	0.909 (0.066-12.524)
Preterm birth	3/12 (25.0)	2/8 (25.0)	1.000	1.000 (0.127-7.893)
CWB triad	0/6 (0)	0/4 (0)	1.000	Cannot be calculated
Overweight	4/11 (36.4)	1/5 (20.0)	1.000	2.286 (0.185-28.186)
LBW	1/13 (7.7)	1/7 (14.3)	1.000	0.500 (0.026-9.4457)
Allergy	2/13 (15.4)	2/7 (28.6)	0.587	0.455 (0.049-4.214)
Hay fever	1/6 (16.7)	2/4 (50.0)	0.500	0.200 (0.011-3.661)
ADHD	0/15 (0)	0/8(0)	1.000	Cannot be calculated
DDA	6/15 (40.0)	1/8 (12.5)	0.345	4.667 (0.451-48.257)
Eczema	2/6 (33.3)	3/4 (75.0)	0.524	0.167 (0.010-2.821)
Excess cough	3/15 (20)	3/7 (42.9)	0.334	0.333 (0.047-2.366)
Stunting	0/14 (0)	0/6 (0)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C : Concentrations
High NO_2 concentrations and heavy maternal smoking: NO_2 concentrations > 17.01 and > 10 cigarettes smoked by mother during pregnancy
Low NO_2 and none maternal smoking: NO_2 concentrations $\leq$ 17.01 and mothers who never smoked at any time during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_2 concentrations ***and*** heavy MSDP versus those with exposure to low NO_2 concentrations and ***no*** MSDP. Again, because of the small numbers in the high exposure group, no statistically significant associations were observed.

4.3.28 Combined high PM_{10} concentrations and heavy maternal smoking during pregnancy

Table 4.37 Associations between combined PM_{10} concentrations - maternal smoking dose categories and birth and child health outcomes

Outcome	↑ PM_{10} C + heavy maternal smokers	↓ PM_{10} C + none maternal smokers	p value	Unadjusted OR (95% CI)
Obesity	*4/11 (36.4)*	*1/17 (5.9)*	*0.062*	*9.143 (0.859-97.265)*
Ever wheeze	7/14 (50.0)	4/18 (22.2)	0.142	3.500 (0.760-16.118)
M: F ratio (%)	42.9 : 57.1	38.1 : 61.9	1.000	0.820 (0.207-3.247)[fb]
Croup	1/13 (7.7)	2/21 (9.5)	1.000	0.792 (0.065-9.711)
Breathlessness	2/14 (14.3)	0/18 (0)	0.183	Cannot be calculated
Bronchitis	2/13 (15.4)	2/20 (10)	1.000	1.636 (0.201-13.344)
IUGR	2/13 (15.4)	1/17 (5.9)	0.565	2.909 (0.234-36.164)
Preterm birth	3/12 (25.0)	2/21 (9.5)	0.328	3.167 (0.447-22.416)
CWB triad	0/5 (0)	1/12 (8.3)	1.000	Cannot be calculated
Overweight	3/10 (30.0)	3/17 (17.6)	0.638	2.000 (0.318-12.588)
LBW	1/12 (8.3)	3/19 (15.8)	1.000	0.485 (0.044-5.290)
Allergy	2/13 (15.4)	4/20 (20.0)	1.000	0.727 (0.113-4.685)
Hay fever	1/6 (16.7)	2/5 (40.0)	0.545	0.300 (0.018-4.908)
ADHD	0/14 (0)	0/20 (0)	1.000	Cannot be calculated
DDA	6/14 (42.9)	4/20 (20.0)	0.252	3.000 (0.654-13.764)
Eczema	2/6 (33.3)	4/6 (66.7)	0.567	0.250 (0.023-2.757)
Excess cough	3/14 (21.4)	6/18 (33.3)	0.694	0.545 (0.109-2.727)
Stunting	0/13 (0)	2/19 (10.5)	0.502	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C : Concentrations
High PM_{10} concentrations and heavy maternal smoking: PM_{10} concentrations > 13.91 and > 10 cigarettes smoked by mother during pregnancy
Low PM_{10} concentrations and none maternal smoking: PM_{10} concentrations $\leq$ 13.91 and mothers who never smoked at any time during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} concentrations ***and*** heavy MSDP versus those with exposure to low PM_{10} concentrations and ***no*** MSDP. Increased odds of obesity were observed in the high exposures group.

4.3.29 Combined high NOx emissions and heavy paternal smoking during pregnancy

Table 4.38 Associations between combined NOx emissions - paternal smoking dose categories and birth and child health outcomes

Outcome	↑ NO_X E + heavy paternal smoking	↓NO_X E + none paternal smoking	p value	Unadjusted OR (95% CI)
Breathlessness	*2/8 (25.0)*	*3/73 (4.1)*	*0.074*	*7.778 (1.080-55.991)*
LBW	*4/8 (50.0)*	*6/67 (9.0)*	*0.009*	*10.167 (2.012-51.366)*
Ever wheeze	2/8 (25.0)	12/73 (16.4)	0.621	1.694 (0.305-9.423)
Obesity	2/8 (25.0)	5/59 (8.0)	0.193	3.600 (0.569-22.759)
M: F ratio (%)	25.0 :75.0	50.7 : 49.3	0.267	3.077 (0.584-16.129)[fb]
Croup	0/8 (0)	8/70 (11.4)	0.591	Cannot be calculated
Bronchitis	1/8 (12.5)	5/72 (6.9)	0.480	1.914 (0.195-18.787)
IUGR	0/7 (0)	5/66 (7.6)	1.000	Cannot be calculated
Preterm birth	1/8 (12.5)	14/69 (20.3)	1.000	0.561 (0.064-4.944)
CWB triad	1/3 (33.3)	3/37 (8.1)	0.277	5.667 (0.390-82.237)
Overweight	4/8 (50.0)	15/59 (25.4)	0.209	2.933 (0.651-13.208)
Allergy	1/7 (14.3)	11/61 (18.0)	1.000	0.758 (0.083-6.943)
Hay fever	1/4 (25.0)	15/26 (57.7)	0.315	0.244 (0.022-2.676)
ADHD	0/8 (0)	2/72 (2.8)	1.000	Cannot be calculated
DDA	2/7 (28.6)	10/74 (13.5)	0.276	2.560 (0.436-15.031)
Eczema	1/4 (25.0)	7/21 (33.3)	1.000	0.667 (0.058-7.635)
Excess cough	2/8 (25.0)	10/73 (13.7)	0.338	2.100 (0.371-11.891)
Stunting	0/7 (0)	3/63 (4.8)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness; E: Emissions
High NOx emissions and heavy paternal smoking: NOx emissions >10 and > 10 cigarettes smoked by father during pregnancy
Low NOx and none paternal smoking: NOx emissions $\leq$ 10 and fathers who never smoked at any time during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x emissions ***and*** heavy PSDP versus those with exposure to low NO_x emissions and ***no*** PSDP. Increased odds of breathlessness and being born LBW were observed in the high exposures group.

4.3.30 Combined high PM_{10} emissions and heavy paternal smoking during Pregnancy

Table 4.39 Associations between combined PM_{10} emissions-paternal smoking dose categories and birth and child health outcomes

Outcome	↑ PM_{10} E + heavy paternal smoking	↓PM_{10} E and none paternal smoking	p value	Unadjusted OR* (95% CI)
Breathlessness	**2/4 (50.0)**	**10/157(6.4)**	**0.028**	**14.200 (1.870-115.564)**
Overweight	3/4 (75.0)	34/128 (26.6)	0.067	8.294 (0.834-82.472)
Obesity	1/4 (25.0)	16/129 (12.4)	0.425	2.345 (0.231-24.026)
Ever wheeze	1/4 (25.0)	29/157 (18.5)	0.565	1.471 (0.148-14.657)
M: F ratio (%)	0 :100	47.3 : 52.7	0.125	Cannot be calculated
Croup	0/4 (0)	19/139 (12.0)	1.000	Cannot be calculated
Bronchitis	0/4 (0)	13/161 (8.1)	1.000	Cannot be calculated
IUGR	0/4 (0)	8/147 (5.4)	1.000	Cannot be calculated
Preterm birth	0/4 (0)	28/156 (17.9)	1.000	Cannot be calculated
CWB triad	0/2 (0)	5/81 (6.2)	1.000	Cannot be calculated
LBW	1/4 (25.0)	11/158 (7.0)	0.267	4.455 (0.427-46.458)
Allergy	0/3 (0)	35/142 (24.6)	1.000	1.327 (1.208-1.458)
Hay fever	1/3 (33.3)	27/65 (41.5)	1.000	0.704 (0.061-8.160)
ADHD	0/4 (0)	3/160 (1.9)	1.000	Cannot be calculated
DDA	1/3(33.3)	26/162 (16.0)	0.417	2.615 (0.229-29.912)
Eczema	0/2 (0)	25/63 (39.7)	0.519	Cannot be calculated
Excess cough	1/4 (25.0)	27/157 (17.2)	0.538	1.605 (0.161-16.021)
Stunting	0/4 (0)	6/138 (4.3)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5 kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E : Emissions
High PM_{10} emissions and heavy paternal smoking: PM_{10} emissions >5 and >10 cigarettes smoked by father during pregnancy period
Low PM_{10} emissions and none paternal smoking: PM_{10} emissions $\leq$ 5 and fathers who never smoked at any time during pregnancy
PM_{10}: Particulate matter < 10 microns
Preterm: Reported as preterm by mothers in questionnaire

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} emissions ***and*** heavy PSDP versus those with exposure to low PM_{10} emissions and ***no*** PSDP. Increased odds of breathlessness and being overweight were observed in the high exposures group.

4.3.31 Combined high NO_2 concentrations and heavy paternal smoking during pregnancy

Table 4.40 Associations between combined NO_2 concentrations- paternal smoking dose categories and birth and child health outcomes

Outcome	↑ NO_2 C + heavy paternal smokers	↓NO_2 C + none paternal smokers	p value	Unadjusted OR* (95%)
Ever wheeze	3/13 (23.1)	2/7 (28.6)	1.000	0.750 (0.093-6.043)
Obesity	4/12 (33.3)	0/6 (0)	0.245	Cannot be calculated
M: F ratio (%)	30.8 : 69.2	28.6 :71.4	1.000	0.900 (0.120-6.756)[fb]
Croup	2/12 (16.7)	0/7 (0)	0.509	Cannot be calculated
Breathlessness	2/13 (15.4)	0/7 (0)	0.521	Cannot be calculated
Bronchitis	3/13 (23.1)	1/7 (14.3)	1.000	1.800 (0.151-21.477)
IUGR	0/10 (0)	1/6 (16.7)	0.375	Cannot be calculated
Preterm birth	1/11(9.1)	1/7 (14.3)	1.000	0.600 (0.031-11.473)
CWB triad	1/7 (14.3)	0/4 (0)	1.000	Cannot be calculated
Overweight	8/12 (66.7)	1/6 (16.7)	0.131	10.01(0.855-117.017)
LBW	4/12 (33.3)	1/6 (16.7)	0.615	2.500 (0.264-29.254)
Allergy	3/11 (27.3)	1/7 (14.3)	1.000	2.250 (0.185-27.369)
Hay fever	2/6 (33.3)	1/2 (50.0)	1.000	0.500 (0.019-12.898)
ADHD	0/13 (0)	13/13 (100)	1.000	Cannot be calculated
DDA	3/12 (25.0)	0/7 (0)	0.263	Cannot be calculated
Eczema	1/5 (20.0)	2/2 (100)	0.143	Cannot be calculated
Excess cough	4/13 (30.8)	1/7 (14.3)	0.613	2.667 (0.237-30.066)
Single parent	2/12 (16.7)	4/6 (66.7)	0.101	0.100 (0.010-0.975)
Stunting	0/0 (0)	12/12 (100)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: Concentrations
High NO_2 concentrations and heavy paternal smoking: NO_2 concentrations > 17.01 and > 10 cigarettes smoked by father during pregnancy
Low NO2 and none paternal smoking: NO_2 concentrations < 17.01 and fathers who never smoked at any time during pregnancy
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_2 concentrations ***and*** heavy PSDP versus those with exposure to low NO_2 concentrations and ***no*** PSDP. Because of the small numbers in the high exposure group, no statistically significant associations were observed.

4.3.32 Combined high PM_{10} concentrations and heavy paternal smoking during pregnancy

Table 4.41 Associations between combined PM_{10} concentrations- paternal smoking dose categories and birth and child health outcomes

Outcome	↑ PM_{10} C + heavy paternal smokers	↓ PM_{10} C + none paternal smoking	p value	Unadjusted OR* (95% CI)
Overweight	**7/8 (87.5)**	**3/17 (17.6)**	**0.002**	**32.667 (2.852-374.155)**
Obesity	**3/8 (37.5)**	**0/17 (0)**	**0.024**	**Cannot be calculated**
DDA	*2/8*	*0/17 (0)*	*0.093*	*Cannot be calculated*
Ever wheeze	2/9 (22.2)	2/16 (12.5)	0.602	2.000 (0.237-17.338)
M:F ratio (%)	22.2 :77.8	55.6 : 44.4	0.214	4.367 (0.705-27.027)[fb]
Croup	2/9 (22.2)	2/18 (11.1)	0.582	2.286 (0.266-19.658)
Breathlessness	2/9 (22.2)	0/16 (0)	0.120	0.778 (0.549-1.103)
Bronchitis	2/9 (22.2)	1/16 (5.9)	0.268	4.571 (0.354-59.106)
IUGR	0/8 (0)	1/14 (7.1)	1.000	Cannot be calculated
Preterm birth	1/8 (12.5)	1/18 (5.6)	0.529	2.429 (0.133-44.501)
CWB triad	0/5 (0)	1/8 (12.5)	1.000	Cannot be calculated
Overweight	7/8 (87.5)	3/17 (17.6)	0.002	32.667 (2.852-374.155)
LBW	2/8 (25.0)	2/15 (13.3)	0.589	2.167 (0.244-19.276)
Allergy	2/8 (25.0)	2/17 (11.8)	0.570	2.500 (0.289-22.042)
Hay fever	2/6 (33.3)	2/4 (50.0)	1.000	0.500 (0.037-6.683)
ADHD	0/9 (0)	9/9 (100)	1.000	Cannot be calculated
Eczema	0/4 (0)	2/3 (66.7)	0.143	3.000 (0.606-14.864)
Excess cough	3/9 (33.3)	2/16 (12.5)	0.312	3.500 (0.460-26.616)
Stunting	0/8 (0)	8/8 (100)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5kg
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
C: Concentrations
High PM_{10} concentrations and heavy paternal smoking: PM_{10} concentrations > 13.91 and > 10 cigarettes smoked by father during pregnancy period
Low PM_{10} concentrations and none paternal smoking: PM_{10} concentrations < 13.91 and fathers who never smoked at any time during pregnancy
Preterm: Reported as preterm by mothers in questionnaire; fb: for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high PM_{10} concentrations ***and*** heavy PSDP versus those with exposure to low PM_{10} concentrations and ***no*** PSDP. Increased odds of obesity / being overweight and doctor diagnosed asthma were observed in the high exposures group.

4.3.33 Combined high NOx-PM_{10} emissions and heavy maternal smoking during pregnancy

Table 4.42 Associations between combined NOx+PM_{10} emissions – maternal smoking dose categories and birth and child health outcomes

Outcome	↑ (NO_X + PM_{10} E) +heavy maternal smokers	↓(NO_X + PM_{10} E) + maternal none smokers	p value	Unadjusted OR* (95% CI)
Obesity	1/2 (50.0)	7/59 (11.9)	0.331	4.833 (0.416-132.591)
Preterm birth	1/2 (50.0)	10/68 (14.7)	0.292	5.800 (0.335-100.459)
LBW	0/1 (0)	6/68 (8.8)	1.000	Cannot be calculated
Eczema	0/0 (0)	11/26 (42.3)	1.000	Cannot be calculated
Breathlessness	0/2 (0)	3/70 (4.3)	1.000	Cannot be calculated
M: F ratio (%)	50.0 : 50.0	47.4 : 52.6	1.000	0.900(0.055-14.706)[fb]
DDA	1/2 (50.0)	12/73 (16.4)	0.319	5.083 (0.297-87.011)
Hay fever	1/1 (100)	17/31 (54.8)	1.000	Cannot be calculated
ADHD	0/2 (0)	3/70 (4.3)	1.000	Cannot be calculated
CWB triad	0/2 (0)	4/41 (9.8)	1.000	Cannot be calculated
Allergy	0/6 (0)	16/61 (26.2)	1.000	Cannot be calculated
Overweight	1/2 (50.0)	16/59 (27.1)	0.483	2.688 (0.159-45.569)
IUGR	0/2 (0)	3/64 (4.7)	1.000	Cannot be calculated
Bronchitis	0/2 (0)	4/72 (5.6)	1.000	Cannot be calculated
Stunting	0/2 (0)	5/64 (7.8)	1.000	Cannot be calculated
Excess cough	0/2 (0)	11/70 (15.7)	1.000	Cannot be calculated
Croup	0/2 (0)	10/70 (14.3)	1.000	Cannot be calculated
Ever wheeze	1/2 (50.0)	12/70 (17.1)	0.331	4.833 (0.282-82.780)

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5 kgs
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions; NO_X: Oxides of Nitrogen; PM_{10}: Particulate matter <10 microns
High NOx-PM_{10} emissions + heavy maternal smoking: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and > 10 cigarettes smoked by mother during pregnancy
Low NOx-PM_{10} emissions + maternal none smoking: NO_X emissions < 10 + PM_{10} emissions < 5 (in tonnes per annum) and mothers who did not smoke during pregnancy.
Preterm: Reported as preterm by mothers in questionnaire
fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x - PM_{10} emissions ***and*** heavy MSDP versus those with exposure to low NO_x - PM_{10} emissions and ***no*** MSDP. Because of the small numbers in the high exposure group, no statistically significant associations were observed.

4.3.34 Combined high NOx-PM_{10} emissions and heavy paternal smoking during pregnancy

Table 4.43 Associations between combined NOx+PM_{10} emissions - paternal smoking dose categories and birth and child health outcomes

Outcome	↑ (NO_X + PM_{10}) E+ heavy paternal smokers	↓(NO_X + PM_{10}) E+ paternal none smokers	p value	Unadjusted OR* (95% CI)
Breathlessness	**2/4 (50.0)**	**3/73 (4.1)**	**0.019**	**23.37(2.398-227.045)**
Overweight	*3/4 (75.0)*	*15/59 (25.4)*	*0.067*	*8.800 (0.850-91.155)*
Eczema	0/2 (0)	7/21 (33.3)	1.000	Cannot be calculated
Croup	0/4 (0)	8/70 (11.4)	1.000	Cannot be calculated
Bronchitis	0/4 (0)	5/72 (6.9)	1.000	Cannot be calculated
M: F ratio (%)	0 : 100	47.3 :52.7	0.125	Cannot be calculated
Ever wheeze	1/4 (25.0)	12/73 (16.4)	0.530	1.694 (0.162-17.701)
IUGR	0/4 (0)	5/66 (7.6)	1.000	Cannot be calculated
Obesity	1/4 (25.0)	5/59 (8.5)	0.337	3.600 (0.313-41.369)
Preterm birth	0/4(0)	14/69 (20.3)	1.000	Cannot be calculated
CWB triad	0/2 (0)	3/37 (8.1)	1.000	Cannot be calculated
LBW	1/4 (25.0)	6/67 (9.0)	0.346	3.389 (0.303-37.865)
Allergy	0/3 (0)	11/61 (18.0)	1.000	Cannot be calculated
Hay fever	1/3 (33.3)	15/26 (57.7)	0.573	0.367 (0.029-4.573)
ADHD	0/4 (0)	2/72 (2.8)	1.000	Cannot be calculated
DDA	1/3 (33.3)	10/74 (13.5)	0.374	3.200 (0.265-38.645)
Excess cough	1/4 (25.0)	10/73 (13.7)	0.467	2.100 (0.198-22.229)
Stunting	0/4 (0)	3/63 (4.8)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5 kgs
ADHD: Attention deficit hyperactivity disorder
DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
M: F: Male: Female
CWB triad: Symptom triad of cough, wheeze and breathlessness
E: Emissions; NOX: Oxides of Nitrogen; PM_{10}: Particulate matter <10 microns
High NOx-PM_{10} emissions + heavy paternal smoking: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and > 10 cigarettes smoked by father during pregnancy period
Low NOx-PM_{10} emissions + paternal none smoking: NO_X emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy.
Preterm: Reported as preterm by mothers in questionnaire ; fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x - PM_{10} emissions ***and*** heavy PSDP versus those with exposure to low NO_x - PM_{10} emissions and ***no*** PSDP. Again, because of the small numbers in the high exposure group, no statistically significant associations were observed.

4.3.35 Combined high NO_2-PM_{10} concentrations and heavy maternal smoking during pregnancy

Table 4.44 Associations between combined NO_2+PM_{10} concentrations - maternal smoking dose categories birth and child health outcomes

Outcome	↑(NO_2 + PM_{10}) C + heavy maternal smokers	↓(NO_2 + PM_{10}) C +none smokers	p value	Unadjusted OR* 95% (CI)
Eczema	2/6 (33.3)	2/3 (66.7)	0.524	0.250 (0.013-4.725)
Obesity	4/11 (36.4)	0/5 (0)	0.245	Cannot be calculated
LBW	1/12 (8.3)	1/6 (16.7)	1.000	0.455 (0.023-8.829)
breathlessness	2/14 (14.3)	0/6 (0)	1.000	Cannot be calculated
M: F ratio (%)	42.9 :57.1	28.6 :71.4	0.738	0.761 (0.194-2.994)[fb]
DDA	6/14 (42.9)	1/7 (14.3)	0.337	4.500 (0.422-47.988)
Hay fever	1/6 (16.7)	2/3 (66.7)	0.226	0.100 (0.004-2.504)
ADHD	0/14 (0)	0/7 (0)	1.000	Cannot be calculated
CWB triad	0/5 (0)	0/3 (7.7)	1.000	Cannot be calculated
Allergy	2/13 (15.4)	2/6 (33.3)	0.557	0.364 (0.038-3.518)
Overweight	3/10 (30.0)	1/5 (20.0)	1.000	1.714 (0.131-22.573)
IUGR	2/13 (15.4)	1/5 (20.0)	1.000	0.727 (0.051-10.390)
Bronchitis	2/13 (15.4)	2/7 (28.6)	0.587	0.455 (0.049-4.214)
Preterm birth	3/12 (25.0)	2/7 (28.6)	1.000	0.833 (0.102-6.783)
Stunting	0/13 (0)	0/5 (0)	1.000	Cannot be calculated
Excess cough	3/14 (21.4)	3/6 (50.0)	0.303	0.273 (0.035-2.112)
Croup	1/13 (7.7)	0/7 (0)	1.000	Cannot be calculated
Ever wheeze	7/14 (50.0)	3/60 (50.0)	1.000	1.000 (0.148-6.772)

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5 kgs
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
M: F : Male : Female
CWB triad: Symptom triad of cough, wheeze and breathlessness; C: concentration
Combined high NO_2-PM_{10} concentration and heavy maternal smoking during pregnancy: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m3 and > 10 cigarettes smoked by mother during pregnancy
Combined low NO_2-PM_{10} concentration and none maternal smoker during pregnancy: NO_2 concentration ≤ 17.01 and PM_{10} concentration ≤ 13.91 microgram per m3 and mothers who did not smoke during pregnancy
PM_{10}: Particulate matter <10 microns; Preterm: Reported as preterm by mothers in questionnaire; fb :for female births

In this analysis, birth and child health outcomes were compared between those respondents with exposure to both high NO_x - PM_{10} concentrations ***and*** heavy MSDP versus those with exposure to low NO_x - PM_{10} concentrations and ***no*** MSDP. No statistically significant associations were observed.

4.3.36 Combined high NO_2-PM_{10} concentrations and heavy paternal smoking during pregnancy

Table 4.45 Associations between combined NO_2+PM_{10} concentrations - paternal smoking dose categories birth and child health outcomes

Outcome	↑ (NO_2 + PM_{10}) C+ heavy paternal smokers	↓ (NO_2 + PM_{10}) C + paternal none smokers	p value	Unadjusted OR (95% CI)
Overweight	**7/8 (87.5)**	**1/5 (20.0)**	**0.032**	**28.00 (1.350-580.591)**
Croup	2/9 (22.2)	0/5 (0)	0.505	Cannot be calculated
Hay fever	2/6 (33.3)	1/1 (100.0)	0.429	Cannot be calculated
Obesity	3/8 (37.5)	0/5 (0)	0.231	Cannot be calculated
Ever wheeze	2/9 (22.2)	2/5 (40.0)	0.580	0.429 (0.040-4.633)
Breathlessness	2/9 (22.2)	0/5 (0)	0.505	Cannot be calculated
M: F ratio (%)	22.2 :77.8	50.0 : 50.0	0.234	3.497 (0.579-21.277)[fb]
Bronchitis	2/9 (22.2)	1/5 (20.0)	1.000	1.143 (0.077-16.947)
IUGR	0/8 (0)	1/4 (25.0)	0.333	Cannot be calculated
Preterm birth	1/7 (12.8)	1/5 (20.0)	1.000	0.571 (0.028-11.843)
CWB triad	0/5 (0)	0/3 (0)	1.000	Cannot be calculated
LBW	2/8 (25.0)	1/4 (25.0)	1.000	1.000 (0.284-22.042)
Allergy	2/8 (25.0)	1/5 (20.0)	1.000	1.337 (0.088-20.108)
ADHD	0/9 (0)	0/5 (0)	1.000	Cannot be calculated
DDA	2/8 (25.0)	0/5 (0)	0.487	Cannot be calculated
Eczema	0/4 (0)	1/1 (100.0)	0.200	Cannot be calculated
Excess cough	3/9 (33.3)	1/5 (20.0)	1.000	2.000 (0.150-26.735)
Stunting	0/8 (0)	0/3(0)	1.000	Cannot be calculated

OR: Odds Ratio * not calculated for many of the outcomes due to small numbers
LBW: Low birthweight: Birthweight < 2.5 kgs ; M: F: Male: Female
ADHD: Attention deficit hyperactivity disorder; DDA: Doctor diagnosed asthma
IUGR: Intrauterine growth restriction: Term baby and less than 2.5 kgs
Stunting: child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile); Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness; C: concentration
Combined high NO_2-PM_{10} concentration and heavy paternal smoking during pregnancy: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m3 and > 10 cigarettes smoked by father during pregnancy
Combined low NO_2-PM_{10} concentration and none paternal smoker during pregnancy: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration $\leq$ 13.91 microgram per m3 and fathers who did not smoke during pregnancy
PM_{10}: Particulate matter <10 microns
Preterm: Reported as preterm by mothers in questionnaire; fb: for female births

In this analysis, birth/child health outcomes were compared between those respondents with exposure to both high NO_x - PM_{10} concentrations ***and*** heavy PSDP versus those with exposure to low NO_x - PM_{10} concentrations and ***no*** PSDP. Increased odds of being overweight were observed in the high exposures group.

4.3.37 Summary of univariate analysis and estimation of odds ratios for birth and child health outcomes in relation to air pollution indicators and combined air pollution and pregnancy smoking exposure indicators

Table 4.46 Summary of univariate analysis and estimation of odds ratios for birth and child health outcomes in relation to air pollution indicators and combined air pollution and pregnancy smoking exposure indicators

Outcome	High Exposure No (%)	Low exposure No (%)	P value	Unadjusted OR (95% CI)
	↑ **NOx E**	↓ **NOx E**		
Hay Fever	46/195 (23.6)	30/73 (41.1)	0.006	0.443 (0.250-0.784)
Croup	32/438 (7.3)	26/196 (13.3)	0.024	0.515 (0.298-0.891)
	↑ **NO_2 C**	↓ **NO_2 C**		
Preterm birth	95/640 (14.8)	7/20 (35.0)	0.024	0.324 (0.126-0.832)
	↑ **PM_{10} E**	↓ **PM_{10} E**		
Ever wheeze	36/133 (27.1)	80/469 (17.1)	0.013	1.805 (1.149-2.836)
Croup	7/138 (5.1)	51/496(10.3)	0.066	0.466 (0.207-1.052)
Breathlessness	14/133(10.5)	28/469 (6.0)	0.082	1.853 (0.946-3.631)
	↑ **PM_{10} C**	↓ **PM_{10} C**		
Obesity	71/504 (14.0)	2/49 (4.1)	0.047	3.853 (0.916-16.218)
LBW	44/612 (7.2)	8/51 (15.7)	0.080	0.416 (0.184-0.940)
	↑ **(NO_X + PM_{10}) E**	↓ **(NO_X + PM_{10}) E**		
Croup	7/138 (5.1)	26/196 (13.3)	0.015	0.349 (0.147-0.830)
Ever wheeze	36/133 (27.1)	32/186 (17.2)	0.038	1.786 (1.041-3.064)
Hay fever	16/63 (25.4)	30/73(41.1)	0.069	0.488 (0.234-1.017)
M:F	42.3:57.7	52.4:47.6	0.066	1.504 (0.978-2.309)
	↑ **(NO_2 + PM_{10}) C**	↓ **(NO_2 + PM_{10}) C**		
Preterm birth	93/601 (15.5)	7/18 (38.9)	0.016	0.288 (0.109-0.761)
	↑ **NO_X E + MSDP**	↓ **NO_X E + NMSDP**		
Ever wheeze	29/104 (27.9)	20/137 (14.6)	0.015	2.262 (1.194-4.286)
Obesity	20/87 (23.0)	10/125 (8.0)	0.003	3.433 (1.517-7.768)
M: F ratio (%)	33.9 :66.1	56.8 : 43.2	<0.001	2.567 (1.545-4.251)[fb]
Croup	5/110 (4.5)	17/147 (11.6)	0.069	0.364 (0.130-1.020)
Hay fever	13/53 (24.5)	22/54 (40.7)	0.099	0.473 (0.206-1.082)
	↑ **NO_X E + PSDP**	↓ **NO_X E + NPSDP**		
Eczema	8/150 (16.0)	17/34 (50.0)	0.001	0.190 (0.069-0.524)
Ever wheeze	32/108 (29.6)	14/92 (15.2)	0.018	2.346 (1.161-4.738)

Obesity	14/95 (14.7)	5/91 (5.5)	0.050	2.973 (1.025-8.626)
Croup	6/113 (5.3)	14/104 (13.5)	0.050	0.360 (0.133-0.977)
Bronchitis	12/113 (10.6)	4/103 (3.9)	0.071	2.941 (0.917-9.428)
Hay fever	12/51 (23.5)	14/33 (42.4)	0.091	0.418 (0.162-1.076)
	↑ NO_X E + HSDP	**↓ NO_X E + NHSDP**		
M: F ratio (%)	42.1:57.9	58.8 :41.2	0.006	1.961 (1.221-3.155)[fb]
Croup	8/178 (4.5)	17/107 (15.9)	0.002	0.249 (0.104-0.600)
Obesity	28/143 (19.6)	8/91 (8.8)	0.027	2.526 (1.096-5.822)
Ever wheeze	41/164 (25.0)	16/100 (16.0)	0.092	1.750 (0.922-3.322)
Breathlessness	15/164 (9.1)	3/100 (3.0)	0.076	3.235 (0.918-11.541)
Hay fever	20/81 (24.7)	17/33 (51.5)	0.008	0.309 (0.132-0.721)
Eczema	23/87 (26.4)	12/27 (44.4)	0.096	0.449 (0.183-1.101)
	↑ PM_{10} E + MSDP	**↓ PM_{10} E + NMSDP**		
Ever wheeze	14/44 (31.8)	53/348 (15.2)	0.010	2.597 (1.292-5.233)
Obesity	9/37 (24.3)	32/309 (10.4)	0.026	2.782 (1.207-6.416)
M: F ratio (%)	38.3 : 61.7	54.6 : 45.4	0.044	1.942 (1.043-3.610)[fb]
Breathlessness	7/46 (15.2)	24/370 (6.5)	0.065	2.588 (1.047-6.394)
	↑ PM_{10} E + PSDP	**↓ PM_{10} E + NPSDP**		
Ever wheeze	14/46 (30.4)	37/244 (15.2)	0.019	2.448 (1.193-5.023)
Croup	1/48 (2.1)	30/270 (11.1)	0.062	0.170 (0.023-1.279)
	↑ PM_{10} E + HSDP	**↓ PM_{10} E + NHSDP**		
Ever wheeze	19/64 (29.7)	41/268 (15.3)	0.011	2.338 (1.244-4.394)
Obesity	12/52 (23.1)	24/231 (10.4)	0.020	2.588 (1.197-5.595)
Croup	2/67(3.0)	35/283 (12.4)	0.025	0.218 (0.051-0.930)
Breathlessness	7/64 (10.9)	12/268 (4.5)	0.067	2.620 (0.988-6.948)
Bronchitis	9/67 (13.4)	17/286 (5.9)	0.064	2.455 (1.043-5.781)
	↑ NO_2 C + MSDP	**↓ NO_2 C + NMSDP**		
Preterm birth	19/158 (12.0)	6/16 (37.5)	0.014	0.228 (0.074-0.698)
	↑ NO_2 C + PSDP	**↓ NO_2 C + NPSDP**		
Preterm birth	27/177 (18.3)	4/11 (36.4)	0.086	0.315 (0.086-1.150)
	↑ NO_2 C + HSDP	**↓ NO_2 C + NHSDP**		
Preterm birth	40/274 (14.6)	**5/15 (33.3)**	0.065	0.342 (0.111-1.053)
	↑ PM_{10} C + MSDP	**↓ PM_{10} C + NMSDP**		
Obesity	27/130 (20.8)	1/41 (2.4)	0.003	10.485 (1.578-79.762)
M: F ratio (%)	35.4 : 64.6	56.3 : 43.8	0.012	2.347 (1.222-4.525)[fb]
	↑ PM_{10} C+ PSDP	**↓ PM_{10} C + NPSDP**		
LBW	11/170 (6.5)	6/31 (19.4)	0.029	0.288 (0.098-0.849)

Obesity	21/148 (14.2)	0/32 (0)	0.028	Cannot be calculated
Eczema	16/75 (21.3)	7/11 (63.6)	0.007	0.155 (0.040-0.596)
Hay fever	23/81 (28.4)	6/11 (54.5)	0.094	0.330 (0.092-1.190)
	↑ PM_{10} C+ HSDP	**↓ PM_{10} C + NHSDP**		
Obesity	37/221 (16.7)	0/34 (0)	0.007	Cannot be calculated
M: F ratio (%)	41.8 :58.2	61.5: 38.5	0.025	2.227 (1.121-4.425)[fb]
	↑ (NO_X + PM_{10}) E + MSDP	**↓ (NO_X + PM_{10}) E + NMSDP**		
Ever wheeze	14/44 (31.8)	20/137 (14.6)	0.015	2.730 (1.236-6.028)
Obesity	9/37 (24.3)	10/125 (8.0)	0.016	3.696 (1.372-9.955)
M: F ratio (%)	38.3:61.7	56.8:43.2	0.031	2.144 (1.085-4.132)[fb]
Bronchitis	7/46 (15.2)	10/151 (6.6)	0.078	2.531 (0.904-7.081)
	↑ (NO_2 + PM_{10}) C + MSDP	**↓ (NO2 + PM_{10}) C + NMSDP**		
Preterm birth	19/154 (12.3)	6/14 (42.9)	0.008	0.188 (0.059-0.600)
	↑ (NO_X + PM_{10}) E + PSDP	**↓ (NO_X + PM_{10}) E + NPSDP**		
Ever wheeze	14/46 (30.4)	14/92 (15.2)	0.045	2.438 (1.045-5.688)
Eczema	4/21 (19.0)	17/34 (50.0)	0.026	0.235 (0.065-0.846)
Croup	1/48 (2.1)	14/104 (13.5)	0.038	0.137 (0.017-1.072)
Bronchitis	7/48 (14.6)	4/103 (3.9)	0.037	4.226 (1.173-15.217)
Obesity	7/45	5/91 (5.5)	0.062	3.168 (0.945-10.620)
	↑ (NO_2 + PM_{10}) C + PSDP	**↓ (NO2 + PM_{10}) C + NPSDP**		
Preterm birth	27/169 (16.0)	4/10 (40.0)	0.073	0.285 (0.075-1.079)
	↑ (NO_X + PM_{10}) E + HSDP	**↓ (NO_X + PM_{10}) E + NHSDP**		
Croup	2/67 (3.0)	17/107 (15.9)	0.011	0.163 (0.036-0.730)
Hay fever	7/29 (24.1)	17/33 (51.5)	0.037	0.299 (0.101-0.891)
Obesity	12/52 (23.1)	8/91 (8.8)	0.034	3.133 (1.179-8.218)
Ever wheeze	19/64 (29.7)	16/100 (16.0)	0.043	2.217 (1.040-4.727)
Breathlessness	7/64 (10.9)	3/100 (3.0)	0.052	3.971 (0.988-15.966)
M:F ratio	45.6 :54.4	58.8 : 41.2	0.093	1.701 (0.929-3.115)
	↑ (NO_2 + PM_{10}) C + HSDP	**↓ (NO_2 + PM_{10}) C + NHSDP**		
Preterm birth	40/264 (15.2)	5/13 (38.5)	0.042	0.286 (0.089-0.918)
	↑ NO_X E + heavy maternal smoking	**↓ NO_X E + none maternal smoking**		
Ever wheeze	4/8 (50)	12/70 (17.1)	0.050	4.833 (1.088-22.075)
DDA	4/8 (50)	12/73 (16.4)	0.045	5.083 (1.114-23.192)
Obesity	3/8 (37.5)	7/59 (11.9)	0.091	4.452 (0.869-22.850)
	↑ NO_X E + heavy paternal smoking	**↓ NO_X E + none paternal smoking**		
LBW	4/8 (50.0)	6/67 (9.0)	0.009	10.167 (2.012-51.366)
Breathlessness	2/8 (25.0)	3/73 (4.1)	0.074	4.452 (0.869-22.850)

LBW	4/8 (50.0)	6/67 (9.0)	0.009	9.143 (0.859-97.265)
	↑ PM_{10} E + heavy paternal smoking	**↓ PM_{10} E + none paternal smoking**		
Breathlessness	2/4 (50.0)	10/157 (6.4)	0.028	14.200 (1.870-115.564)
Overweight	3/4 (75.0)	34/128 (26.6)	0.067	8.294 (0.834-82.972)
	↑ PM_{10} C + heavy maternal smokers	**↓ PM_{10} C + none maternal smoking**		
Obesity	4/11 (36.4)	1/17 (5.9)	0.062	9.143 (0.859-97.265)
	↑ PM_{10} C + heavy paternal smokers	**↓ PM_{10} C + none paternal smoking**		
Overweight	7/8 (87.5)	3/17 (17.6)	0.002	32.667 (2.852-374.155)
Obesity	3/8 (37.5)	0/17 (0)	0.024	Cannot be calculated
DDA	2/8 (1/2)	0/17 (0)	0.093	Cannot be calculated
	↑ (NO_X + PM_{10}) E + heavy maternal smokers	**↓ (NO_X + PM_{10}) E + none maternal smoking**		
Overweight	3/4 (75.0)	15/59 (25.4)	0.067	8.800 (0.895-91.155)
	↑ (NO_X + PM_{10}) E + heavy paternal smokers	**↓ (NO_X + PM_{10}) E + none paternal smoking**		
Breathlessness	2/4 (50.0)	3/73 (4.1)	0.019	23.37(2.398-227.045)
	↑ (NO_2 + PM_{10}) C + heavy paternal smokers	**↓ (NO_2 + PM_{10}) C + none paternal smoking**		
Overweight	7/8 (87.5)	1/5 (20.0)	0.032	28.00 (1.350-580.591)

Decreased odds ratios
Increased odds ratios
P<0.1 but >0.05
Exposure category headings
Odds ratio cannot be calculated

OR: Odds Ratio
M: F Male: Female
LBW: Low birthweight: Birthweight < 2.5kg)
DDA: Doctor diagnosed asthma
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
Preterm: Reported as preterm by mothers in questionnaire
C: Concentration
E: Emissions
fb-for female births
PM_{10}: Particulate matter <10 microns
High (↑) NOx emissions: NOx emissions >10 tonnes per annum; Low (↓) NOx emissions: NOx emissions ≤10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3; Low (↓) NO_2 C: NO_2 concentrations ≤ 17.07 microgram per m^3
High (↑) PM_{10} emissions: PM_{10} emissions >5 tonnes per annum; Low (↓) PM_{10} emissions: PM_{10} emissions ≤ 5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3; Low (↓) PM_{10} C: PM_{10} concentrations ≤ 13.91 microgram per m3
High (↑) NOx-PM_{10} emissions: NOx emissions >10 and PM_{10} emissions > 5 tonnes per annum;
Low (↓) NOx-PM_{10} emissions: NOx emissions ≤ 10 and PM_{10} emissions ≤ 5 tonnes per annum
High (↑) NO_2-PM_{10} concentration: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3;

Low (↓) NO_2-PM_{10} concentration: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration $\leq$ 13.91 microgram per m^3
MSDP: Maternal smoking during pregnancy; NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period; NPSDP: Fathers who did not smoke during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period; NHSDP: Household members who did not smoke during pregnancy period
High (↑) NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
Low (↓) NOx emissions + NPSDP: NOx emissions $\leq$ 10 (in tonnes per annum) and fathers who did not smoke during their partner's pregnancy period
High (↑) NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
Low (↓) NOx emissions + NMSDP: NOx emissions $\leq$10 (in tonnes per annum) and mothers who did not smoke during pregnancy
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
Low (↓) NOx emissions + NHSDP: NOx emissions $\leq$ 10 (in tonnes per annum) and household member who did not smoke during their partner's pregnancy period
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
Low (↓) PM_{10} emissions + NMSDP: PM_{10} emissions $\leq$5 (in tonnes per annum) and mothers who did not smoke during pregnancy
High (↑) PM_{10} emissions + PSDP: PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
Low (↓) PM_{10} emissions + NPSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy period
High (↑) PM_{10} emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
Low (↓) PM_{10} emissions + NHSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and household member who did not smoke during pregnancy period
High (↑) NO_2 concentration + PSDP: NO_2 concentration >17.01 microgram per m^3 and paternal smoking during pregnancy
Low (↓) NO_2 concentration + NPSDP: NO_2 concentration $\leq$ 17.01 microgram per m^3 and fathers who did not smoke during their partner's pregnancy period
High (↑) NO_2 concentration + MSDP: NO_2 concentration >17.01 microgram per m^3 and maternal smoking during pregnancy
Low (↓) NO_2 concentration + NMSDP: NO_2 concentration < 17.01 microgram per m^3 and mothers who did not smoke during pregnancy
High (↑) NO_2 concentration + HSDP: NO_2 concentration >17.01 microgram per m^3 and household member smoking during pregnancy
Low (↓) NO_2 concentration + NHSDP: NO_2 concentration $\leq$ 17.01 microgram per m^3 and household member who did not smoke during their partner's pregnancy period
High (↑) PM_{10} concentration + MSDP: PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
Low (↓) PM_{10} concentration + NMSDP: PM_{10} concentration $\leq$ 13.91 microgram per m^3 and mothers who did not smoke during pregnancy
High (↑) PM_{10} concentration + PSDP: PM_{10} concentration >13.91 microgram per m^3 and paternal smoking during pregnancy
Low (↓) PM_{10} concentration + NPSDP: PM_{10} concentration $\leq$ 13.91 microgram per m^3 and fathers who did not smoke during pregnancy period
High (↑) PM_{10} concentration + HSDP: PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy
Low (↓) PM_{10} concentration + NHSDP: PM_{10} concentration $\leq$ 13.91 microgram per m^3 and household member who did not smoke during pregnancy period
High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
Low (↓) NOx-PM_{10} emissions + NMSDP: NOx emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and mothers who did not smoke during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
Low (↓) NOx-PM_{10} emissions + NPSDP: NOx emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
Low (↓) NOx-PM_{10} emissions + NHSDP: NO_X emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and household members who did not smoke during pregnancy
High (↑) NO_2-PM_{10} concentration and MSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
Low (↓) NO_2-PM_{10} concentration and NMSDP: NO_2 concentration $\leq$ 17.01 & PM_{10} concentration $\leq$ 13.91 microgram per m^3 and mothers who did not smoke during pregnancy
High (↑) NO_2-PM_{10} concentration and PSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and paternal smoking during pregnancy
Low (↓) NO_2-PM_{10} concentration and NPSDP: NO_2 concentration $\leq$ 17.01 & PM_{10} concentration $\leq$ 13.91 microgram per m^3 and fathers who did not smoke during pregnancy
High (↑) NO_2-PM_{10} concentration and HSDP: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy
Low (↓) NO_2-PM_{10} concentration and NHSDP: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration $\leq$ 13.91 microgram per m^3 and household member who did not smoke during pregnancy
High (↑) NOx emissions and heavy maternal smoking during pregnancy: NOx emissions >10 and > 10 cigarettes smoked by mother during pregnancy
Low (↓) NOx emissions and none maternal smoking during pregnancy: NOx emissions $\leq$10 and mothers who never smoked at any time during pregnancy
High (↑) NOx emissions and heavy paternal smoking during pregnancy: NOx emissions >10 and > 10 cigarettes smoked by father during pregnancy
Low (↓) NOx emissions and none paternal smoking during pregnancy: NOx emissions $\leq$10 and fathers who never smoked at any time during pregnancy
High (↑) PM_{10} emissions and heavy paternal smoking: PM_{10} emissions >5 and >10 cigarettes smoked by father during pregnancy period
Low (↓) PM_{10} emissions and none paternal smoking: PM_{10} emissions $\leq$5 and fathers who never smoked at any time during pregnancy
High (↑) PM_{10} concentrations and heavy paternal smoking during pregnancy: PM_{10} concentrations > 13.91 and > 10 cigarettes smoked by father during pregnancy period
Low (↓) PM_{10} concentrations and none paternal smoking during pregnancy: PM_{10} concentrations $\leq$ 13.91 and fathers who never smoked at any time during pregnancy period
High (↑) NOx-PM_{10} emissions and heavy paternal smoking during pregnancy: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and > 10 cigarettes smoked by father during pregnancy period

Low (↓) NOx-PM_{10} emissions and paternal none smoking during pregnancy: NO_X emissions $\leq$ 10 + PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy.
High (↑) NO_2-PM_{10} concentration and heavy paternal smoking during pregnancy: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3 and > 10 cigarettes smoked by father during pregnancy
Low (↓) NO_2-PM_{10} concentration and none paternal smoker during pregnancy: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration $\leq$ 13.91 microgram per m^3 and fathers who did not smoke during pregnancy

In summary, the following statistically significant ($p<0.10$) associations were observed:

- *High NOx emissions*: decreased odds of hay fever and croup.
- *High NO_2 concentrations*: decreased odds of preterm birth.
- *High PM_{10} emissions*: increased odds of ever wheeze and breathlessness; decreased odds of croup.
- *High PM_{10} concentrations*: increased odds of obesity; decreased odds of being born LBW.
- *High NO_x + high PM_{10} emissions*: increased odds ever wheeze and of female birth; decreased odds of croup and hay fever.
- *High NO_2 + high PM_{10} concentrations*: decreased odds of preterm birth.
- *High NOx emissions + MSDP*: increased odds of obesity, ever wheeze and female birth; decreased odds of croup and hay fever.
- *High NOx emissions + PSDP*: increased odds of ever wheeze, obesity and bronchitis; decreased odds of eczema and croup.
- *High NOx emissions + HSDP*: increased odds of female births, obesity, ever wheeze and breathlessness; decreased odds of croup, hay fever and eczema.
- *High PM_{10} emissions + MSDP*: increased odds of childhood obesity, ever wheeze, bronchitis and female birth;
- *High PM_{10} emissions + PSDP*: increased odds of ever wheeze and bronchitis; decreased odds of croup.
- *High PM_{10} emissions + HSDP*: increased odds of ever wheeze, obesity, breathlessness and bronchitis; decreased odds of croup.
- *High NO_2 concentrations + MSDP or PSDP or HSDP*: decreased odds of preterm birth.
- *High PM_{10} concentrations + MSDP*: increased odds of childhood obesity and female birth;
- *High PM_{10} concentrations + PSDP*: increased odds of obesity; decreased odds of being born LBW, hay fever and eczema.
- *High PM_{10} concentrations + HSDP*: increased odds of obesity and female birth.
- *High NO_x and PM_{10} emissions + MSDP*: increased odds of ever wheeze, obesity, female birth and bronchitis; decreased odds of croup.
- *High NO_x and PM_{10} concentrations + MSDP*: decreased odd of preterm birth.
- *High NO_x and PM_{10} emissions + PSDP*: increased odds of ever wheeze, obesity, and bronchitis; decreased odds of croup and eczema.

- *High NO_x and PM_{10} concentrations + PSDP*: decreased odd of preterm birth.
- *High NO_x and PM_{10} emissions + HSDP*: increased odds of ever wheeze, obesity, breathlessness and female birth; decreased odds of croup and hay fever.
- *High NO_x and PM_{10} concentrations + HSDP*: decreased odd of preterm birth.
- *High NO_x emissions + heavy MSDP*: increased odds of ever wheeze, doctor diagnosed asthma and obesity.
- *High PM_{10} emissions + heavy MSDP*: nil.
- *High NO_2 concentrations + heavy MSDP*: nil.
- *High PM_{10} concentrations + heavy MSDP*: increased odds of obesity.
- *High NO_x emissions + heavy PSDP*: increased odds of breathlessness and LBW.
- *High PM_{10} emissions + heavy PSDP*: increased odds of breathlessness and being overweight.
- *High NO_2 concentrations + heavy PSDP*: nil.
- *High PM_{10} concentrations + heavy PSDP*: increased odds of obesity, overweight and doctor diagnosed asthma.
- *High NO_x - PM_{10} emissions + heavy MSDP*: nil.
- *High NO_x - PM_{10} emissions + heavy HSDP*: increased odds of breathlessness and overweight.
- *High NO_x - PM_{10} concentrations + heavy MSDP*: nil.
- *High NO_x - PM_{10} concentrations + heavy PSDP*: increased odds of overweight

4.4 Interaction analysis between exposures and socio-economic variables

In the following analyses, the associations between the exposure (pollution) and selected important socio-economic measures are explored, in an attempt to gain a better understanding of the extent to which environment and pollution were potentially inter-related in the study area.

4.4.1 Interaction analysis for NO_x emissions categories

Table 4.47 Interaction analysis for NO_x emissions categories

Variable	↑ NOx E (%)	↓ NOx E (%)	p value	OR (95% CI)
Upper quartile	**361/380 (95.0)**	**155/177 (87.6)**	**0.003**	**2.697 (1.419-5.125)**
MSDP	**112/436 (25.7)**	**46/201 (22.9)**	**<0.001**	**1.165 (0.786-1.726)**
High NO_2 C	**451/458 (98.5)**	**197/210 (93.8)**	**0.002**	**4.252 (1.671-10.819)**
High PM_{10} C	**443/458 (96.7)**	**167/210 (79.5)**	**<0.001**	**7.604 (4.113-14.053)**
High PM_{10} E	**142/158 (31.0)**	**0/210 (0)**	**<0.001**	**Cannot be calculated**
Breast feeding	**145/435 (33.3)**	**85/202 (42.1)**	**0.034**	**0.688 (0.488-0.970)**
PSDP	119/344 (34.6)	59/66 (35.5)	0.843	0.959 (0.651-1.414)
HSDP	183/434 (42.2)	87/201 (43.3)	0.796	0.955 (0.681-1.339)
Single parent	149/421 (35.4)	68/176 (38.6)	0.457	0.870 (0.605-1.251)
Maternal heavy smokers	8/150 (5.3)	8/82 (9.8)	0.278	0.521 (0.188-1.444)
Paternal heavy smokers	8/148 (5.4)	6/81 (7.4)	0.572	0.714 (0.239-2.135)

MSDP: Mothers who smoked during pregnancy
HSDP: Household members who smoked during pregnancy
PSDP: Fathers who did not smoke during pregnancy period
OR: Odds Ratio
Upper quartile: (Townsend score > 4)
NO_2: Nitrogen dioxide
NOx: Oxides of Nitrogen
PM_{10}: Particulate matter <10 microns
High (↑) NOx E: NOx emissions >10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
High (↑) PM_{10} E: PM_{10} emissions >5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3
C: Concentrations
E: Emissions
Maternal heavy smokers during pregnancy: > 10 cigarettes smoked by mother during pregnancy period
Paternal heavy smokers during pregnancy: > 10 cigarettes smoked by father during pregnancy period

In this analysis, respondents with exposure to high NO_x emission levels were compared to those with exposure to low NO_x exposure levels. High levels of NO_x emissions were associated with increased odds of lower socio-economic status, maternal smoking during pregnancy, high NO_2 concentrations, high PM_{10} concentrations and emissions, and decreased odds of breast feeding.

4.4.2 Interaction analysis for PM_{10} emissions categories

Table 4.48 Interaction analysis for PM_{10} emissions categories

Variable	↑ PM_{10} E (%)	↓ PM_{10} E (%)	p value	OR (95% CI)
Upper quartile	**123/124 (99.2)**	**393/433 (90.8)**	**0.001**	**12.519 (1.703 -92.008)**
MSDP	**47/138 (34.1)**	**111/499 (22.2)**	**0.005**	**1.805 (1.198-2.722)**
PSDP	**49/104 (47.1)**	**129/406 (31.8)**	**0.004**	**1.913 (1.234-2.965)**
High PM_{10} C	**141/142 (99.3)**	**469/526 (89.2)**	**<0.001**	**17.136 (2.352-124.865)**
High NO_x E	**142/142 (100.0)**	**316/526 (60.1)**	**<0.001**	**Cannot be calculated**
HSDP	**68/135 (50.4)**	**202/500 (40.4)**	**0.040**	**1.497 (1.022-2.193)**
High NO_2 C	139/142 (97.9)	509/526 (96.8)	0.591	1.547 (0.447-5.356)
Breast feeding	41/135 (30.4)	189/502 (37.6)	0.130	0.722 (0.480-1.087)
Single parent	41/130 (36.2)	170/467 (36.4)	1.000	0.989 (0.660-1.482)
Maternal heavy smokers	2/47 (4.3)	14/185 (7.6)	0.537	0.543 (0.119-2.476)
Paternal heavy smokers	4/52 (7.7)	10/177 (5.6)	0.527	1.392 (0.418-4.635)

MSDP: Mothers who smoked during pregnancy
HSDP: Household members who smoked during pregnancy
PSDP: Fathers who did not smoke during pregnancy period
OR: Odds Ratio
Upper quartile: (Townsend score > 4)
NO_2: Nitrogen dioxide
NOx: Oxides of Nitrogen
PM_{10}: Particulate matter <10 microns
High (↑) NOx E: NOx emissions >10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
High (↑) PM_{10} E: PM_{10} emissions >5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3
C: Concentrations
E: Emissions
Maternal heavy smokers during pregnancy: > 10 cigarettes smoked by mother during pregnancy period
Paternal heavy smokers during pregnancy: > 10 cigarettes smoked by father during pregnancy period

In this analysis, respondents with exposure to high PM_{10} emission levels were compared to those with exposure to low PM_{10} exposure levels. High levels of PM_{10} emissions were associated with increased odds of lower socio-economic status, maternal / paternal / household member smoking during pregnancy, high NO_x emissions and high PM_{10} concentrations.

4.4.3 Interaction analysis for NO_2 concentrations categories

Table 4.49 Interaction analysis for NO_2 concentrations categories

Variable	↑ NO_2 C (%)	↓ NO_2 C (%)	p value	OR (95% CI)
Upper quartile	**551/576 (95.7)**	**0/16 (0)**	**<0.001**	**Cannot be calculated**
High PM_{10} C	**646/686 (94.2)**	**2/20 (10.0)**	**<0.001**	**145.350 (32.583-648.403)**
High NOx E	**451/648 (69.6)**	**7/20 (35.0)**	**0.002**	**4.252 (1.671-10.819)**
Heavy PSDP	**13/235 (5.8)**	**3/10 (30.0)**	**0.021**	**0.137 (0.032-0.590)**
MSDP	169/652 (25.9)	3/19 (15.8)	0.429	1.866 (0.537-6.484)
PSDP	191/527 (36.2)	3/14 (21.4)	0.398	2.084 (0.537-6.484)
HSDP	*291/652 (44.6)*	*4/19 (21.1)*	*0.058*	*3.023 (0.993-9.206)*
High PM_{10} E	139/648 (21.5)	3/20 (15.0)	0.591	1.547 (0.447-5.356)
Breast feeding	240/654 (36.7)	8/19 (42.1)	0.636	0.797 (0.316-2.009)
Single parent	220/614 (35.8)	7/15 (46.7)	0.421	0.638 (0.228-1.783)
Maternal heavy smokers	15/241 (6.2)	1/9 (11.1)	0.454	0.531 (0.062-4.529)

MSDP: Mothers who smoked during pregnancy
HSDP: Household members who smoked during pregnancy
PSDP: Fathers who did not smoke during pregnancy period
OR: Odds Ratio
Upper quartile: (Townsend score > 4)
NO_2: Nitrogen dioxide
NOx: Oxides of Nitrogen
PM_{10}: Particulate matter <10 microns
High (↑) NOx E: NOx emissions >10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
High (↑) PM_{10} E: PM_{10} emissions >5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3
C: Concentrations
E: Emissions
Maternal heavy smokers during pregnancy: > 10 cigarettes smoked by mother during pregnancy period
Paternal heavy smokers during pregnancy: > 10 cigarettes smoked by father during pregnancy period

In this analysis, respondents with exposure to high NO_2 concentration levels were compared to those with exposure to low NO_2 concentration levels. High levels of NO_2 concentrations were associated with increased odds of lower socio-economic status, high PM_{10} concentrations, high NO_x emissions, and household member smoking during pregnancy, but decreased odds of heavy paternal smoking during pregnancy.

4.4.4 Interaction analysis for PM_{10} concentrations categories

Table 4.50 Interaction analysis for PM_{10} concentrations categories

Exposure	↑ PM_{10} C (%)	↓ PM_{10} C (%)	p value	OR (95% CI)
Upper quartile	**533/544 (98.0)**	**18/48 (37.5)**	**<0.001**	**80.758 (35.025-186.206)**
High NOx E	**443/610 (72.6)**	**15/58 (25.9)**	**<0.001**	**7.604 (4.115-14.053)**
High NO_2 C	**646/648 (99.7)**	**40/58 (69.0)**	**<0.001**	**145.35 (32.58-648.40)**
High PM10 E	**141/ 610 (23.1)**	**1/58 (1.7)**	**<0.001**	**17.136 (2.352-124.865)**
Breast feeding	**213/617 (34.5)**	**35/56 (62.5)**	**<0.001**	**0.316 (0.180-0.557)**
Paternal heavy smokers	**9/220 (4.1)**	**7/25 (28.0)**	**<0.001**	**0.110 (0.037-0.329)**
HSDP	**280/617 (45.4)**	**15/54 (27.8)**	**0.015**	**2.160 (1.167-4.001)**
MSDP	164/615 (26.7)	8/56 (14.3)	0.054	2.182 (1.011-4.720)
PSDP	183/494 (37.0)	11/47 (23.4)	0.079	1.926 (0.957-3.876)
Single parent	202/571 (35.0)	25/52 (48.1)	0.070	0.582 (0.329-1.029)
Maternal heavy smokers	14/227 (6.2)	2/23 (8.7)	0.648	0.690 (0.147-3.245)

MSDP: Mothers who smoked during pregnancy
HSDP: Household members who smoked during pregnancy
PSDP: Fathers who did not smoke during pregnancy period
OR: Odds Ratio
Upper quartile: (Townsend score > 4)
NO_2: Nitrogen dioxide
NOx: Oxides of Nitrogen
PM_{10}: Particulate matter <10 microns
High (↑) NOx E: NOx emissions >10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
High (↑) PM_{10} E: PM_{10} emissions >5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3
C: Concentrations
E: Emissions
Maternal heavy smokers during pregnancy: > 10 cigarettes smoked by mother during pregnancy period
Paternal heavy smokers during pregnancy: > 10 cigarettes smoked by father during pregnancy period

In this analysis, respondents with exposure to high PM_{10} concentration levels were compared to those with exposure to low PM_{10} concentration levels. High levels of PM_{10} concentrations were associated with increased odds of lower socio-economic status, high NO_x emissions, high NO_2 concentrations, high PM_{10} emissions and maternal / paternal / household member smoking during pregnancy, but decreased odds of breast feeding, being a single parent and heavy paternal smoking during pregnancy.

4.4.5 Interaction analysis for socio-economic categories

Table 4.51 Interaction analysis for socio-economic categories*

Exposure category*	Upper quartile (%)	Lower quartile (%)	p value	OR (95% CI)
HSDP	**251/264 (95.1)**	**279/314 (88.9)**	**0.009**	**2.422 (1.253-4.682)**
High NO_2 C	**551/576 (95.7)**	**0/16 (0)**	**<0.001**	**Cannot be calculated**
High PM_{10} E	**123/124 (99.2)**	**393/433 (90.8)**	**0.001**	**12.519 (1.702-92.008)**
High NOx E	**361/380 (95.0)**	**155/177 (87.6)**	**0.003**	**2.697 (1.419-5.125)**
High PM_{10} C	**533/544 (98.0)**	**18/48 (37.5)**	**<0.001**	**80.758 (35.025-186.206)**
MSDP	**154/160 (96.3)**	**374/417 (89.7)**	**0.012**	**2.951 (1.231-7.016)**
Paternal heavy smokers	**10/14 (71.4)**	**192/207 (92.8)**	**0.023**	**0.195 (0.055-0.698)**
Breast feeding	**182/206 (88.3)**	**346/370 (93.5)**	**0.040**	**0.526 (0.281-0.952)**
PSDP	163/174 (93.7)	266/297 (89.6)	0.179	1.727 (0.845-3.530)
Single parent	179/197 (90.9)	320/345 (92.8)	0.509	0.777 (0.413-1.463)
Maternal heavy smokers	13/14 (92.9)	193/209 (92.3)	1.000	1.078 (0.132-8.774)

MSDP: Mothers who smoked during pregnancy
HSDP: Household members who smoked during pregnancy
PSDP Fathers who did not smoke during pregnancy period
OR: Odds Ratio
Upper quartile: (Townsend score > 4)
Lower quartile: (Townsend score <-4)
NO_2: Nitrogen dioxide
NOx: Oxides of Nitrogen
PM_{10}: Particulate matter <10 microns
High (↑) NOx emissions: NOx emissions >10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
High (↑) PM_{10} emissions: PM_{10} emissions >5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per $m^{3;}$
C: Concentrations
E: Emissions
Maternal heavy smokers during pregnancy: > 10 cigarettes smoked by mother during pregnancy period
Paternal heavy smokers during pregnancy: > 10 cigarettes smoked by father during pregnancy period
*Breast feeding and single parent also included

In this analysis, respondents in the lowest SES quartile were compared to those in the highest SES quartile. Being in the lowest SES quartile was associated with increased odds of maternal and/or household member smoking during pregnancy, high NO_2 concentrations, high PM_{10} emissions, high NO_x emissions and high PM_{10} concentrations, but decreased odds of heavy paternal smoking during pregnancy and breast feeding.

4.5 Comparison of means

4.5.1 Comparing mean NO_x and PM_{10} emissions in areas with change in health outcomes

Table 4.52 Comparing mean NO_x and PM_{10} emissions in relation to areas with change in health outcomes

Variable	Outcome category	Mean NOx E Mean ± SD	P value	Mean PM_{10} E Mean ± SD	P value
Childhood obesity	Yes	1.73 ± 1.27	0.145	0.09 ± 0.10	0.941
	No	1.51 ± 1.16		0.09 ± 0.12	
Childhood overweight	Yes	1.50 ± 0.17	0.623	0.08 ± 0.11	0.356
	No	1.55 ± 1.18		0.09 ± 0.13	
DDA	Yes	1.52 ± 1.18	0.991	0.10 ± 0.13	0.521
	No	1.53 ± 1.16		0.09 ± 0.12	
MSDP	Yes	1.83 ± 1.28	**0.001**	0.12 ± 0.14	**0.003**
	No	1.47 ± 1.15		0.08 ± 0.12	
PSDP	Yes	1.74 ± 1.27	**0.001**	0.11 ± 0.14	**0.011**
	No	1.74 ± 1.09		0.08 ± 0.12	
HSDP	Yes	1.77 ± 1.24	**0.001**	0.11 ± 0.14	**0.000**
	No	1.41 ± 1.16		0.07 ± 0.10	
Preterm status	Yes	1.36 ± 1.06	0.078	0.08 ± 0.11	0.314
	No	1.58 ± 1.21		0.09 ± 0.11	
LBW	Yes	1.50 ± 1.12	0.843	0.11 ± 0.16	0.265
	No	1.50 ± 1.18		0.09 ± 0.12	
Upper quartile	Yes	1.63 ± 1.21	**<0.001**	0.10 ± 0.13	**0.000**
	No	0.83 ± 0.39		0.03 ± 0.02	
Bronchitis	Yes	1.81 ± 1.33	**0.014**	0.13 ± 0.15	**0.022**
	No	1.53 ± 1.17		0.09 ± 0.12	
Croup	Yes	1.07 ± 0.81	**0.001**	0.07 ± 0.13	0.266
	No	1.60 ± 1.21		0.09 ± 0.12	
Allergy	Yes	1.45 ± 1.21	0.323	0.08 ± 0.11	0.339
	No	1.57 ± 1.17		0.09 ± 0.12	
Hay fever	Yes	1.44 ± 1.21	0.249	0.09 ± 0.12	0.420
	No	1.63 ± 1.23		0.10 ± 0.13	
Eczema	Yes	1.61 ± 1.25	0.963	0.09 ± 0.11	0.291
	No	1.60 ± 1.22		0.10 ± 0.14	
Excess cough last 12 months	Yes	1.59 ± 1.22	0.693	0.11 ± 0.15	0.099
	No	1.54 ± 1.18		0.09 ± 0.12	
Breathlessness	Yes	1.88 ± 1.38	0.057	0.12 ± 0.14	0.148
	No	1.53 ± 1.17		0.09 ± 0.12	
Ever wheeze	Yes	1.77 ± 1.33	**0.027**	0.12 ± 0.14	**0.011**
	No	1.50 ± 1.15		0.09 ± 0.12	
Breast feeding	Yes	1.47 ± 1.09	0.223	0.09 ± 0.13	0.874
	No	1.58 ± 1.23		0.09 ± 0.11	
ADHD	Yes	1.43 ± 1.24	0.684	0.07 ± 0.01	0.383
	No	1.55 ± 1.18		0.09 ± 0.13	
IUGR	Yes	1.31 ± 1.05	0.279	0.70 ± 0.11	0.328
	No	1.57 ± 1.21		0.10 ± 0.13	
Gender	Male	2.29 ± 0.76	0.383	1.17 ± 0.54	0.215
	Female	2.23 ± 0.81		1.13 ± 0.47	
Stunting	Yes	1.48 ± 1.18	0.818	0.10 ± 0.14	0.821
	No	1.54 ± 1.17		0.09 ± 0.13	

ADHD: Attention Deficit Hyperactivity Disorder
LBW: Low birthweight: Birthweight < 2.5kg
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Average NOx emissions in tonnes per annum
Average PM_{10} emissions in tonnes per annum
MSDP: Maternal smoking during pregnancy
PSDP: Paternal smoking during pregnancy
HSDP: Household member smoking during pregnancy
DDA: Doctor diagnosed asthma
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
Preterm: Reported as preterm by mothers in questionnaire
Upper quartile: (Townsend score > 4)

In this analysis, the mean NO_x and PM_{10} emissions were compared between areas with and without birth and child health outcomes/exposures/breast feeding/Upper quartile. Mean NO_x emissions and PM_{10} emissions were higher in areas with increased prevalence of maternal smoking during pregnancy, paternal smoking during pregnancy and household smoking compared to areas with non-smoking household members. The mean NO_x and PM_{10} emissions were higher in areas with lower socio economic status, and with increased prevalence of bronchitis, ever wheeze, and lower in areas with decreased prevalence of croup.

4.5.2 Comparing mean NO_2 and PM_{10} concentrations in relation to areas with change in health outcomes

Table 4.53 Comparing mean NO_2 and PM_{10} concentrations in relation to areas with change in health outcomes

Variable		Mean NO_2 C Mean ± SD	P value	Mean PM_{10} C Mean ± SD	P value
Childhood obesity	Yes	22.00 ± 1.65	**0.009**	15.61 ± 0.71	**0.043**
	No	21.24 ± 2.38		15.33 ± 1.18	
Childhood overweight	Yes	21.52 ± 2.05	0.240	15.44 ± 0.86	0.284
	No	21.27 ± 2.42		15.32 ± 1.18	
Doctor diagnosed asthma	Yes	21.37 ± 2.11	0.755	15.41 ± 0.84	0.552
	No	21.29 ± 2.34		15.34 ± 1.10	
Maternal smoking during pregnancy	Yes	21.69 ± 1.91	**0.010**	15.47 ± 0.80	0.088
	No	21.16 ± 2.43		15.31 ± 1.14	
Paternal smoking during pregnancy	Yes	21.49 ± 1.91	0.097	15.40 ± 0.79	0.249
	No	21.14 ± 2.58		15.29 ± 1.25	
Any household smoking during pregnancy	Yes	21.56 ± 1.95	**0.016**	15.43 ± 0.80	0.114
	No	21.12 ± 2.54		15.30 ± 1.27	
Preterm status	Yes	21.25 ± 2.62	0.825	15.42 ± 1.02	0.468
	No	21.31 ± 2.29		15.34 ± 1.09	
LBW	Yes	20.95 ± 2.38	0.227	15.18 ± 0.98	0.179
	No	21.35 ± 2.29		15.39 ± 1.06	
Upper quartile	Yes	21.67 ± 1.86	<**0.001**	15.52 ± 0.77	<**0.001**
	No	17.39 ± 1.56		13.69 ± 0.42	
Bronchitis	Yes	21.44 ± 2.03	0.718	15.37 ± 0.85	0.928
	No	21.32 ± 2.18		15.38 ± 0.89	
Croup	Yes	20.91 ± 2.56	0.087	15.37 ± 0.95	0.857
	No	21.40 ± 2.10		15.39 ± 0.87	
Allergy	Yes	21.14 ± 2.12	0.432	15.35 ± 0.83	0.919
	No	21.33 ± 2.42		15.34 ± 1.15	
Hay fever	Yes	21.11 ± 2.08	0.230	15.37 ± 0.81	0.527
	No	21.45 ± 2.19		15.44 ± 0.85	
Eczema	Yes	21.24 ± 2.18	0.335	15.33 ± 0.87	0.207
	No	21.50 ± 2.08		15.47 ± 0.81	
Excess cough last 12 months	Yes	21.28 ± 2.13	0.856	15.35 ± 0.86	0.913
	No	21.32 ± 2.36		15.36 ± 1.11	
Breathlessness	Yes	21.72 ± 1.74	0.224	15.49 ± 0.75	0.391
	No	21.28 ± 2.35		15.35 ± 1.09	
Ever wheeze	Yes	21.29 ± 2.01	0.924	15.37 ± 0.82	0.916
	No	21.32 ± 2.39		15.36 ± 1.12	
Breast feeding	Yes	20.91 ± 2.31	**0.001**	15.15 ± 0.98	<**0.001**
	No	21.54 ± 2.29		15.47 ± 1.09	
ADHD	Yes	22.09 ± 1.83	0.133	15.75 ± 0.69	0.113
	No	21.26 ± 2.32		15.34 ± 1.07	
IUGR	Yes	21.06 ± 2.17	0.585	15.31 ± 0.85	0.773
	No	21.32 ± 2.43		15.37 ± 1.10	
Gender	Male	21.08 ± 2.43	0.585	15.26 ± 1.22	**0.018**
	Female	21.50 ± 2.19		15.45 ± 0.87	
Stunting	Yes	21.53 ± 1.99	0.639	15.44 ± 0.82	0.707
	No	21.32 ± 2.29		15.38 ± 1.08	

ADHD: Attention Deficit Hyperactivity Disorder
LBW: Low birthweight: Birthweight < 2.5kg
IUGR: Intrauterine growth restriction: Term baby and less than 2.5kgs
Average NOx emissions in tonnes per annum
Average PM_{10} emissions in tonnes per annum
MSDP: Maternal smoking during pregnancy
PSDP: Paternal smoking during pregnancy
HSDP: Household member smoking during pregnancy
DDA: Doctor diagnosed asthma
Stunting: Child height for age z-score less than −2 standard deviations
Obesity: BMI z-score >1.64 (>95th centile)
Overweight: BMI z-score >1.04 (>85th centile)
CWB triad: Symptom triad of cough, wheeze and breathlessness
Preterm: Reported as preterm by mothers in questionnaire
Upper quartile: (Townsend score > 4)

In this analysis, the mean NO_2 concentrations and PM_{10} concentrations were compared between areas with and without birth and child health outcomes/exposures/breast feeding/Upper quartile. Mean NO_x concentrations and PM_{10} concentrations were higher in areas with increased prevalence of childhood obesity and lower socio economic status and lower in areas with increased breast feeding. There was increased mean NO_2 concentrations in relation to maternal smoking during pregnancy and household member smoking during pregnancy and there was increased mean PM_{10} concentrations in areas with more female births.

4.6 Comparison of growth parameters

4.6.1 Comparison of growth parameters in relation to high and low NO_x and PM_{10} emissions categories

Table 4.54 Growth parameters in relation to high and low NO_x and PM_{10} emissions

Growth parameter	Category for NOx E	Mean ± SD	P value	Category for PM_{10} E	Mean ± SD	P value
Child weight	High	28.23 ± 8.76	0.907	High	27.61 ± 8.68	0.401
	Low	28.14 ± 8.78		Low	28.36 ± 8.79	
Child height	High	125.82 ± 14.51	0.617	High	124.42 ± 15.38	0.287
	Low	125.19 ± 13.52		Low	125.94 ± 13.86	
Birthweight	High	3.21 ± 0.62	0.706	High	3.25 ± 0.60	0.529
	Low	3.24 ± 0.69		Low	3.21 ± 0.65	
Body mass index	High	17.46 ± 3.63	0.472	High	17.62 ± 4.68	0.830
	Low	17.73 ± 4.71		Low	17.53 ± 3.82	
Weight for age z score	High	0.62 ± 1.10	0.702	High	0.77 ± 1.08	0.111
	Low	0.66 ± 1.07		Low	0.59 ± 1.09	
Height for age z score	High	0.46 ± 1.63	0.180	High	0.64 ± 1.61	0.356
	Low	0.66 ± 1.47		Low	0.49 ± 1.58	
BMI z score	High	0.37 ± 1.56	0.386	High	0.36 ± 1.78	0.702
	Low	0.49 ± 1.25		Low	0.42 ± 1.38	

PM_{10}: Particulate matter <10 microns
High (↑) NOx emissions: NOx emissions >10 tonnes per annum
Low (↓) NOx emissions: NOx emissions ≤10 tonnes per annum
High (↑) PM_{10} emissions: PM_{10} emissions >5 tonnes per annum
Low (↓) PM_{10} emissions: PM_{10} emissions ≤ 5 tonnes per annum
BMI: Body Mass Index; SD: Standard deviation
E: Emissions

In this analysis growth parameters were compared in relation to high and low NO_x and PM_{10} emissions. There was variation in different growth parameters in relation to NO_x and PM_{10} emissions. No significant associations were identified.

4.6.2 Comparison of growth parameters in relation to high and low NO_2 and PM_{10} concentrations categories

Table 4.55 Growth parameters in relation to high and low NO_2 and PM_{10} concentrations

Growth parameter	Category for NO_2 C	Mean ± SD	P value	Category for PM_{10} C	Mean ± SD	P value
Child weight	High	28.31 ± 8.82	0.632	High	28.21 ± 8.89	0.492
	Low	27.28 ± 7.73		Low	29.08 ± 7.53	
Child height	High	125.70 ± 14.15	0.448	High	125.40 ± 14.25	0.184
	Low	123.07 ± 14.09		Low	128.13 ± 12.74	
Birthweight	High	3.23 ± 0.63	0.873	High	3.21 ± 0.63	0.093
	Low	3.20 ± 0.62		Low	3.73 ± 0.62	
Body mass index	High	17.56 ± 3.98	0.616	High	17.58 ± 4.07	0.573
	Low	17.05 ± 2.15		Low	17.25 ± 2.26	
Weight for age z score	High	0.625 ± 1.08	0.790	High	0.61 ± 1.07	0.192
	Low	0.702 ± 1.76		Low	0.83 ± 1.38	
Height for age z score	High	0.502 ± 1.54	0.358	High	0.50 ± 1.54	0.599
	Low	0.882 ± 2.44		Low	0.62 ± 1.97	
BMI z score	High	0.405 ± 1.47	0.737	High	0.38 ± 1.49	0.356
	Low	0.272 ± 0.83		Low	0.60 ± 1.00	

PM_{10}: Particulate matter <10 microns
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
Low (↓) NO_2 C: NO_2 concentrations $\leq$ 17.07 microgram per m^3
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3
Low (↓) PM_{10} C: PM_{10} concentrations $\leq$ 13.91 microgram per m^3
BMI: Body Mass Index; SD: Standard deviation
C: Concentrations

In this analysis growth parameters were compared in relation to high and low NO2 and PM10 concentrations. There was variation in different growth parameters in relation to NO_2 and PM_{10} concentrations. No significant associations were identified.

4.6.3 Comparison of growth parameters in relation to combined NO_X-PM_{10} emissions and combined NO_2-PM_{10} concentrations categories

Table 4.56 Growth parameters in relation to combined NO_X-PM_{10} emissions and combined NO_2-PM_{10} concentrations categories

Growth parameter	Category for combined NO_x-PM_{10} emissions	Mean ± SD	p value	Category for combined NO_2-PM_{10} concentrations	Mean ± SD	p value
Child weight	High	27.61 ± 8.68	0.401	High	28.22 ± 8.90	0.564
	Low	28.36 ± 8.80		Low	28.94 ± 7.52	
Child height	High	124.42 ± 15.38	0.287	High	125.45 ± 14.25	0.312
	Low	125.94 ± 13.86		Low	127.49 ± 12.94	
Birth weight	High	3.25 ± 0.60	0.529	High	3.21 ± 0.63	0.082
	Low	3.21 ± 0.65		Low	3.37 ± 0.61	
Body mass index	High	17.62 ± 4.67	0.830	High	17.58 ± 4.07	0.550
	Low	17.53 ± 3.82		Low	17.24 ± 2.24	
Weight for age z score	High	0.77 ± 1.08	0.111	High	0.67 ± 1.07	0.128
	Low	0.59 ± 1.09		Low	0.86 ± 1.36	
Height for age z score	High	0.64 ± 1.61	0.356	High	0.49 ± 1.55	0.554
	Low	0.49 ± 1.58		Low	0.64 ± 1.95	
BMI z score	High	0.36 ± 1.78	0.702	High	0.38 ± 0.49	0.399
	Low	0.42 ± 1.38		Low	0.58 ± 0.99	

High (↑) NOx-PM_{10} emissions: NOx emissions >10 and PM_{10} emissions > 5 tonnes per annum;
Low (↓) NOx-PM_{10} emissions: NOx emissions $\leq$ 10 and PM_{10} emissions $\leq$ 5 tonnes per annum
High (↑) NO_2-PM_{10} concentration: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3
Low (↓) NO_2-PM_{10} concentration: NO_2 concentration $\leq$ 17.01 and PM_{10} concentration $\leq$ 13.91 microgram per m^3
BMI: Body Mass Index; SD: Standard deviation

In this analysis growth parameters were compared in relation to combined high and low NOX-PM10 emissions and combined high and low NO2-PM10 concentrations. There was variation in different growth parameters in relation to combined NO_X-PM_{10} emissions and combined NO_2-PM_{10} concentrations. No significant association were identified.

4.6.4 Comparison of growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NOx emissions categories

Table 4.57 Growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NOx emissions categories

Growth parameter	Category*	MSDP+NOx emissions	P value	PSDP+NOx emissions	P value	HSDP+NOx emissions	P value
Child	Yes/High	27.60 ± 8.58	0.361	27.83 ± 8.32	0.555	28.68 ± 8.66	0.739
weight	No/Low	28.67 ± 8.83		28.54 ± 8.63		29.06 ± 9.16	
Child	Yes/High	124.53 ± 14.53	0.374	126.66 ± 15.13	0.845	126.36 ± 14.97	0.847
height	No/Low	126.17 ± 13.49		126.27 ± 13.48		126.39 ± 13.40	
Birth	Yes/High	3.131 ± 0.559	0.132	3.232 ± 0.647	0.101	3.197 ± 0.610	0.200
weight	No/Low	3.265 ± 0.722		3.395 ± 0.713		3.306 ± 0.710	
Body mass	Yes/High	17.86 ± 4.69	0.926	17.48 ± 4.42	0.569	17.81 ± 4.22	0.505
index	No/Low	17.92 ± 5.27		17.91 ± 5.88		18.26 ± 5.95	
Weight for	Yes/High	0.58 ± 1.09	0.467	0.684 ± 1.004	0.796	0.690 ± 1.086	0.466
age z-score	No/Low	0.47 ± 1.08		0.643 ± 1.150		0.586 ± 1.113	
Height for	Yes/High	0.41 ± 1.34	0.407	0.67 ± 1.40	0.865	0.51 ± 1.34	0.846
age z-score	No/Low	0.25 ± 1.45		0.63 ± 1.43		0.47 ± 1.61	
BMI z	Yes/High	0.38 ± 1.40	0.578	0.404 ± 1.24	0.794	0.549 ± 1.19	0.369
score	No/Low	0.46 ± 1.21		0.454 ± 1.21		0.364 ± 1.91	

MSDP: Maternal smoking during pregnancy
NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
NPSDP: Fathers who did not smoke during his partner's pregnancy period
BMI: Body Mass Index
*Smoking/Emissions category
High (↑) NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
Low (↓) NOx emissions + NPSDP: NOx emissions $\leq$ 10 (in tonnes per annum) and fathers who did not smoke during their partner's
pregnancy period
High (↑) NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
Low (↓) NOx emissions + NMSDP: NOx emissions $\leq$ 10 (in tonnes per annum) and mothers who did not smoke during pregnancy
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
Low (↓) NOx emissions + NHSDP: NOx emissions $\leq$ 10 (in tonnes per annum) and household member who did not smoke during their
partner's pregnancy period

In this analysis growth parameters were compared in relation to maternal, paternal and household smoking categories during pregnancy combined with NOx emissions data. There was variation in different growth parameters in relation to combined categories of NOx emissions with maternal, paternal and household smoking during pregnancy, but no significant associations were identified.

4.6.5 Comparison of growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with PM_{10} emissions categories

Table 4.58 Growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with PM_{10} emissions categories

Growth parameter	Category*	MSDP+PM_{10} emissions	P value	PSDP+PM_{10} emissions	P value	HSDP+PM_{10} emissions	P value
Child	Yes/High	28.44 ± 9.59	0.467	26.89 ± 8.82	0.266	28.44 ± 9.45	0.847
weight	No/Low	28.82 ± 9.03		28.51 ± 9.20		28.69 ± 9.26	
Child	Yes/High	123.26 ± 15.09	0.164	122.77 ± 15.33	0.193	124.28 ± 14.90	0.368
height	No/Low	126.47 ± 13.93		125.77 ± 14.29		126.06 ± 13.50	
Birth	Yes/High	3.15 ± 0.53	0.467	3.26 ± 0.63	0.889	3.28 ± 0.43	0.785
weight	No/Low	3.23 ± 0.68		3.27 ± 0.66		3.26 ± 0.64	
Body mass	Yes/High	18.61 ± 6.48	0.211	17.71 ± 5.81	0.965	18.36 ± 5.94	0.370
index	No/Low	17.64 ± 4.19		17.68 ± 4.58		17.69 ± 4.52	
Weight for	Yes/High	0.70 ± 1.17	0.437	0.740 ± 0.97	0.533	0.861 ± 1.13	0.105
age z score	No/Low	0.55 ± 1.13		0.616 ± 1.19		0.594 ± 1.13	
Height for	Yes/High	0.21 ± 1.51	0.546	0.770 ± 1.39	0.319	0.505 ± 1.43	0.765
age z score	No/Low	0.37 ± 1.54		0.507 ± 1.58		0.437 ± 1.58	
BMI z-	Yes/High	0.65 ± 1.17	0.287	0.507 ± 1.00	0.659	0.846 ± 1.06	0.847
score	No/Low	0.41 ± 1.31		0.404 ± 1.38		0.571 ± 1.63	

MSDP: Maternal smoking during pregnancy
NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
NPSDP: Fathers who did not smoke during his partner's pregnancy period
BMI: Body Mass Index
*Smoking/Emissions category
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
Low (↓) PM_{10} emissions + NMSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and mothers who did not smoke during pregnancy
High (↑) PM_{10} emissions + PSDP: PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
Low (↓) PM_{10} emissions + NPSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy period
High (↑) PM_{10} emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
Low (↓) PM_{10} emissions + NHSDP: PM_{10} emissions $\leq$ 5 (in tonnes per annum) and household member who did not smoke during pregnancy period

In this analysis growth parameters were compared in relation to maternal, paternal and household smoking categories during pregnancy combined with PM10 emissions data. There was variation in different growth parameters in relation to combined categories of PM_{10} emissions with maternal, paternal and household smoking during pregnancy, but no significant associations were identified.

4.6.6 Comparison of growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NO_2 concentrations categories

Table 4.59 Growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NO_2 concentrations categories

Growth parameter	Category*	MSDP+NO_2 concentrations	P value	PSDP+NO_2 concentrations	P value	HSDP+NO_2 concentrations	P value
Child	Yes/High	27.69 ± 8.66	0.904	27.69 ± 8.58	0.648	28.13 ± 8.41	0.479
weight	No/Low	27.41 ± 8.30		26.41 ± 9.60		26.44 ± 7.78	
Child	Yes/High	124.52 ± 14.10	0.491	125.88 ± 14.62	0.346	125.46 ± 14.41	0.355
height	No/Low	121.79 ± 15.02		121.11 ± 16.99		121.79 ± 15.02	
Birth	Yes/High	3.157 ± 0.57	0.507	3.24 ± 0.62	0.606	3.203 ± 0.62	0.703
weight	No/Low	3.27 ± 0.71		3.35 ± 0.65		3.273 ± 0.37	
Body mass	Yes/High	17.73 ± 4.04	0.739	17.41 ± 3.814	0.476	17.59 ± 3.70	0.814
index	No/Low	17.34 ± 2.24		16.49 ± 17.09		17.34 ± 2.24	
Weight for	Yes/High	0.72 ± 1.05	0.668	0.61 ± 1.03	0.905	0.692 ± 1.08	0.706
age z score	No/Low	0.59 ± 0.99		0.56 ± 1.19		0.576 ± 1.03	
Height for	Yes/High	0.59 ± 1.33	0.510	0.54 ± 1.37	0.741	0.511 ± 1.40	0.408
age z score	No/Low	0.84 ± 0.95		0.70 ± 0.95		0.850 ± 0.99	
BMI z-	Yes/High	0.44 ± 1.31	0.878	0.39 ± 1.27	0.437	0.517 ± 1.17	0.662
score	No/Low	0.38 ± 1.11		0.77 ± 0.79		0.359 ± 1.58	

MSDP: Maternal smoking during pregnancy
NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period;
NPSDP: Fathers who did not smoke during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
NHSDP: Household members who did not smoke during pregnancy period
High (↑) NO_2 concentration + PSDP: NO_2 concentration >17.01 microgram per m^3 and paternal smoking during pregnancy
Low (↓) NO_2 concentration + NPSDP: NO_2 concentration $\leq$ 17.01 microgram per m^3 and fathers who did not smoke during their partner's
pregnancy period
High (↑) NO_2 concentration + MSDP: NO_2 concentration >17.01 microgram per m^3 and maternal smoking during pregnancy
Low (↓) NO_2 concentration + NMSDP: NO_2 concentration < 17.01 microgram per m^3 and mothers who did not smoke during pregnancy
High (↑) NO_2 concentration + HSDP: NO_2 concentration >17.01 microgram per m^3 and household member smoking during pregnancy
Low (↓) NO_2 concentration + NHSDP: NO_2 concentration $\leq$ 17.01 microgram per m^3 and household member who did not smoke during their partner's pregnancy period
*Smoking/Concentrations category

In this analysis growth parameters were compared in relation to maternal, paternal and household smoking categories during pregnancy combined with NO_2 concentrations data. There was variation in different growth parameters in relation to combined categories of NO_2 concentrations data with maternal, paternal and household smoking during pregnancy, but no significant associations were identified.

4.6.7 Comparison of growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with PM_{10} concentrations categories

Table 4.60 Growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with PM_{10} concentrations categories

Growth parameter	Category*	MSDP+PM_{10} concentrations	P value	PSDP+PM_{10} concentrations	P value	HSDP+PM_{10} concentrations	P value
Child weight	Yes/High	27.72 ± 8.75	0.300	27.59 ± 8.59	0.276	28.10 ± 8.48	0.525
	No/Low	29.25 ± 7.79		29.34 ± 8.09		29.05 ± 7.81	
Child height	Yes/High	124.68 ± 14.19	0.084	125.72 ± 14.61	0.236	125.31 ± 14.39	0.355
	No/Low	128.90 ± 13.16		129.03 ± 13.28		127.69 ± 12.67	
Birth weight	Yes/High	3.16 ± 0.58	0.010	3.23 ± 0.63	0.071	3.19 ± 0.62	0.150
	No/Low	3.44 ± 0.66		3.46 ± 0.66		3.37 ± 0.69	
Body mass index	Yes/High	17.69 ± 4.09	0.347	17.37 ± 3.87	0.427	17.59 ± 3.69	0.655
	No/Low	17.06 ± 2.16		16.81 ± 2.01		17.29 ± 2.25	
Weight for age score	Yes/High	0.72 ± 1.11	0.410	0.61 ± 1.03	0.909	0.70 ± 1.08	0.900
	No/Low	0.56 ± 1.13		0.59 ± 1.02		0.68 ± 1.08	
Height for age z score	Yes/High	0.58 ± 1.34	0.633	0.53 ± 1.36	0.582	0.51 ± 1.37	0.932
	No/Low	0.46 ± 1.50		0.71 ± 1.42		0.53 ± 1.38	
BMI z score	Yes/High	0.45 ± 1.32	0.582	0.39 ± 1.27	0.401	0.53 ± 1.70	0.320
	No/Low	0.58 ± 1.01		0.61 ± 0.95		0.78 ± 0.93	

MSDP: Maternal smoking during pregnancy
NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
NPSDP: Fathers who did not smoke during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
NHSDP: Household members who did not smoke during pregnancy period
High (↑) PM_{10} concentration + MSDP: PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
Low (↓) PM_{10} concentration + NMSDP: PM_{10} concentration $\leq$ 13.91 microgram per m^3 and mothers who did not smoke during pregnancy
High (↑) PM_{10} concentration + PSDP: PM_{10} concentration >13.91 microgram per m^3 and paternal smoking during pregnancy
Low (↓) PM_{10} concentration + NPSDP: PM_{10} concentration $\leq$ 13.91 microgram per m^3 and fathers who did not smoke during pregnancy period
High (↑) PM_{10} concentration + HSDP: PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy
Low (↓) PM_{10} concentration + NHSDP: PM_{10} concentration $\leq$ 13.91 microgram per m^3 and household member who did not smoke during pregnancy period
BMI: Body Mass Index
*Smoking/Concentrations category

In this analysis growth parameters were compared in relation to maternal, paternal and household smoking categories during pregnancy combined with PM_{10} concentrations data. There was variation in different growth parameters in relation to combined categories of PM_{10} concentrations data with maternal, paternal and household smoking during pregnancy, but no significant associations were identified.

4.6.8 Comparison of growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NO_x-PM_{10} emissions categories

Table 4.61 Growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NO_x-PM_{10} emissions categories

Growth parameter	Category*	MSDP+NOx–PM_{10} emissions	P value	PSDP+NOx–PM_{10} emissions	P value	HSDP+NOx-PM_{10} emissions	P value
Child weight	Yes/High	28.43 ± 9.59	0.803	26.89 ± 8.82	0.266	28.43 ± 9.45	0.847
	No/Low	28.81 ± 9.03		28.51 ± 9.20		28.69 ± 9.25	
Child height	Yes/High	123.26 ± 15.09	0.164	122.77 ± 15.33	0.193	124.28 ± 14.91	0.368
	No/Low	126.49 ± 13.93		125.76 ± 14.29		126.06 ± 13.49	
Birth weight	Yes/High	3.15 ± 0.53	0.467	3.26 ± 0.63	0.889	3.28 ± 0.43	0.785
	No/Low	3.23 ± 0.68		3.27 ± 0.66		3.26 ± 0.64	
Body mass index	Yes/High	18.61 ± 6.48	0.211	17.71 ± 5.81	0.965	18.36 ± 5.94	0.370
	No/Low	17.64 ± 4.19		17.67 ± 4.57		17.69 ± 4.16	
Weight for age score	Yes/High	0.70 ± 1.17	0.437	0.74 ± 0.97	0.533	0.86 ± 1.13	0.105
	No/Low	0.55 ± 1.13		0.61 ± 1.19		0.59 ± 1.13	
Height for age score	Yes/High	0.21 ± 1.51	0.546	0.77 ± 1.39	0.319	0.51 ± 1.43	0.765
	No/Low	0.37 ± 1.54		0.51 ± 1.58		0.44 ± 1.58	
BMI z score	Yes/High	0.65 ± 1.17	0.287	0.51 ± 0.99	0.659	0.85 ± 1.05	0.056
	No/Low	0.41 ± 1.29		0.40 ± 1.32		0.57 ± 1.63	

MSDP: Maternal smoking during pregnancy; NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period; NPSDP: Fathers who did not smoke during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period; NHSDP: Household members who did not smoke during pregnancy period
High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
Low (↓) NOx-PM_{10} emissions + NMSDP: NOx emissions ≤ 10 + PM_{10} emissions ≤ 5 (in tonnes per annum) and mothers who did not smoke during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
Low (↓) NOx-PM_{10} emissions + NPSDP: NOx emissions ≤ 10 + PM_{10} emissions ≤ 5 (in tonnes per annum) and fathers who did not smoke during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
Low (↓) NOx-PM_{10} emissions + NHSDP: NO_X emissions ≤ 10 + PM_{10} emissions ≤ 5 (in tonnes per annum) and household members who did not smoke during pregnancy
BMI: Body Mass Index
*Smoking/Emissions category

In this analysis growth parameters were compared in relation to maternal, paternal and household smoking categories during pregnancy combined with NOx-PM10 emissions data. There was variation in different growth parameters in relation to NOx-PM_{10} emissions data combined separately with maternal, paternal and household smoking during pregnancy, but no significant associations were identified.

4.6.9 Comparison of growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NO_2-PM_{10} concentration categories

Table 4.62 Growth parameters in relation to maternal, paternal and household smoking during pregnancy categories combined with NO_2- PM_{10} concentration categories

Growth parameter	Category*	MSDP+ NO_2–PM_{10} C Mean ± SD	P value	PSDP + NO_2–PM_{10} C Mean ± SD	P value	HSDP+ NO_2-PM_{10} C Mean ± SD	P value
Child weight	Yes/High	27.72 ± 8.75	0.350	27.59 ± 8.59	0.333	28.10 ± 8.48	0.609
	No/Low	29.08 ± 7.78		29.13 ± 8.07		28.85 ± 7.79	
Child height	Yes/High	124.68 ± 14.19	0.154	125.72 ± 14.61	0.295	125.31 ± 14.39	0.557
	No/Low	128.01 ± 13.41		128.61 ± 13.29		126.78 ± 12.93	
Birthweight	Yes/High	3.16 ± 0.58	**0.009**	3.23 ± 0.63	0.060	3.19 ± 0.62	0.132
	No/Low	3.44 ± 0.65		3.46 ± 0.65		3.37 ± 0.67	
BMI	Yes/High	17.69 ± 4.08	0.331	17.37 ± 3.87	0.412	17.59 ± 3.69	0.625
	No/Low	17.05 ± 2.14		16.81 ± 1.97		17.27 ± 2.22	
Weight for age z score	Yes/High	0.72 ± 1.11	0.315	0.61 ± 1.03	0.824	0.70 ± 1.08	0.719
	No/Low	0.52 ± 1.12		0.57 ± 1.19		0.63 ± 1.07	
Height for age z score	Yes/High	0.58 ± 1.34	0.612	0.56 ± 1.36	0.664	0.51 ± 1.37	0.960
	No/Low	0.46 ± 1.47		0.68 ± 1.41		0.52 ± 1.31	
BMI z score	Yes/High	0.45 ± 1.32	0.769	0.40 ± 1.27	0.438	0.53 ± 1.17	0.537
	No/Low	0.52 ± 1.04		0.59 ± 0.94		0.66 ± 0.98	

MSDP: Maternal smoking during pregnancy
NMSDP Mothers who did not smoke during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
NPSDP: Fathers who did not smoke during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
NHSDP: Household members who did not smoke during pregnancy period
High (↑) NO_2-PM_{10} concentration and MSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
Low (↓) NO_2-PM_{10} concentration and NMSDP: NO_2 concentration ≤ 17.01 & PM_{10} concentration ≤ 13.91 microgram per m^3 and mothers who did not smoke during pregnancy
High (↑) NO_2-PM_{10} concentration and PSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and paternal smoking during pregnancy
Low (↓) NO_2-PM_{10} concentration and NPSDP: NO_2 concentration ≤ 17.01 & PM_{10} concentration ≤ 13.91 microgram per m^3 and fathers who did not smoke during pregnancy
High (↑) NO_2-PM_{10} concentration and HSDP: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy period
Low (↓) NO_2-PM_{10} concentration and NHSDP: NO_2 concentration ≤ 17.01 and PM_{10} concentration ≤ 13.91 microgram per m^3 and household member who did not smoke during pregnancy
BMI: Body Mass Index
*Smoking/Concentrations category

In this analysis growth parameters were compared in relation to maternal, paternal and household smoking categories during pregnancy combined with NO2-PM10 concentration data. There was variation in different growth parameters in relation to NO_2 and PM_{10} concentrations data combined separately with maternal, paternal and household smoking during pregnancy, but no significant associations were identified, except for mean birthweight which was lower with higher NO_2–PM_{10} concentrations when combined with maternal smoking during pregnancy (p=0.009).

4.7 Logistic regression analysis

The variables included in the model were based on significance from univariate analysis and on an empirical basis if possible risk factors from previous studies.

4.7.1 Logistic regression for childhood obesity

Table 4.63 Logistic regression for childhood obesity

Risk factor	Unadjusted odds ratio	95% CI	P value	Adjusted odds ratio	95% CI	P value
High NO_X-PM_{10} E + MSDP	3.696	1.372-9.955	0.016	4.468	1.215-16.429	**0.024**
High NO_X E + MSDP	3.433	1.517-7.768	0.003	9.000	1.936-86.522	**0.038**
High NO_X E + PSDP	2.973	1.025-8.626	0.052	9.143	1.104-75.708	**0.040**
High NO_X E + HSDP	2.526	1.096-5.822	0.027	4.266	1.137-16.001	**0.032**
High PM_{10} E + MSDP	2.782	1.207-6.416	0.026	3.352	1.088-10.208	**0.035**
High PM_{10} E + HSDP	2.588	1.197-5.595	0.020	2.591	0.942-7.123	0.065
High PM_{10} C + MSDP	10.485	1.378-79.762	0.003	3.112	0.355-27.256	0.303
MSDP	1.785	1.073-2.970	0.035	1.840	1.014-3.341	**0.045**
Male gender	0.935	0.582-1.502	0.810	0.218	0.052-1.201	0.126
LBW	1.351	0.604-5.022	0.501	5.295	0.841-33.336	0.576
ADHD	6.170	2.284-19.715	0.001	5.983	0.425-84.281	0.185
Upper quartile	1.121	0.422-2.980	1.000	1.074	0.107-10.728	0.952
DDA	1.653	0.921-2.966	0.099	1.095	0.190-6.296	0.961
Preterm birth	0.813	0.386-1.710	0.720	0.540	0.056- 5.162	0.592

**Variables included in the adjusted model : Household lower socio–economic status (upper quartile), doctor diagnosed asthma, maternal smoking during pregnancy, low birth weight , preterm birth, ADHD, gender, high NO_X emissions + MSDP, high NO_X emissions + PSDP, high NO_X emissions + HSDP, high PM_{10} emissions + MSDP, high PM_{10} emissions + HSDP, high PM_{10} concentrations + MSDP. High NO_X-PM_{10} concentrations + MSDP was excluded due to less number of count for the regression analysis.

OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg)
DDA: Doctor diagnosed asthma; ADHD: Attention Deficit Hyperactivity Disorder
Obesity: BMI z-score >1.64 (>95th centile)
Preterm: Reported as preterm by mothers in questionnaire
Upper quartile: (Townsend score > 4)
C: Concentration; E: Emissions
MSDP: Maternal smoking during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) PM_{10} emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
High (↑) PM_{10} concentration + MSDP: PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy

High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy

In this analysis, the significant independent association of childhood obesity with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There was a significant independent association of childhood obesity with high NO_X-PM_{10} emissions + MSDP (p=0.024), high NOx E + MSDP (p=0.038), high NO_X-E + PSDP (p=0.040), high NO_X-E + HSDP (p=0.032), high PM_{10} E + MSDP (p=0.035) and MSDP alone (p =0.045) with no other significant associations.

4.7.2 Logistic regression for ever wheeze

Table 4.64 Logistic regression for ever wheeze

Risk factor	Unadjusted odds ratio	95% CI	P value	Adjusted odds ratio	95% CI	P value
DDA	22.521	13.321-37.192	**0.000**	21.162	7.971-56.182	**0.000**
High NO_X E + PSDP	2.353	1.162-4.744	0.018	6.381	1.434-28.412	**0.015**
High PM_{10} E + PSDP	2.452	1.192-5.021	0.019	5.372	1.212-23.792	**0.027**
High NO_X E + MSDP	2.264	1.193-4.292	0.015	5.204	0.991-27.442	0.052
High PM_{10} E + HSDP	2.342	1.243-4.391	0.011	4.163	0.973-17.752	0.054
MSDP	1.752	1.152-2.661	**0.012**	2.412	0.941-6.242	0.068
High PM_{10} E	1.815	1.151-2.845	**0.013**	0.954	0.881-1.032	0.179
High NOx + PM_{10} E	2.733	1.242-6.031	**0.015**	2.353	0.872-6.351	0.092
High NO_X-PM_{10} E + MSDP	2.734	1.245-6.032	**0.015**	2.426	0.942-6.252	0.068
High NO_X-PM_{10} E + PSDP	2.445	1.053-5.663	0.045	4.323	0.601-30.921	0.145
High NO_X-PM_{10} E + HSDP	2.223	1.043-4.732	0.050	4.854	0.842-28.161	0.078
High PM_{10} E + MSDP	2.601	1.291-5.232	0.010	2.203	0.423-11.581	0.351
LBW	1.146	0.551-2.372	0.702	2.431	0.671-8.891	0.179
Upper quartile	1.074	0.501-2.292	1.000	0.884	0.172-4.581	0.881
C+W+B+	1.253	0.452-3.461	0.590	0.571	0.071-4.523	0.595
Male Gender	1.352	0.912-1.982	0.141	1.204	0.491-2.931	0.685
Preterm birth	2.431	1.503-3.931	0.000	2.091	0.712-6.161	0.179
Maternal asthma	1.753	1.042-2.941	0.040	2.973	0.762-11.213	0.079
Paternal asthma	3.361	1.862-3.491	<0.001	2.942	0.881-9.802	0.107

**Variables included in the adjusted model : Household socio–economic status (upper quartile), doctor diagnosed asthma, maternal smoking during pregnancy, high PM_{10} emissions, high NOx + PM_{10} emissions, high NO_X-PM_{10} emissions + MSDP, high NO_X-PM_{10} emissions + PSDP, high NO_X-PM_{10} emissions + HSDP, high NO_X emissions + MSDP, high NO_X emissions + PSDP, high PM_{10} emissions + MSDP, high PM_{10} emissions + PSDP, high PM_{10} emissions + HSDP, Low birthweight, Upper quartile, symptom triad of C+W+B+, male child, preterm birth, maternal and paternal asthma

OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg)
DDA: Doctor diagnosed asthma; ADHD: Attention Deficit Hyperactivity Disorder
Obesity: BMI z-score >1.64 (>95th centile)
Preterm: Reported as preterm by mothers in questionnaire
Upper quartile: (Townsend score > 4)
C: Concentration; E: Emissions
MSDP: Maternal smoking during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) PM_{10} emissions + PSDP: PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy

High (↑) PM10 emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy

In this analysis, the significant independent association of childhood ever wheeze with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There was a significant independent association of childhood ever wheeze with DDA (p=000), high NO_X emissions + PSDP (p=0.015) and high PM_{10} emissions + PSDP (p=0.027) with no other significant associations after adjusting for confounding factors.

4.7.3 Logistic regression for Croup

Table 4.65 Logistic regression for Croup

Risk factor	Unadjusted odds ratio	95% CI	P value	Adjusted odds ratio	95% CI	P value
High NO_X E	0.515	0.298-0.891	0.024	0.430	0.235-0.787	**0.006**
High NO_X E + PSDP	0.360	0.133-0.977	0.050	0.206	0.064-0.662	**0.008**
High NO_X E + HSDP	0.249	0.104-0.600	0.002	0.224	0.086-0.581	**0.001**
High NO_X-PM_{10} E + HSDP	0.163	0.036-0.250	0.011	0.152	0.037-0.698	**0.009**
High NO_X-PM_{10} E + PSDP	0.137	0.017-1.072	0.038	0.106	0.013-0.840	**0.034**
High NOx + PM_{10} E	0.349	0.147-0.830	0.015	0.279	0.110-0.710	0.070
HSDP	0.591	0.342-1.0202	0.004	0.604	0.319-1.141	0.120
High PM_{10} E + HSDP	0.218	0.051-0.930	0.025	0.249	0.058-1.077	0.063
Upper Quartile	2.251	0.529-9.568	0.415	2.404	0.549-10.526	0.245

**Variables included in the adjusted model: Household smoking during pregnancy, household socio–economic status (upper quartile), high NO_X emissions, high NO_X emissions + PSDP, high NO_X emissions + HSDP, high NO_X-PM_{10} emissions + PSDP, high NO_X-PM_{10} emissions + HSDP, high PM_{10} emissions + HSDP, high NOx + PM_{10} emissions, HSDP

OR: Odds Ratio
E: Emissions
Upper quartile: (Townsend score > 4)
PM_{10}: Particulate matter <10 microns
High (↑) NOx emissions: NOx emissions >10 tonnes per annum
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
High (↑) PM_{10} emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy

In this analysis, the significant independent association of croup with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There were significant independent association of decreased croup with high NO_X emissions (p=0.006), high NOx emissions + PSDP (p=0.008), high NO_X emissions + HSDP (p=0.001), high NO_X-PM_{10} emissions + HSDP (p=0.009) and high NO_X-PM_{10} emissions + PSDP (p=0.034) with no other significant association after adjusting for confounding factors.

4.7.4 Logistic regression for Hay fever

Table 4.66 Logistic regression for Hay fever

Risk factor	Unadjusted odds ratio	95% CI	P value	Adjusted odds ratio	95% CI	P value
HSDP	0.803	0.477-1.354	0.429	1.443	0.387-5.376	0.332
PSDP	0.946	0.534-1.739	1.000	2.080	0.467-9.261	0.831
High NOx E	0.443	0.250-0.784	0.006	0.684	0.175-2.667	0.998
High NOx E + HSDP	0.309	0.132-0.721	0.008	0.735	0.093-5.809	0.770
High NOx + PM_{10} E + HSDP	0.299	0.101-0.891	0.037	0.643	0.074-5.594	0.920
Allergy	403.92	53.64-3041.511	0.000	439.599	57.489-3361.451	0.410
Upper quartile	1.147	0.353-3.728)	1.000	1.486	0.225-9.801	0.240

**Variables included in the adjusted model : HSDP, PSDP, high NOx Emissions, high NOx + PM_{10} emissions + HSDP, Allergy and upper quartile

OR: Odds Ratio
Upper quartile: (Townsend score > 4)
E: Emissions
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy

In this analysis, the significant independent association of hay fever with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There was no significant independent association of hay fever with any of the air pollution or pregnancy smoking indicators after adjusting for the confounding factors.

4.7.5 Logistic regression for Bronchitis

Table 4.67 Logistic regression for Bronchitis

Risk factor	Unadjusted odds ratio	(95% CI)	P value	Adjusted odds ratio	(95% CI)	P value
DDA	5.176	2.906-9.219	0.000	5.874	1.804-19.122	**0.020**
PSDP	2.102	1.083-4.07	0.034	1.574	0.442-5.602	0.484
High NO_X + PM_{10} E	1.576	0.699-3.290	0.319	1.478	0.382-5.710	0.571
High PM_{10} E +MSDP	3.349	1.262-8.890	0.020	4.750	0.349-64.738	0.242
High NOx + PM_{10} E +PSDP	4.226	1.173-15.217	0.037	2.506	0.119-52.592	0.554
HSDP	1.444	0.819-2.546	0.244	1.756	0.713-4.321	0.220
Upper quartile	0.720	0.270-1.916	0.573	0.460	0.119-1.773	0.259
C+W+B+	0.892	0.200-3.970	0.483	1.000	0.122-8.204	1.000
LBW	0.882	0.306-2.543	1.000	0.438	0.055-3.465	0.434
Male Gender	0.704	0.404-1.225	0.265	0.770	0.311-1.906	0.573
Preterm birth	1.626	0.803-3.294	0.217	3.304	0.904-12.078	0.071

**Variables included in the adjusted model: Paternal smoking during pregnancy, high NO_X +PM_{10} emissions, high NOx + PM_{10} emissions + PSDP, high PM_{10} E + MSDP,
HSDP, Upper quartile, DDA, C+W+B+, LBW, male gender and preterm birth
OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg)
DDA: Doctor diagnosed asthma
Preterm: Reported as preterm by mothers in questionnaire
Upper quartile: (Townsend score > 4)
C+W+B+: Symptom triad of cough, wheeze and breathlessness
E: Emissions
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx-PM_{10} emissions: NOx emissions >10 and PM_{10} emissions > 5 tonnes per annum;
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy

In this analysis, the significant independent association of bronchitis with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There only significant independent association of bronchitis was with doctor diagnosed asthma ($p = 0.020$), after adjusting for confounding factors.

4.7.6 Logistic regression for female birth

Table 4.68 Logistic regression for female birth

Risk factor	Unadjusted odds ratio	(95% CI)	p value	Adjusted odds ratio	(95% CI)	p value
High PM_{10} C + MSDP	2.347	1.222-4.525	0.012	3.268	1.572-6.758	**0.001**
MSDP	1.942	1.369-2.755	0.000	1.597	1.720-3.530	**0.003**
High PM_{10} C + HSDP	2.227	1.121-4.424	0.025	2.673	1.236-5.780	**0.012**
High NOx E + HSDP	1.961	1.221-3.155	0.006	1.901	1.149-3.164	**0.013**
High NOx E + MSDP	2.567	1.545-4.251	<0.001	2.074	1.124-3.831	**0.020**
HSDP	1.684	1.244-2.273	0.001	2.197	1.103-4.391	**0.025**
High PM_{10} E + MSDP	1.942	1.043-3.610	0.044	2.123	1.104-4.082	**0.024**
High NOx E+ PM_{10} E + MSDP	2.114	2.277-4.132	0.031	1.629	0.586-4.534	0.249
Upper quartile	1.122	0.628-2.001	0.768	1.326	0.256-3.521	0.350

**Variables included in the adjusted model: MSDP, HSDP, high NOx E + MSDP, high NOx E + HSDP, high PM_{10} E + MSDP, high PM_{10} C + MSDP, high PM_{10} C + HSDP, high NOx E+ PM_{10} E + MSDP and upper quartile
OR: Odds Ratio; C: Concentrations; E: Emissions
Upper quartile: (Townsend score > 4); PM_{10}: Particulate matter <10 microns
MSDP: Maternal smoking during pregnancy
HSDP: Household members who smoked during pregnancy period
High (↑) NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) PM_{10} concentration + MSDP: PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
High (↑) PM_{10} concentration + HSDP: PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy
High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy

In this analysis, the significant independent association of female births with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There were significant independent associations of increased female births with high PM_{10} concentrations + MSDP (p=0.001), MSDP (0.003), high PM_{10} concentrations + HSDP (0.012), high NOx Emissions + HSDP (0.013), high NOx emissions + MSDP (0.020), HSDP (0.025), high PM_{10} emissions + MSDP (0.024) after adjusting for confounding factors with no other significant associations

4.7.7 Logistic regression for preterm birth

Table 4.69 Logistic regression for preterm birth

Risk factor	Unadjusted odds ratio	95% CI	P value	Adjusted odds ratio	95% CI	P value
Maternal asthma	2.041	1.191-3.480	0.011	2.082	0.192-4.353	0.081
Gender (Male)	0.933	0.612-1.410	0.751	0.491	0.362-1.921	0.927
High NO_2 C + MSDP	0.232	0.073-0.704	0.014	**0.182**	**0.050-0.631**	**0.007**
High NO_2-PM_{10} C + MSDP	0.741	0.302-1.891	0.481	2.081	0.142-30.372	0.594
High NO_2-PM_{10} C + HSDP	1.042	0.412-2.643	1.000	3.792	0.364-40.292	0.269
High NO_2-PM_{10} C	1.021	0.481 -2.142	1.000	0.381	0.091 -1.642	0.248
Upper Quartile	0.782	0.361-1.662	0.531	2.422	0.541 – 10.842	0.802
LBW	1.065	0.481-2.342	0.839	1.011	0.312 – 3.291	0.994

**Variables included in the adjusted model: MSDP+NO_X-PM_{10} concentrations, HSDP+NO_X-PM_{10} concentrations maternal asthma, gender (male), highNO_2-PM_{10} concentrations, upper quartile and low birth weight.
OR: Odds Ratio
LBW: Low birthweight: Birthweight < 2.5kg); C: Concentration
PM_{10}: Particulate matter <10 microns; Upper quartile: (Townsend score > 4)
MSDP: Maternal smoking during pregnancy; HSDP: Household members who smoked during pregnancy period
High (↑) NO_2-PM_{10} concentration and MSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
High (↑) NO_2-PM_{10} concentration and HSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy
High (↑) NO2 concentration + MSDP: NO_2 concentration >17.01 microgram per m^3 and maternal smoking during pregnancy
High (↑) NO_2-PM_{10} concentration: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3

In this analysis, the significant independent association of preterm birth with risk factors was conducted by backward stepwise logistic regression method after adjusting for confounding factors. There was no significant independent association of preterm births with any of the air pollution or pregnancy smoking indicators after adjusting for confounding factors,except for high NO2 C + MSDP (0.007).

4.8 Summary findings of the logistic regression analysis showing significant birth and child health outcomes in relation to air pollution categories and combined air pollution and pregnancy smoking categories

Table 4.70 Summary of backward stepwise logistic regression analysis showing significant association of birth and child health outcomes in relation to air pollution categories and their combinations with pregnancy smoking categories.

Air pollution categories*			
High NO_X E	**High PM_{10} E**	**High NO_2 C**	**High PM_{10} C**
Croup			
Combined air pollution and pregnancy smoking categories			
High NO_X E + MSDP	**High PM_{10} E + MSDP**	**High NO_2 C + MSDP**	**High PM_{10} C + MSDP**
Obesity **Female birth**	**Obesity** **Female birth**	Preterm birth	**Female birth**
High NO_X E + PSDP	**High PM_{10} E + PSDP**	**High NO_2 C + PSDP**	**High PM_{10} C + PSDP**
Obesity **Ever wheeze** **Croup**	**Ever wheeze**		
High NO_X E + HSDP	**High PM_{10} E + HSDP**	**High NO_2 C + HSDP**	**High PM_{10} C + HSDP**
Obesity **Female birth** **Croup**			**Female birth**
	MSDP + High NO_X- PM_{10} E	**MSDP + High NO_2-PM_{10} C**	
	Obesity** **AOR 4.47, 95% CI 1.22-16.43, p=0.024^**		
PSDP + High NO_X- PM_{10} E	**PSDP + High NO_2-PM_{10} C**	**HSDP +High NO_X- PM_{10} E**	**HSDP +High NO_2-PM_{10} C**
Croup** **AOR 0.02, 95% CI 0.01-0.84, p=0.034**		**Croup**** **AOR 0.15, 95% CI 0.04-0.70, p=0.015**	

*Combined air pollution categories not included as there was no significant outcomes in relation to combined air pollution categories

** Adjusted odds ratio (AOR) only added for the final significant child health outcomes in relation to combined air pollution and pregnancy smoking exposure (shaded in yellow)

^ of pubic health importance

Exposure category headings
Exposure sub category headings
Outcome
Increased adjusted odds ratio
Decreased adjusted odds ratio

Obesity: BMI z-score >1.64 (>95th centile)
C: Concentration
E: Emissions
NOx: oxides of Nitrogen
NO_2: Nitrogen dioxide
PM_{10}: Particulate matter <10 microns
High (↑) NOx emissions: NOx emissions >10 tonnes per annum
High (↑) NO_2 C: NO_2 concentrations >17.07 microgram per m^3
High (↑) PM_{10} emissions: PM_{10} emissions >5 tonnes per annum
High (↑) PM_{10} C: PM_{10} concentrations >13.91 microgram per m^3
High (↑) NOx-PM_{10} emissions: NOx emissions >10 and PM_{10} emissions > 5 tonnes per annum
High (↑) NO_2-PM_{10} concentration: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3;
MSDP: Maternal smoking during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx emissions + PSDP: NOx emissions > 10 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx emissions + MSDP: NOx emissions > 10 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx emissions + HSDP: NOx emissions > 10 (in tonnes per annum) and household member smoking during pregnancy
High (↑) PM_{10} emissions + MSDP: PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) PM_{10} emissions + PSDP: PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) PM_{10} emissions + HSDP: PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
High (↑) NO_2 concentration + PSDP: NO_2 concentration >17.01 microgram per m^3 and paternal smoking during pregnancy
High (↑) NO_2 concentration + MSDP: NO_2 concentration >17.01 microgram per m^3 and maternal smoking during pregnancy
High (↑) NO_2 concentration + HSDP: NO_2 concentration >17.01 microgram per m^3 and household member smoking during pregnancy
High (↑) PM_{10} concentration + MSDP: PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
High (↑) PM_{10} concentration + PSDP: PM_{10} concentration >13.91 microgram per m^3 and paternal smoking during pregnancy
High (↑) PM_{10} concentration + HSDP: PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy
High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy
High (↑) NO_2-PM_{10} concentration and MSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and maternal smoking during pregnancy
High (↑) NO_2-PM_{10} concentration and PSDP: NO_2 concentration >17.01 & PM_{10} concentration >13.91 microgram per m^3 and paternal smoking during pregnancy
High (↑) NO_2-PM_{10} concentration and HSDP: NO_2 concentration >17.01 and PM_{10} concentration >13.91 microgram per m^3 and household member smoking during pregnancy

Significant independent associations of birth and child health outcomes obtained from backward stepwise logistic regression after adjusted for relevant confounding factors (included below each table) are listed in bold in the table above (Table 4.70) with increases shaded in green and decreases shaded in red. Significant outcomes in relation to independent pregnancy smoking categories have not been included as it has been already previously studied for a large sample data from 15 schools in the same area. Other relevant significant interaction factors other than outcomes have been excluded from this table and only child and birth outcomes have been included.

There was significantly increased childhood obesity with maternal smoking during pregnancy when combined separately with high NOx emissions and high PM_{10} emissions respectively; significantly increased female birth independently with high PM_{10} concentrations combined with maternal smoking during pregnancy and household member smoking during pregnancy and significantly increased childhood obesity, ever wheeze and decreased croup with high NOx emissions combined with paternal smoking during pregnancy after adjusting for relevant confounding factors.

High PM_{10} emissions with paternal smoking during pregnancy was significantly associated with increase ever wheeze and high PM_{10} emissions with household member smoking during pregnancy with increased childhood obesity, female births and decreased croup and there was significantly increased independent association of childhood obesity with combined high (NOx +PM10 emissions) and maternal smoking during pregnancy.

To summarise the association of child health outcomes with combined air pollution and smoking cateogries, there was significantly increased association of childhood obesity with high (NOx + PM10) emissions when combined with maternal smoking during pregnancy.

There was significantly decreased independent association of croup with high (NOx + PM10) emissions when combined with paternal smoking during pregnancy and household member smoking during pregnancy separately.

4.9 Population attributable risk for birth and child health outcomes from logistic regression in relation to smoking and air pollution exposures*

Table 4.71 Risk estimates for population attributable risk based on significant associations of child health and birth outcomes in relation to combined pregnancy smoking and air pollution exposure**

Exposure	Prevalence (%)	Outcome	Prevalence (%)	Odds ratio	95% CI	Adjusted odds ratio	95% CI	P value	Population Attributable Risk (%)
High NOx-PM_{10} E + MSDP	23.3% (47/202)	Obesity	24% (9/37)	3.041	1.336-6.920	4.468	1.215-16.43	**0.024**	**44.38 (4.85-77.97)**
High NOx-PM_{10} E + PSDP	31.4% (49/156)	Croup	2.1% (1/48)	0.155	0.021-1.143	0.016	0.013-0.840	**0.034**	**-42.86 (-5.26 - -44.93)**
High NOx-PM_{10} E+ HSDP	37.4% (68/182)	Croup	3.0% (2/67)	0.188	0.045-0.787	0.152	0.037-0.698	**0.015**	**-45.87 (-55.05- -12.49)**

*Population attributable risks calculated only for the significantly associated child health outcomes in relation to combined high NOx-PM10 categories and pregnancy smoking category

**Adjusted odds ratios used instead of relative risk for better results. Obesity: BMI z-score >1.64 (>95th centile)

C: Concentration
E: Emissions
NOx: oxides of Nitrogen
NO_2: Nitrogen dioxide
PM_{10}: Particulate matter <10 microns
MSDP: Maternal smoking during pregnancy
PSDP: Fathers who smoked during his partner's pregnancy period
HSDP: Household members who smoked during pregnancy period
High (↑) NOx-PM_{10} emissions + MSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and maternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + PSDP: NOx emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and paternal smoking during pregnancy
High (↑) NOx-PM_{10} emissions + HSDP: NO_X emissions > 10 + PM_{10} emissions > 5 (in tonnes per annum) and household member smoking during pregnancy

The population attributable risk for birth and child health outcomes from logistic regression in relation to smoking and air pollution exposures are summarised in table above (Table 4.71). The population attributable risk (PAR) estimates were: For childhood obesity, it was 44.38% due to high NOx-PM_{10} emissions + MSDP. The PAR for childhood croup associated with high NOx- PM_{10} emissions + PSDP was -42.86%, and -45.87% for high NOx-PM_{10} E+ HSDP. The population attributable risks for the remaining significant child health outcomes in relation to individual air pollution categories combined with pregnancy smoking categories have not been included in the table. The PAR for childhood ever wheeze associated with high NOx emissions + PSDP was 61.40 % and -57.10 % for high PM_{10} E+ PSDP.

4.10 Venn diagram representation of birth and child health outcomes in relation to pregnancy smoking and air pollution (Childhood obesity used as the main example)

The interactions of childhood obesity assessed in relation to combinations of different pregnancy smoking indicators and air pollution categories have been included in the tables and figures below to illustrate how the Venn diagrams can be used as a health promotion tool to illustrate the association of childhood obesity with air pollution and pregnancy smoking.

Childhood obesity has been used as the main example in this section for Venn diagrams because of it public health importance. The overlap between the three variables in each Venn diagram has been coloured as red to show the meeting point of the three variables. The tables list the numbers corresponding to each part of the Venn diagram, which also includes the numbers showing the interaction between each of the variables used in the Venn diagram along with the numbers common to each variable obtained from the univariate - cross tabulation analysis using sub categorisation and corresponding Venn diagrams generated using the Venn diagram software. The number for each category for the Venn diagrams are indicated in the tables below (Tables 4.72 to 4.91), and the Venn diagrams illustrated below from figures 4.12 to 4.31.

A total of nine children had obesity associated with high NOx-PM_{10} emissions + MSDP. There was one child with croup associated with high NOx-PM_{10} emissions + PSDP, compared to two children with croup associated with high NOx-PM_{10} emissions + HSDP. One child had croup associated with high NOx-PM_{10} emissions + PSDP and high NOx-PM_{10} emissions + HSDP.

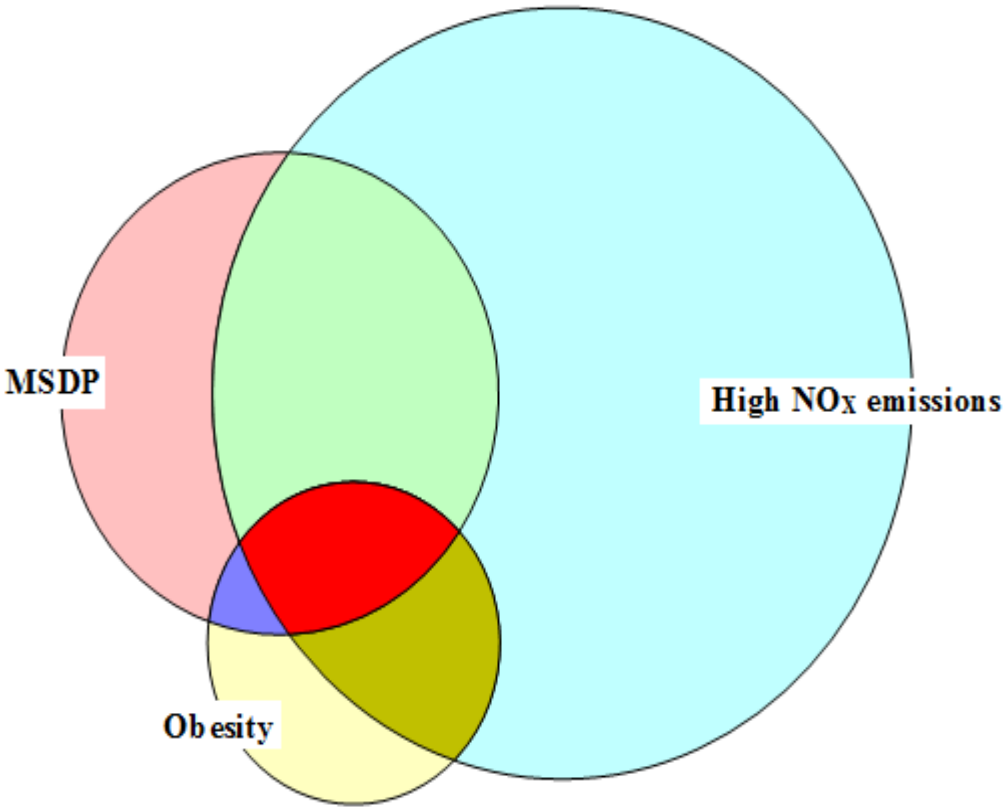
To summarise, the Venn diagrams and tables below shows that the childhood obesity was more commonly associated with household smoking during pregnancy followed by maternal smoking during pregnancy and paternal smoking during pregnancy, when combined with individual and combined forms of air pollution indicators as illustrated by the common red area formed by the interaction between the three variables forming the three circles in each Venn diagram. The description for each table and Venn diagram is provided below each table and above each Venn diagram.

4.10.01 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NOx emissions and MSDP

Table 4.72 Childhood obesity in relation to high NOx emissions and MSDP

Venn diagram variables	**Prevalence (%)**
High NO_X Emissions	458/668 (68.6)
MSDP	179/704 (25.4)
Obesity	80/577 (13.9)
MSDP x High NO_X emissions	112/158 (70.9)
High NO_X emissions x Obesity	51/355 (14.4)
MSDP x Obesity	28/140 (20.0)
Venn diagram variables	**Numbers for Venn diagram**
High NO_X Emissions	458
MSDP	179
Obesity	80
MSDP ∩ High NO_X emissions	112
High NO_X emissions ∩ Obesity	51
MSDP ∩ Obesity	28
MSDP ∩ High NO_X emissions ∩ Obesity	**20**

Figure 4.12 Childhood obesity in relation to high NOx emissions and MSDP



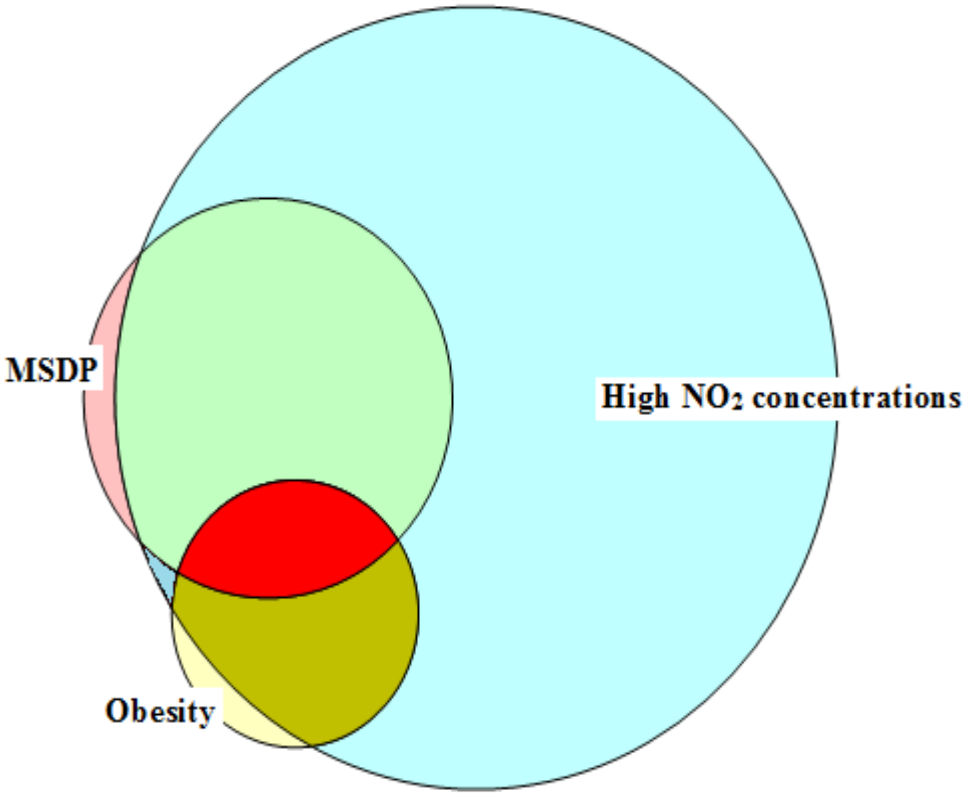
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.72) along with the proportional Venn diagram for childhood obesity in relation to high NOx emissions and MSDP which has been illustrated below the table (Figure 4.12). The Venn diagram shows that the proportion of childhood obesity associated with high NOx emissions was higher when compared to that of maternal smoking during pregnancy. The proportion of childhood obesity associated with MSDP and high NOx emissions was higher when compared to that of MSDP alone, whereas it was lower when compared to high NOx emissions alone.

4.10.02 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NO_2 concentrations and MSDP

Table 4.73 Childhood obesity in relation to high NO_2 concentrations and MSDP

Venn diagram variables	**Prevalence (%)**
High NO_2 concentrations	686/706 (97.2)
MSDP	179/704 (25.4)
Obesity	80/577 (13.9)
MSDP x High NO_2 concentrations	169/172 (98.3)
High NO_2 concentrations x Obesity	73/539 (13.5)
MSDP x Obesity	28/140 (20.0)
Venn diagram variables	**Numbers for Venn diagram**
High NO_2 concentrations	686
MSDP	179
Obesity	80
MSDP ∩ High NO_2 concentrations	169
High NO_2 concentrations ∩ Obesity	73
MSDP ∩ Obesity	28
MSDP ∩ High NO_2 concentrations ∩ Obesity	**28**

Figure 4.13 Childhood obesity in relation to high NO_2 concentrations and MSDP



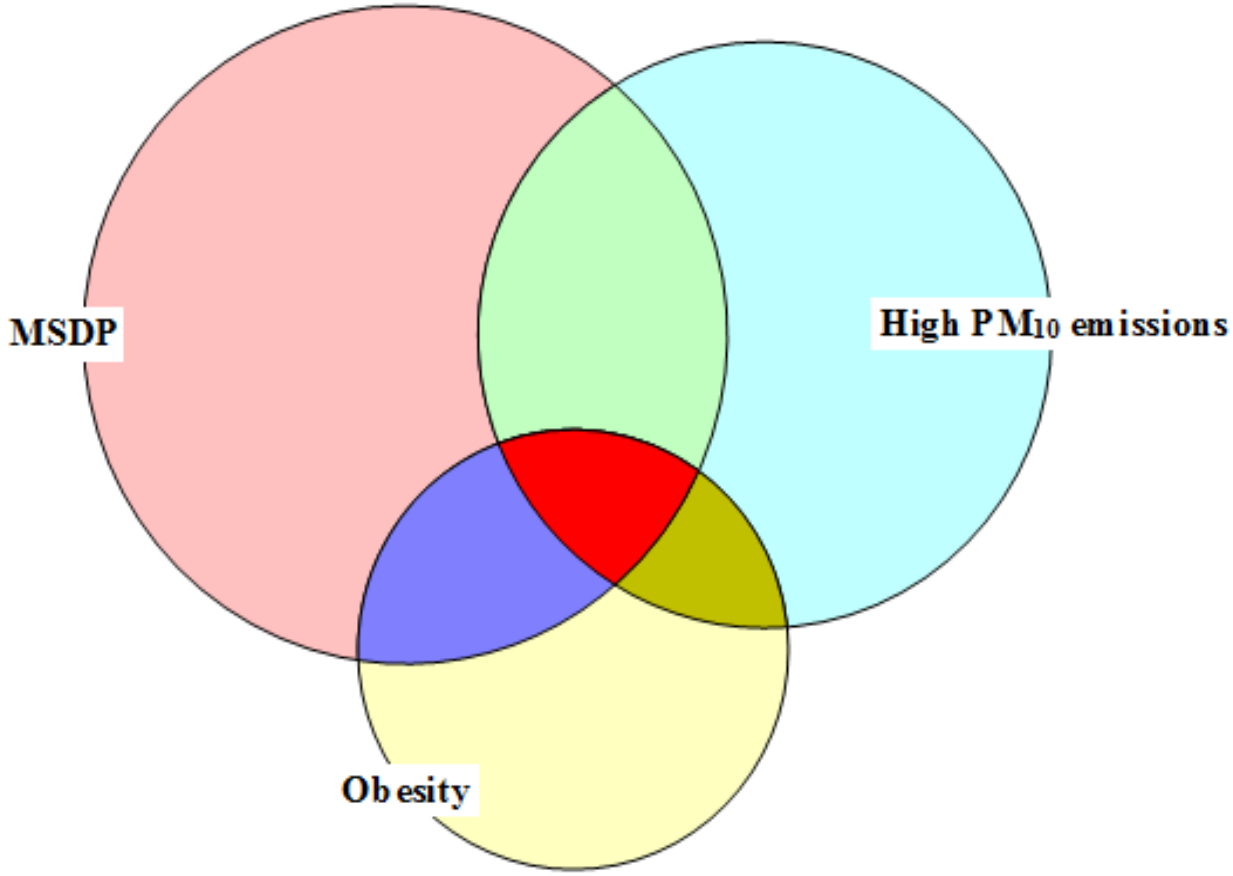
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.73) along with the proportional Venn diagram for childhood obesity in relation to high NO2 concentrations and MSDP which has been illustrated below the table (Figure 4.13). The Venn diagram shows that the proportion of childhood obesity associated with high NO2 concentrations was higher when compared to that of maternal smoking during pregnancy. The proportion of childhood obesity associated with MSDP and high NO2 concentrations was equal when compared to that of MSDP alone, whereas it was lower when compared to high NO2 concentrations alone.

4.10.03 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high PM10 emissions and MSDP

Table 4.74 Childhood obesity in relation to high PM_{10} emissions and MSDP

Venn diagram variables	**Prevalence (%)**
High PM_{10} emissions	142/668 (21.3)
MSDP	179/704 (25.4)
Obesity	80/577 (13.9)
MSDP x High PM_{10} emissions	47/158 (29.7)
High PM_{10} emissions x Obesity	18/109 (16.5)
MSDP x Obesity	28/140 (20.0)
Venn diagram variables	**Number for Venn diagram**
High PM_{10} emissions	142
MSDP	179
Obesity	80
MSDP ∩ High PM_{10} emissions	47
High PM_{10} emissions ∩ Obesity	18
MSDP ∩ Obesity	28
MSDP ∩ High PM_{10} emissions ∩ Obesity	**9**

Figure 4.14 Childhood obesity in relation to high PM_{10} emissions and MSDP



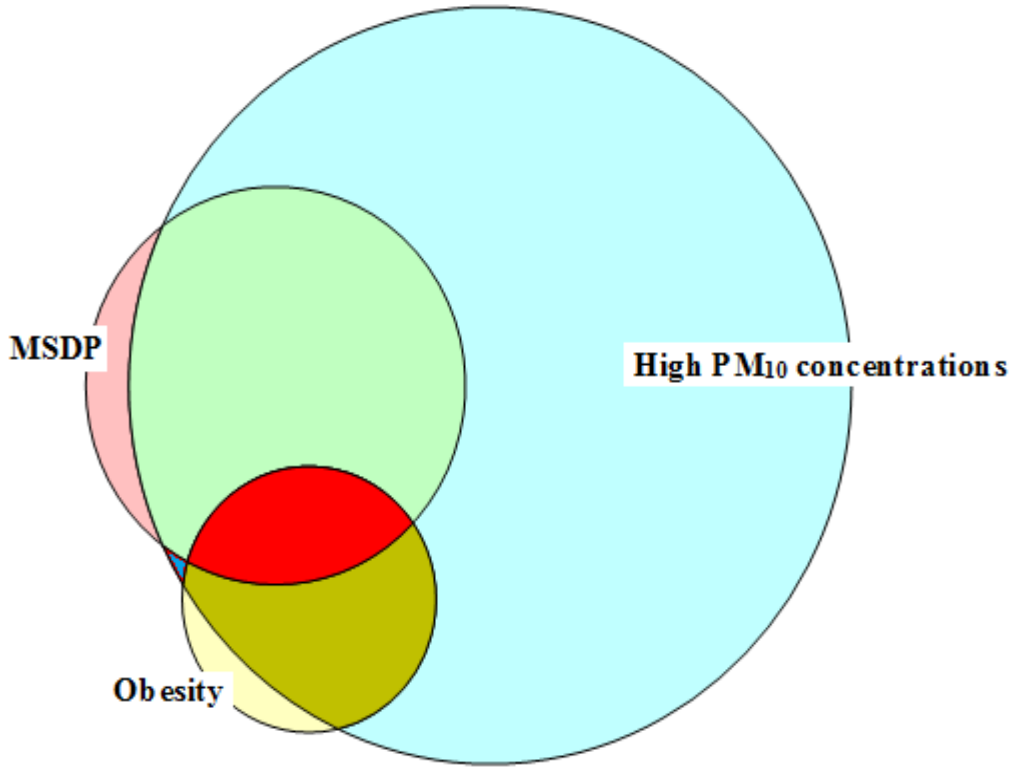
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.74) along with the proportional Venn diagram for childhood obesity in relation to high PM_{10} emissions and MSDP which has been illustrated below the table (Figure 4.14). The Venn diagram shows that the proportion of childhood obesity associated with high PM_{10} emissions was lower when compared to that of maternal smoking during pregnancy. The proportion of childhood obesity associated with MSDP and high PM_{10} emissions was lower when compared to that of MSDP or high PM_{10} emissions alone.

4.10.04 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high PM10 concentrations and MSDP

Table 4.75 Childhood obesity in relation to high PM_{10} concentrations and MSDP

Venn diagram variables	**Prevalence (%)**
High PM_{10} concentrations	648/706 (91.8)
MSDP	179/704 (25.4)
Obesity	80/577 (13.9)
MSDP x High PM_{10} concentrations	164/172 (95.3)
High PM_{10} concentrations x Obesity	71/504 (14.1)
MSDP x Obesity	28/140 (20.0)
Venn diagram variables	**Numbers for Venn diagram**
High PM_{10} concentrations	648
MSDP	179
Obesity	80
MSDP ∩ High PM_{10} concentrations	164
High PM_{10} concentrations ∩ Obesity	71
MSDP ∩ Obesity	28
MSDP ∩ High PM_{10} concentrations ∩ Obesity	**27**

Figure 4.15 Childhood obesity in relation to high PM_{10} concentrations and MSDP



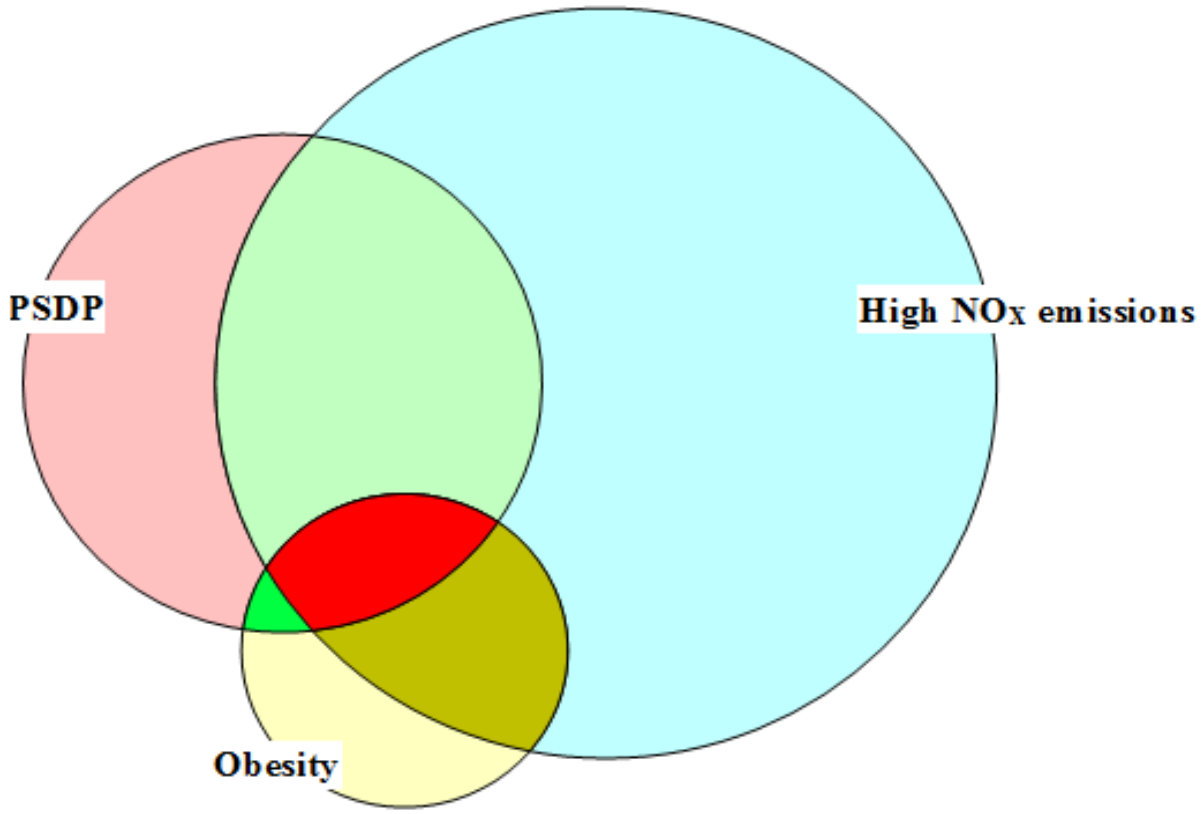
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.75) along with the proportional Venn diagram for childhood obesity in relation to high PM10 concentrations and MSDP which has been illustrated below the table (Figure 4.15). The Venn diagram shows that the proportion of childhood obesity associated with high PM10 concentrations was higher when compared to that of maternal smoking during pregnancy. The proportion of childhood obesity associated with MSDP and high PM10 concentrations was equal when compared to that of MSDP alone, whereas it was lower when compared to high PM10 concentrations.

4.10.05 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NOx emissions and PSDP

Table 4.76 Childhood obesity in relation to high NOx emissions and PSDP

Venn diagram variables	**Prevalence (%)**
High NO_X emissions	458/668 (68.6)
PSDP	202/565 (35.8)
Obesity	80/577 (13.9)
PSDP x High NO_X emissions	119/178 (66.9)
High NO_X emissions x Obesity	51/355 (14.4)
PSDP x Obesity	22/162 (13.6)
Venn diagram variables	**Number for Venn diagram**
High NO_X emissions	458
PSDP	202
Obesity	80
PSDP ∩ High NO_X emissions	119
High NO_X emissions ∩ Obesity	51
PSDP ∩ Obesity	22
PSDP ∩ High NO_X emissions ∩ Obesity	**14**

Figure 4.16 Childhood obesity in relation to high NOx emissions and PSDP



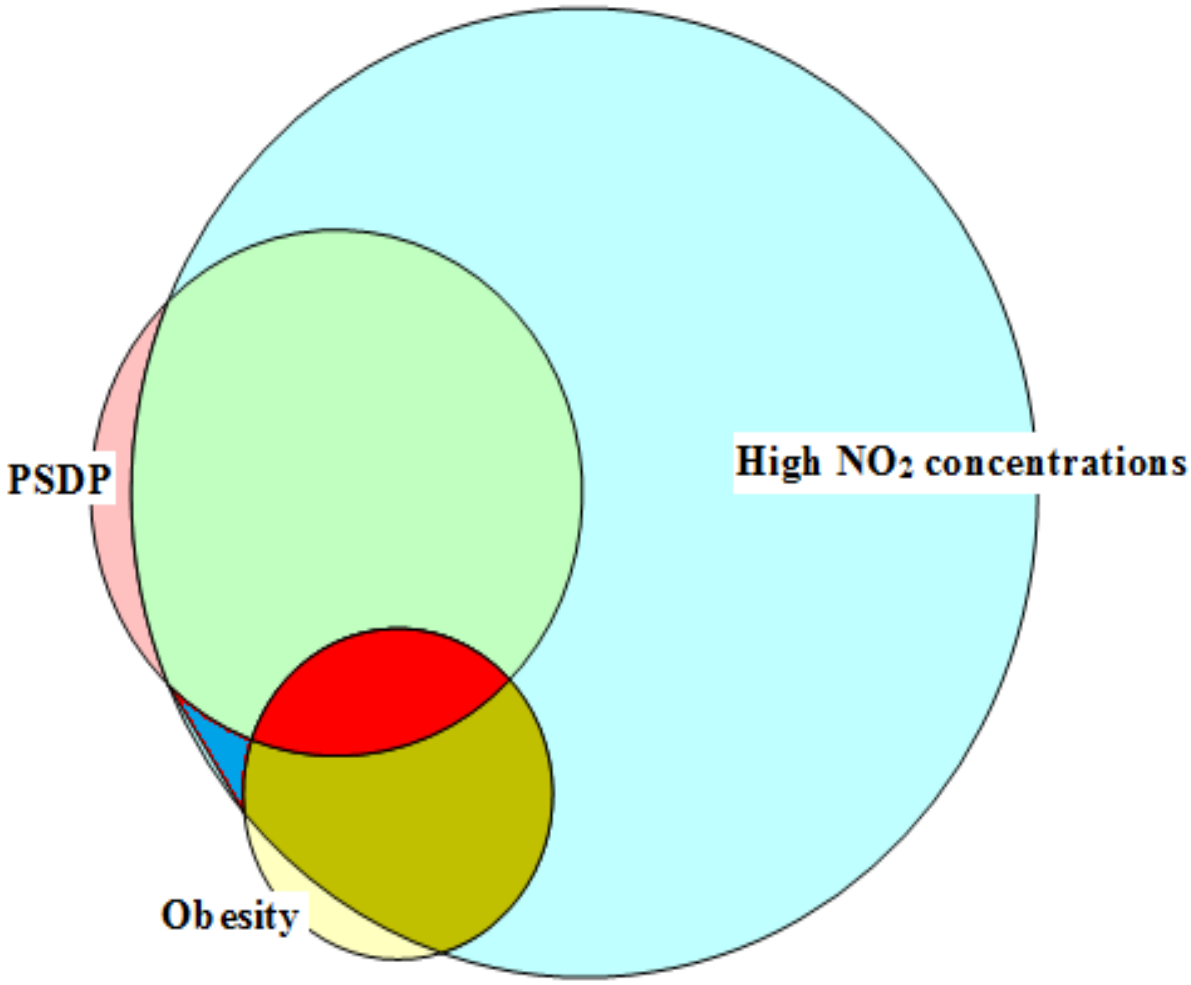
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.76) along with the proportional Venn diagram for childhood obesity in relation to high NOx emissions and PSDP which has been illustrated below the table (Figure 4.16). The Venn diagram shows that the proportion of childhood obesity associated with high NOx emissions was higher when compared to that of paternal smoking during pregnancy. The proportion of childhood obesity associated with PSDP and high NOx emissions was higher when compared to that of PSDP alone, whereas it was lower when compared to high NOx emissions alone.

4.10.06 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NO_2 concentrations and PSDP

Table 4.77 Childhood obesity in relation to high NO_2 concentrations and PSDP

Venn diagram variables	**Prevalence (%)**
High NO_2 concentrations	686/706 (97.2)
PSDP	202/565 (35.8)
Obesity	80/577 (13.9)
PSDP x High NO_2 concentrations	191/194 (98.5)
High NO_2 concentrations x Obesity	73/539 (13.5)
PSDP x Obesity	22/162 (13.6)
Venn diagram variables	**Numbers for Venn diagram**
High NO_2 concentrations	686
PSDP	202
Obesity	80
PSDP ∩ High NO_2 concentrations	191
High NO_2 concentrations ∩ Obesity	73
PSDP ∩ Obesity	22
PSDP ∩ High NO_2 concentrations ∩ Obesity	**22**

Figure 4.17 Childhood obesity in relation to high NO_2 concentrations and PSDP



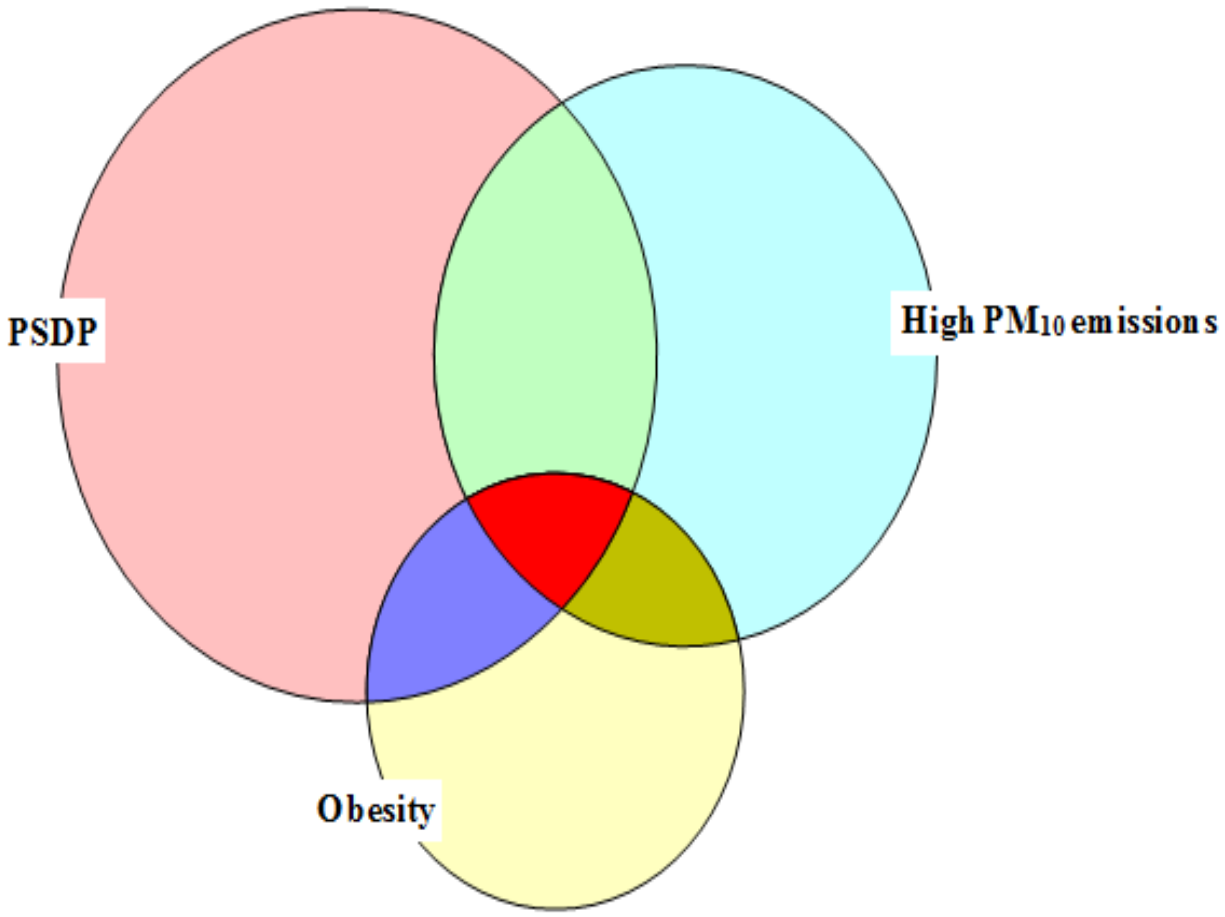
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.77) along with the proportional Venn diagram for childhood obesity in relation to high NO2 concentrations and PSDP which has been illustrated below the table (Figure 4.17). The Venn diagram shows that the proportion of childhood obesity associated with high NO2 concentrations was higher when compared to that of paternal smoking during pregnancy. The proportion of childhood obesity associated with PSDP and high NO2 concentrations was equal when compared to that of PSDP alone, whereas it was lower when compared to high NO2 concentrations alone.

4.10.07 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high PM10 emissions and PSDP

Table 4.78 Childhood obesity in relation to high PM_{10} emissions and PSDP

Venn diagram variables	**Prevalence (%)**
High PM_{10} emissions	142/668 (21.3)
PSDP	202/565 (35.8)
Obesity	80/577 (13.9)
PSDP x High PM_{10} emissions	49/178 (27.5)
High PM_{10} emissions x Obesity	18/109 (16.5)
PSDP x Obesity	22/162 (13.6)
Venn diagram variables	**Numbers for Venn diagram**
High PM_{10} emissions	142
PSDP	202
Obesity	80
PSDP ∩ High PM_{10} emissions	49
High PM_{10} emissions ∩ Obesity	18
PSDP ∩ Obesity	22
PSDP ∩ High PM_{10} emissions ∩ Obesity	**7**

Figure 4.18 Childhood obesity in relation to high PM_{10} emissions and PSDP



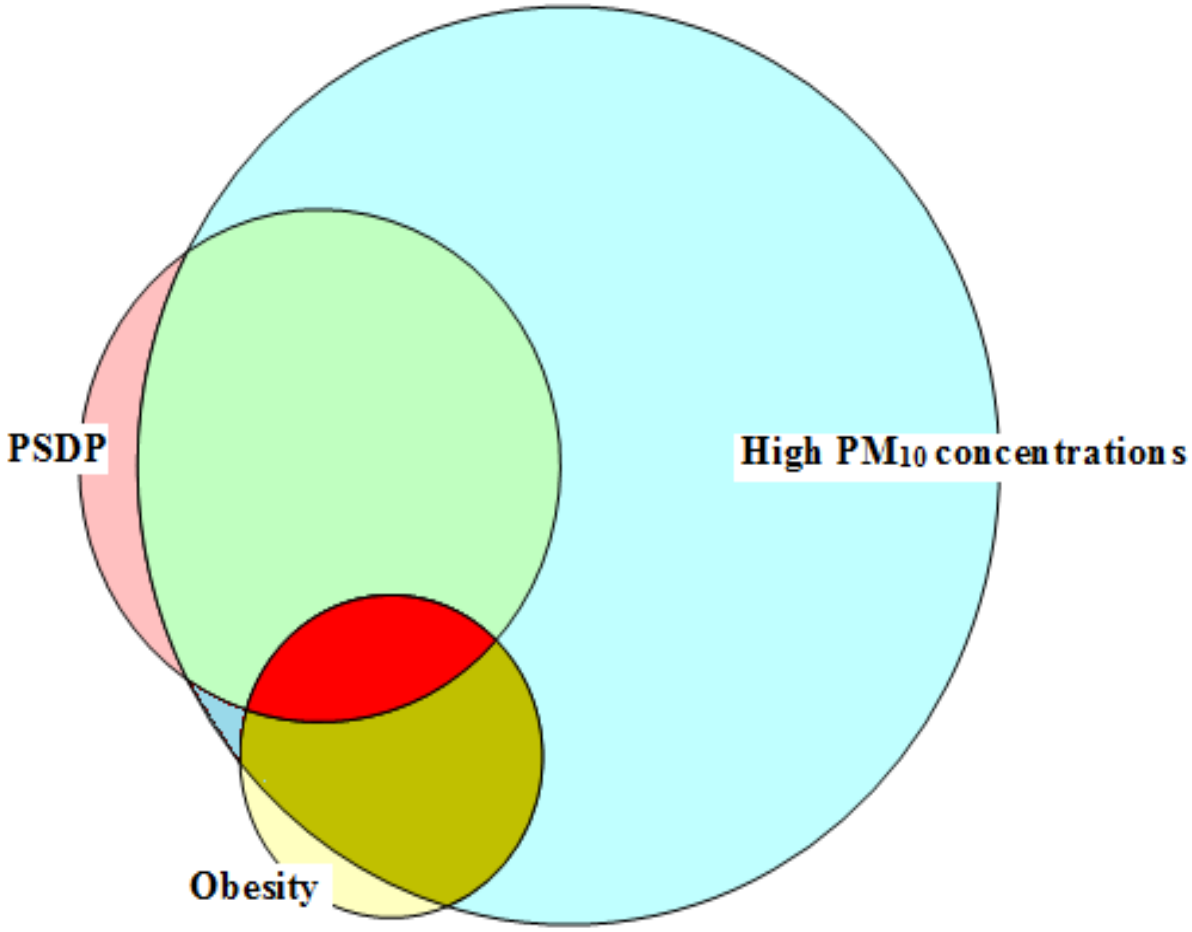
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.78) along with the proportional Venn diagram for childhood obesity in relation to high PM10 emissions and PSDP which has been illustrated below the table (Figure 4.18). The Venn diagram shows that the proportion of childhood obesity associated with high PM10 emissions was similar to that of paternal smoking during pregnancy. The proportion of childhood obesity associated with PSDP and high PM10 emissions was lower when compared to that of PSDP or high PM10 emissions alone.

4.10.08 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high PM10 concentrations and PSDP

Table 4.79 Childhood obesity in relation to high PM_{10} concentrations and PSDP

Venn diagram variables	**Prevalence (%)**
High PM_{10} concentrations	648/706 (91.8)
PSDP	202/565 (35.8)
Obesity	80/577 (13.9)
PSDP x High PM_{10} concentrations	183/194 (94.3)
High PM_{10} concentrations x Obesity	71/504 (14.1)
PSDP x Obesity	22/162 (13.6)
Venn diagram variables	**Numbers for Venn diagram**
High PM_{10} concentrations	648
PSDP	202
Obesity	80
PSDP ∩ High PM_{10} concentrations	183
High PM_{10} concentrations ∩ Obesity	71
PSDP ∩ Obesity	22
PSDP ∩ High PM_{10} concentrations ∩ Obesity	**21**

Figure 4.19 Childhood obesity in relation to high PM_{10} concentrations and PSDP



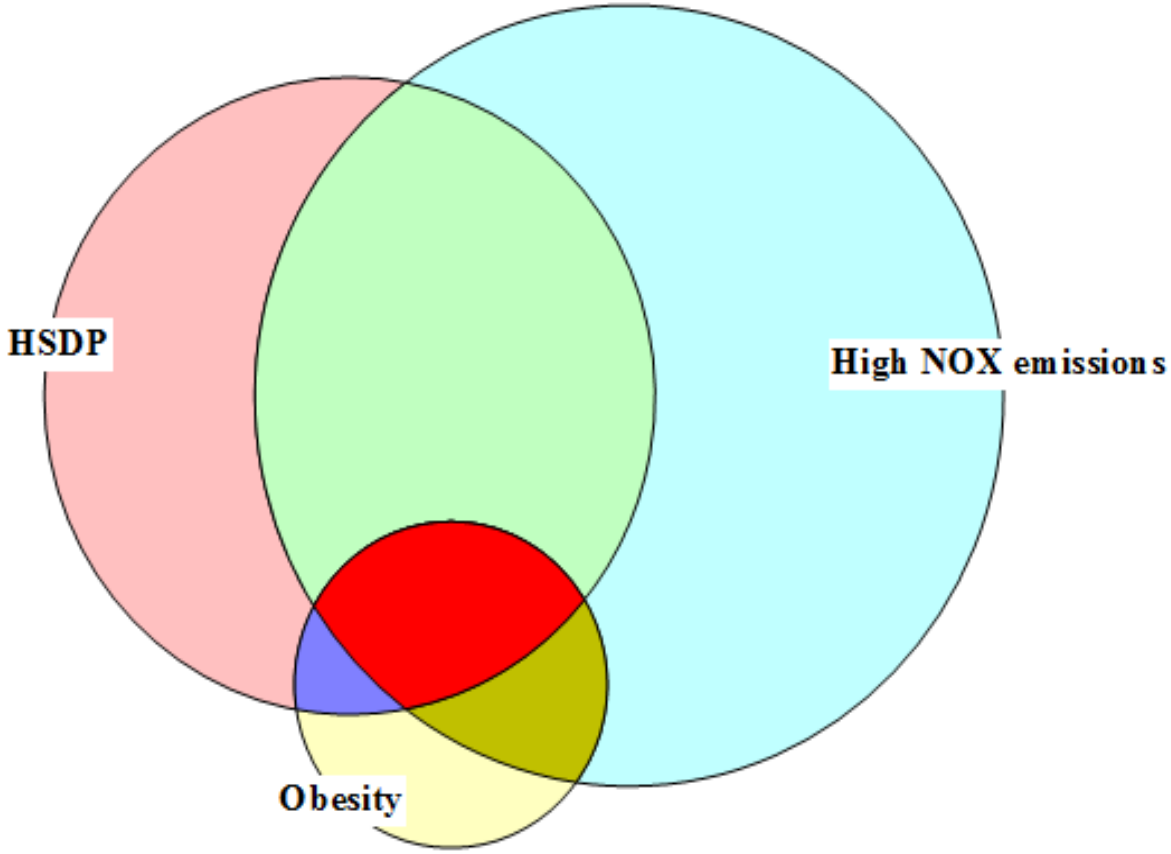
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.79) along with the proportional Venn diagram for childhood obesity in relation to high PM10 concentrations and PSDP which has been illustrated below the table (Figure 4.19). The Venn diagram shows that the proportion of childhood obesity associated with high PM10 concentrations was higher when compared to that of paternal smoking during pregnancy. The proportion of childhood obesity associated with PSDP and high PM10 concentrations was equal when compared to that of PSDP alone, whereas it was lower when compared to high PM10 concentrations alone.

4.10.09 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NOx emissions and HSDP

Table 4.80 Childhood obesity in relation to high NOx emissions and HSDP

Venn diagram variables	**Prevalence (%)**
High NO_X emissions	458/668 (68.6)
HSDP	305/698 (43.7)
Obesity	80/577 (13.9)
HSDP x High NOx emissions	183/270 (67.8)
High NOx emissions x Obesity	51/355 (14.4)
HSDP x Obesity	39/238 (16.4)
Venn diagram variables	**Numbers for Venn diagram**
High NO_X Emissions	458
HSDP	305
Obesity	80
HSDP ∩ High NO_X emissions	183
High NOX emissions ∩ Obesity	51
HSDP ∩ Obesity	39
HSDP ∩ High NO_X emissions ∩ Obesity	**28**

Figure 4.20 Childhood obesity in relation to high NOx emissions and HSDP



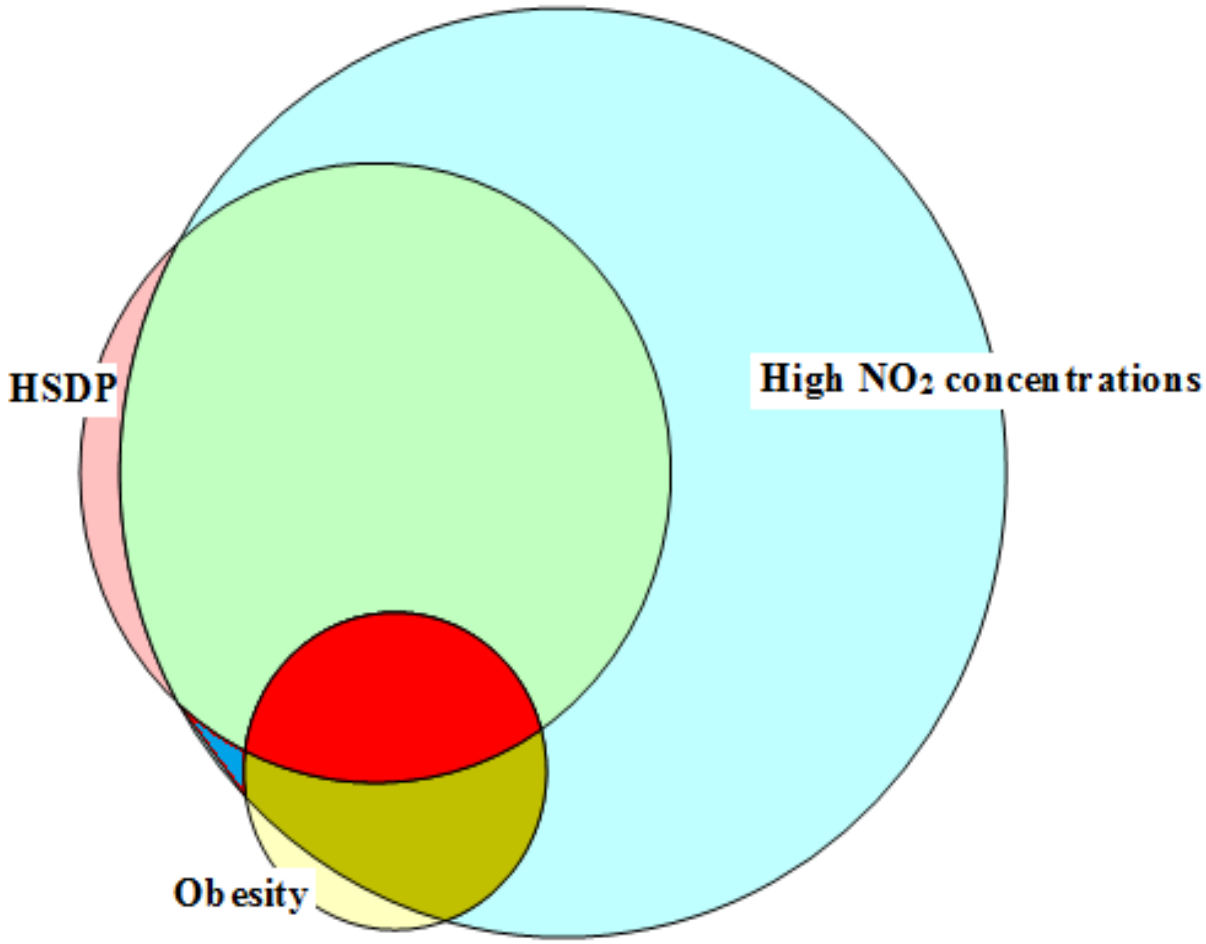
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.80) along with the proportional Venn diagram for childhood obesity in relation to high NOx emissions and HSDP which has been illustrated below the table (Figure 4.20). The Venn diagram shows that the proportion of childhood obesity associated with high NOx emissions was higher when compared to that of household member smoking during pregnancy. The proportion of childhood obesity associated with HSDP and high NOx emissions was lower when compared to that of HSDP alone, and also when compared to high NOx emissions alone.

4.10.10 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NO_2 concentrations and HSDP

Table 4.81 Childhood obesity in relation to high NO_2 concentrations and HSDP

Venn diagram variables	**Prevalence (%)**
High NO_2 concentrations	686/706 (97.2)
HSDP	305/698 (43.7)
Obesity	80/577 (13.9)
HSDP x High NO_2 concentrations	291/295 (98.6)
High NO_2 concentrations x Obesity	73/539 (13.5)
HSDP x Obesity	39/238 (16.4)
Venn diagram variables	**Numbers for Venn diagram**
High NO_2 concentrations	686
HSDP	305
Obesity	80
HSDP ∩ High NO_2 concentrations	291
High NO_2 concentrations ∩ Obesity	73
HSDP ∩ Obesity	39
HSDP ∩ High NO_2 concentrations ∩ Obesity	**39**

Figure 4.21 Childhood obesity in relation to high NO_2 concentrations and HSDP



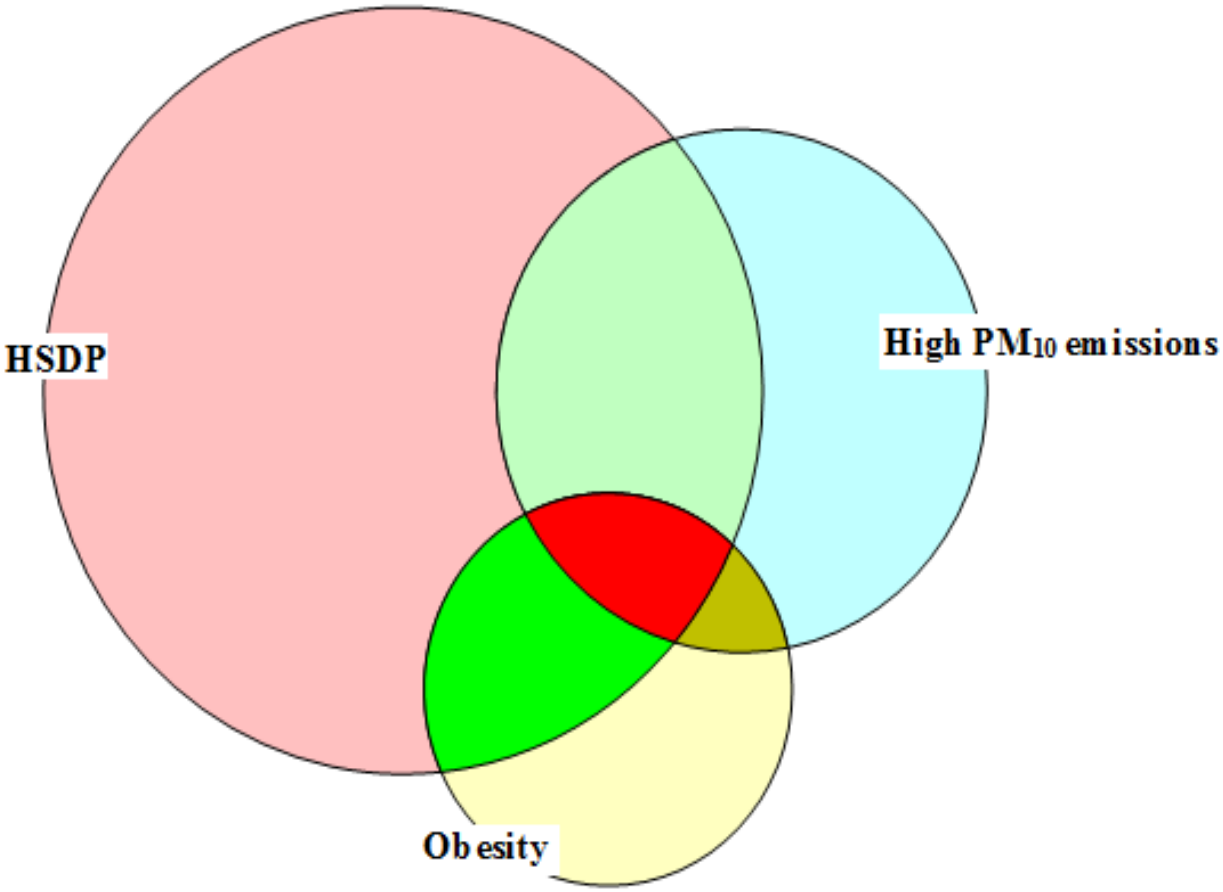
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.81) along with the proportional Venn diagram for childhood obesity in relation to high NO2 concentrations and HSDP which has been illustrated below the table (Figure 4.21). The Venn diagram shows that the proportion of childhood obesity associated with high NO_2 concentrations was higher when compared to that of household member smoking during pregnancy. The proportion of childhood obesity associated with HSDP and high NO_2 concentrations was equal when compared to that of HSDP alone, whereas it was lower when compared to high NO_2 concentrations alone.

4.10.11 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high PM_{10} emissions and HSDP

Table 4.82 Childhood obesity in relation to high PM_{10} emissions and HSDP

Venn diagram variables	**Prevalence (%)**
High PM_{10} emissions	142/668 (21.3)
HSDP	305/698 (43.7)
Obesity	80/577 (13.9)
HSDP x High PM_{10} emissions	68/270 (25.2)
High PM_{10} emissions x Obesity	18/109 (16.5)
HSDP x Obesity	39/238 (16.4)
Venn diagram variables	**Numbers for Venn diagram**
High PM_{10} emissions	142
HSDP	305
Obesity	80
HSDP ∩ High PM_{10} emissions	68
High PM_{10} emissions ∩ Obesity	18
HSDP ∩ Obesity	39
HSDP ∩ High PM_{10} emissions ∩ Obesity	**12**

Figure 4.22 Childhood obesity in relation to high PM_{10} emissions and HSDP



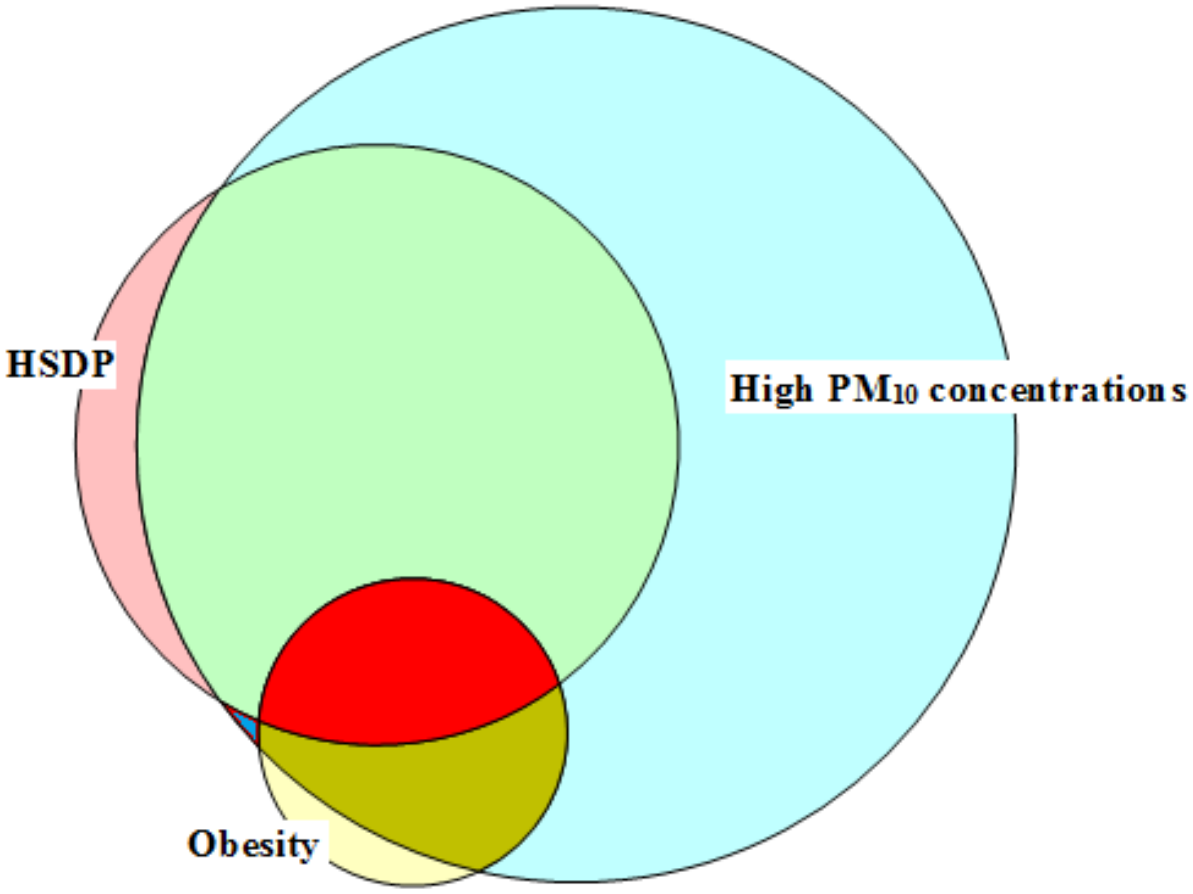
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.82) along with the proportional Venn diagram for childhood obesity in relation to high PM_{10} emissions and HSDP which has been illustrated below the table (Figure 4.22). The Venn diagram shows that the proportion of childhood obesity associated with high PM_{10} emissions was lower when compared to that of household member smoking during pregnancy. The proportion of childhood obesity associated with HSDP and high PM_{10} emissions was lower when compared to that of HSDP or high PM_{10} emissions alone.

4.10.12 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high PM10 concentrations and HSDP

Table 4.83 Childhood obesity in relation to high PM_{10} concentrations and HSDP

Venn diagram variables	**Prevalence (%)**
High PM_{10} concentrations	648/706 (91.8)
HSDP	305/698 (43.7)
Obesity	80/577 (13.9)
HSDP x High PM_{10} concentrations	280/295 (94.9)
High PM_{10} concentrations x Obesity	71/504 (14.1)
HSDP x Obesity	39/238 (16.4)
Venn diagram variables	**Numbers for Venn diagram**
High PM_{10} concentrations	648
HSDP	305
Obesity	80
HSDP ∩ High PM_{10} concentrations	280
High PM_{10} concentrations ∩ Obesity	71
HSDP ∩ Obesity	39
HSDP ∩ High PM_{10} concentrations ∩ Obesity	**37**

Figure 4.23 Childhood obesity in relation to high PM_{10} concentrations and HSDP



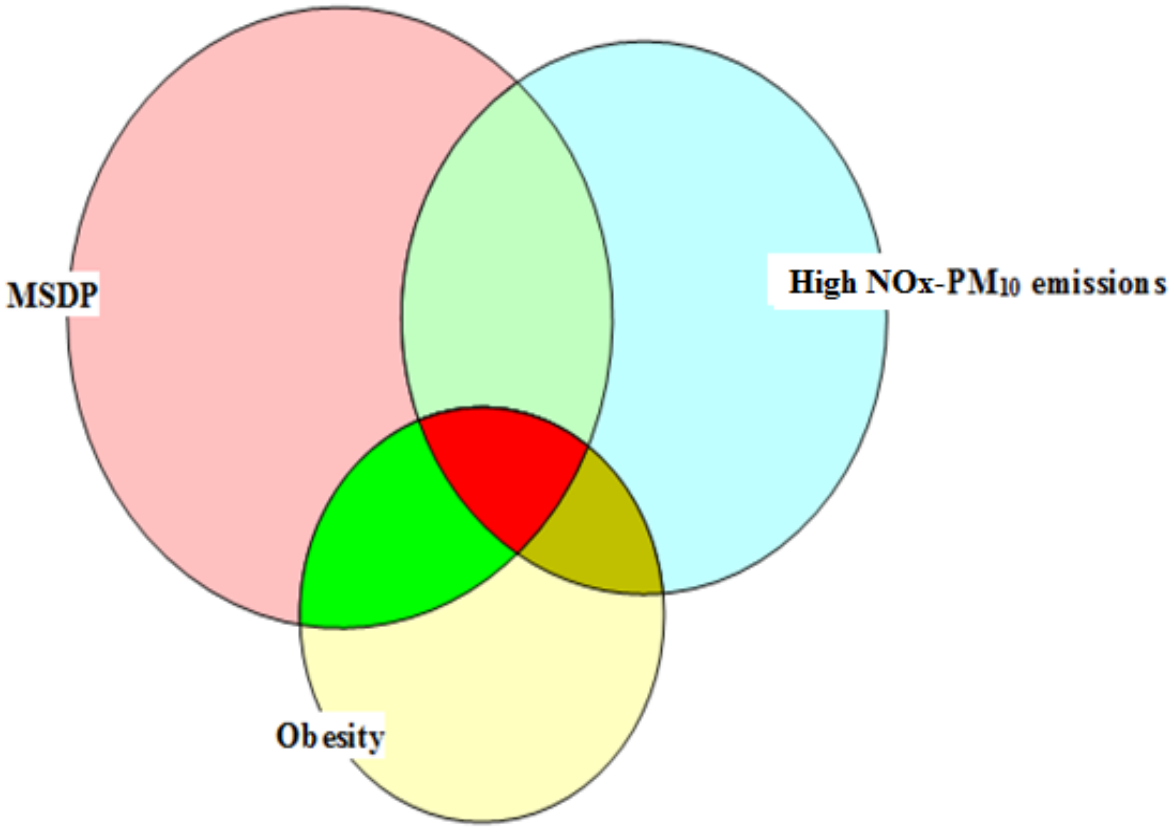
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.83) along with the proportional Venn diagram for childhood obesity in relation to high PM10 concentrations and HSDP which has been illustrated below the table (Figure 4.23). The Venn diagram shows that the proportion of childhood obesity associated with high PM_{10} concentrations was higher when compared to that of household member smoking during pregnancy. The proportion of childhood obesity associated with HSDP and high PM_{10} concentrations was equal when compared to that of HSDP alone, whereas it was lower when compared to high PM_{10} concentrations.

4.10.13 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NOx-PM_{10} emissions and MSDP

Table 4.84 Childhood obesity in relation to high NOx-PM_{10} emissions with MSDP

Venn diagram variables	**Prevalence (%)**
High NOx-PM_{10} emissions	142/668 (21.3)
MSDP	179/704 (25.4)
Obesity	80/577 (13.9)
MSDP x High NOx-PM_{10} emissions	47/158 (29.7)
High NOx-PM_{10} emissions x Obesity	18/109 (16.5)
MSDP x Obesity	28/140 (20.0)
Venn diagram variables	**Numbers for Venn diagram**
High NOx-PM_{10} emissions	142
MSDP	179
Obesity	80
MSDP ∩ High NOx-PM_{10} emissions	47
High NOx-PM_{10} emissions ∩ Obesity	18
MSDP ∩ Obesity	28
MSDP ∩ High NOx-PM_{10} emissions ∩ Obesity	**9**

Figure 4.24 Childhood obesity in relation to high NOx-PM_{10} emissions and MSDP



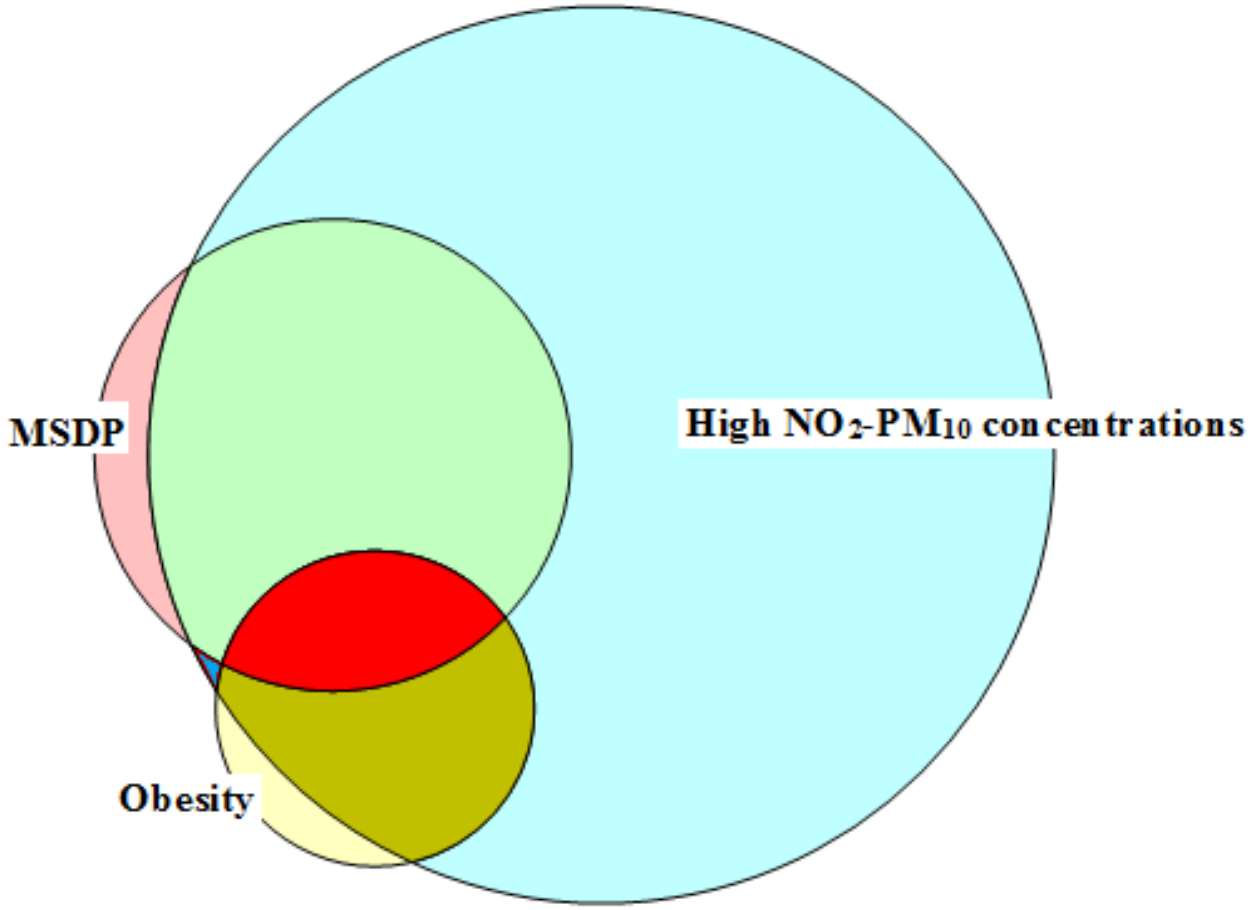
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.84) along with the proportional Venn diagram for childhood obesity in relation to high NOx-PM_{10} emissions and MSDP which has been illustrated below the table (Figure 4.24). The Venn diagram shows that the proportion of childhood obesity associated with MSDP was higher when compared to that of high NOx-PM_{10} emissions. The proportion of childhood obesity associated with combined MSDP and high NOx-PM_{10} emissions was less than obesity due to MSDP, whereas it was equal to obesity due to high NOx-PM10 emissions, when mothers did not smoke.

4.10.14 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NO_2-PM_{10} concentrations and MSDP

Table 4.85 Childhood obesity in relation to high NO_2-PM_{10} concentrations and MSDP

Venn diagram variables	**Prevalence (%)**
High NO_2-PM_{10} concentrations	646/706 (91.5)
MSDP	179/704 (25.4)
Obesity	80/577 (13.9)
MSDP x High NO_2-PM_{10} concentrations	164/172 (95.3)
High NO_2-PM_{10} concentrations x Obesity	71/503 (14.1)
MSDP x Obesity	28/140 (20.0)
Venn diagram variables	**Numbers for Venn diagram**
High NO_2-PM_{10} concentrations	646
MSDP	179
Obesity	80
MSDP ∩ High NO_2-PM_{10} concentrations	164
High NO_2-PM_{10} concentrations ∩ Obesity	71
MSDP ∩ Obesity	28
MSDP ∩ High NO_2-PM_{10} concentrations ∩ Obesity	**27**

Figure 4.25 Childhood obesity in relation to high NO_2-PM_{10} concentrations and MSDP



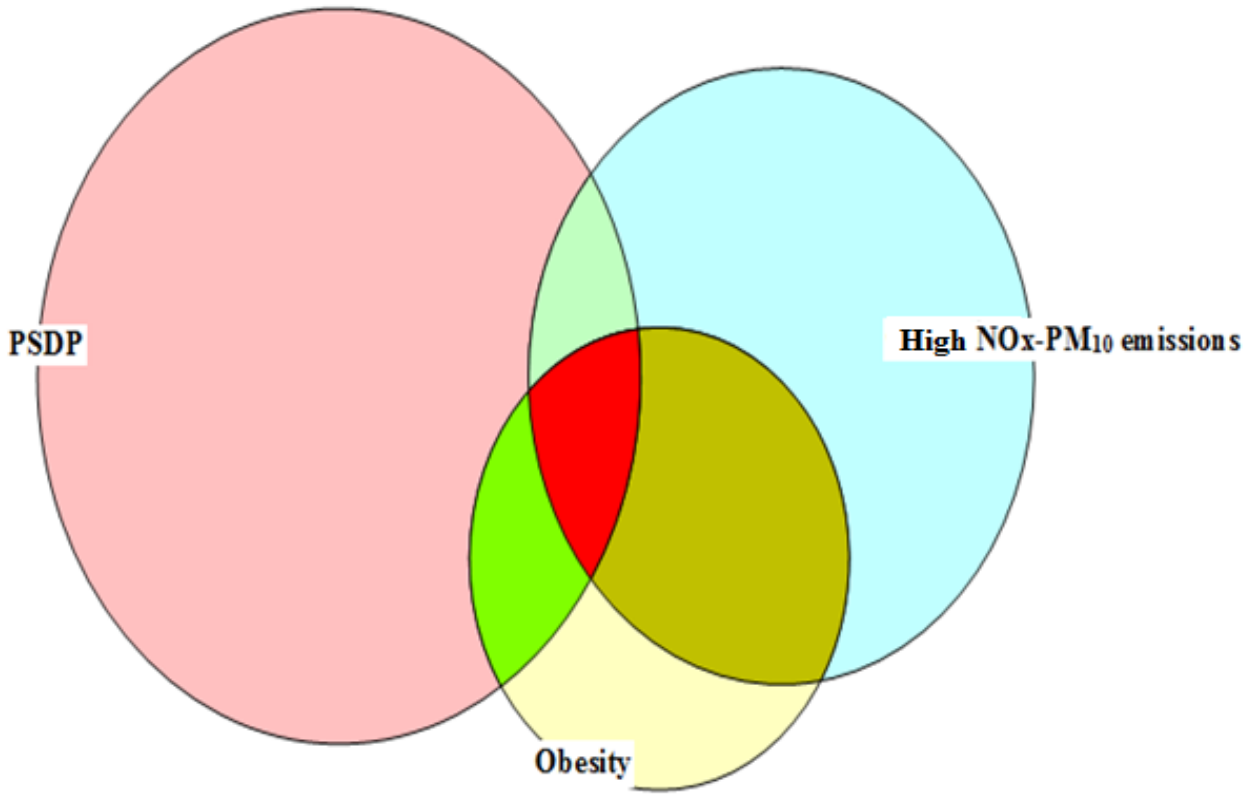
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.85) along with the proportional Venn diagram for childhood obesity in relation to high NO_2-PM_{10} concentrations and MSDP which has been illustrated below the table (Figure 4.25). The Venn diagram shows that the proportion of childhood obesity associated with high NO_2-PM_{10} concentrations was higher when compared to that of maternal smoking during pregnancy. The proportion of childhood obesity associated with MSDP and high NO_2-PM_{10} concentrations was equal when compared to that of MSDP alone, whereas it was lower when compared to high NO_2-PM_{10} concentrations.

4.10.15 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NOx-PM_{10} emissions and PSDP

Table 4.86 Childhood obesity in relation to high NOx-PM_{10} emissions with PSDP

Venn diagram variables	**Prevalence (%)**
High NOx-PM_{10} emissions	142/668 (21.3)
PSDP	202/565 (35.8)
Obesity	80/557 (13.9)
High NOx-PM_{10} emissions x PSDP	18/109 (16.5)
High NOx-PM_{10} emissions x Obesity	49/178 (27.5)
PSDP x Obesity	22/162 (13.6)
Venn diagram variables	**Numbers for Venn diagram**
High NOx-PM_{10} emissions	142
PSDP	202
Obesity	80
High NOx-PM_{10} emissions ∩ PSDP	18
High NOx-PM_{10} emissions ∩ Obesity	49
PSDP ∩ Obesity	22
PSDP ∩ High NOx-PM_{10} emissions ∩ Obesity	**7**

Figure 4.26 Childhood obesity in relation to high NOx-PM_{10} emissions with PSDP



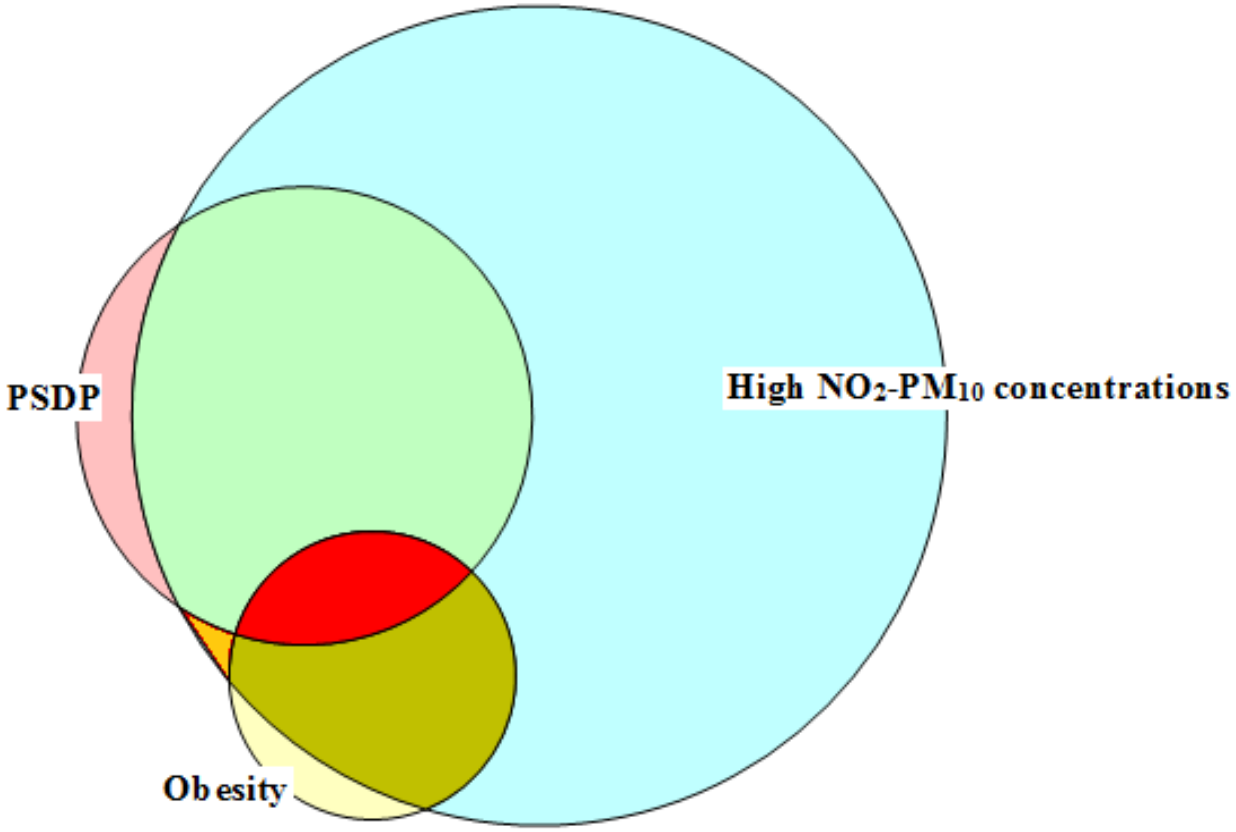
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.86) along with the proportional Venn diagram for childhood obesity in relation to high NOx-PM_{10} emissions and PSDP which has been illustrated below the table (Figure 4.26). The Venn diagram shows that the proportion of childhood obesity associated with high NOx-PM_{10} emissions was higher when compared to that of paternal smoking during pregnancy. The proportion of childhood obesity associated with PSDP and high NOx-PM_{10} emissions was lower when compared to that of PSDP alone, and also when compared to that of high NOx-PM_{10} emissions alone.

4.10.16 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NO_2-PM_{10} concentrations and PSDP

Table 4.87 Childhood obesity in relation to combined high NO_2-PM_{10} concentrations and PSDP

Venn diagram variables	**Prevalence (%)**
High NO_2-PM_{10} concentrations	646/706 (91.5)
PSDP	202/565 (35.8)
Obesity	80/577 (13.9)
PSDP x High NO_2-PM_{10} concentrations	183/194 (94.3)
High NO_2-PM_{10} concentrations x Obesity	71/503 (14.1)
PSDP x Obesity	22/162 (16.2)
Venn diagram variables	**Numbers for Venn diagram**
High NO_2-PM_{10} concentrations	646
PSDP	202
Obesity	80
PSDP ∩ High NO_2-PM_{10} concentrations	183
High NO_2-PM_{10} concentrations ∩ Obesity	71
PSDP ∩ Obesity	22
PSDP ∩ High NO_2-PM_{10} concentrations ∩ Obesity	**21**

Figure 4.27 Childhood obesity in relation to high NO_2-PM_{10} concentrations and PSDP



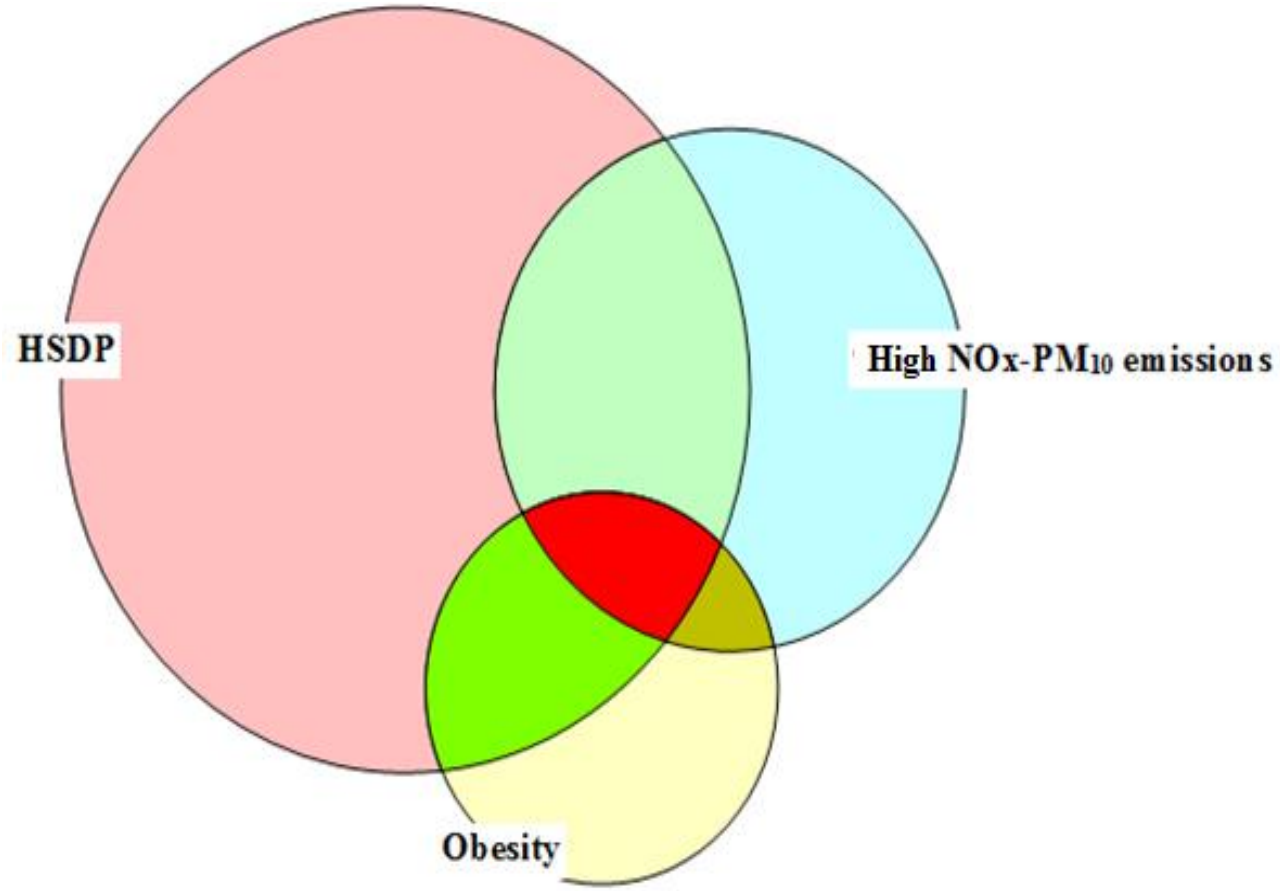
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.87) along with the proportional Venn diagram for childhood obesity in relation to high NO_2-PM_{10} concentrations and PSDP which has been illustrated below the table (Figure 4.27). The Venn diagram shows that the proportion of childhood obesity associated with high NO_2-PM_{10} concentrations was higher when compared to that of paternal smoking during pregnancy. The proportion of childhood obesity associated with PSDP and high NO_2-PM_{10} concentrations was equal when compared to that of PSDP alone, whereas it was lower when compared to high NO_2-PM_{10} concentrations alone.

4.10.17 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to high NOx-PM_{10} emissions and HSDP

Table 4.88 Childhood obesity in relation to high NOx-PM_{10} emissions with HSDP

Venn diagram variables	**Prevalence (%)**
High NOx-PM_{10} emissions	142/668 (21.3)
HSDP	305/698 (43.7)
Obesity	80/577 (13.9)
HSDP x High NOx-PM_{10} emissions	68/270 (25.2)
High NOx-PM_{10} emissions x Obesity	18/109 (16.5)
HSDP x Obesity	39/238 (16.4)
Venn diagram variables	**Numbers for Venn diagram**
High NOx-PM_{10} emissions	142
HSDP	305
Obesity	80
HSDP ∩ High NOx-PM_{10} emissions	68
High NOx-PM_{10} emissions ∩ Obesity	18
HSDP ∩ Obesity	39
HSDP ∩ High NOx-PM_{10} emissions ∩ Obesity	**12**

Figure 4.28 Childhood obesity in relation to high NOx-PM_{10} emissions with HSDP



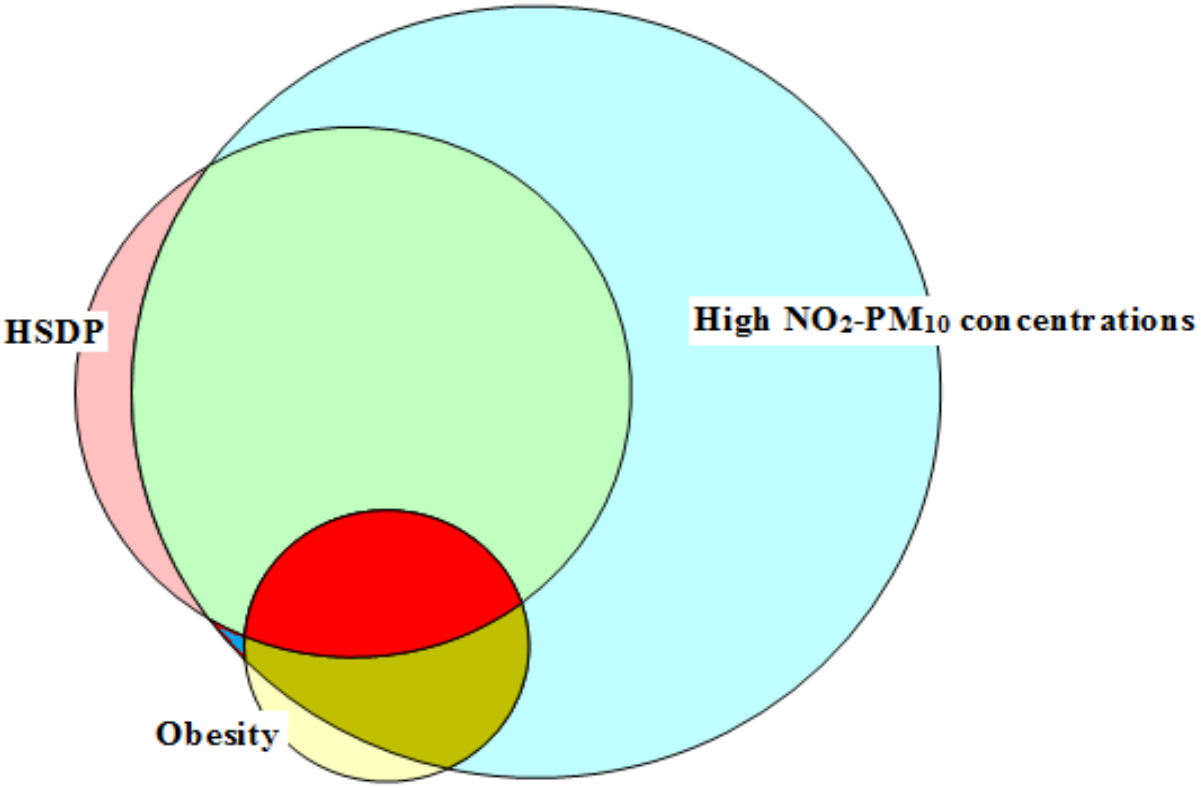
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.88) along with the proportional Venn diagram for childhood obesity in relation to high NOx-PM_{10} emissions and HSDP which has been illustrated below the table (Figure 4.28). The Venn diagram shows that the proportion of childhood obesity associated with high NOx-PM_{10} emissions was lower when compared to that of household member smoking during pregnancy. The proportion of childhood obesity associated with HSDP and high NOx-PM_{10} emissions was lower when compared to that of HSDP alone, and was also was lower when compared to high NOx-PM_{10} emissions alone.

4.10.18 Venn diagram table summary and proportional Venn diagram for childhood obesity in relation to combined high NO_2-PM_{10} concentrations and HSDP

Table 4.89 Childhood obesity in relation to combined high NO_2-PM_{10} concentrations and HSDP

Venn diagram variables	**Prevalence (%)**
High NO_2-PM_{10} concentrations	646/706 (91.5)
HSDP	305/698 (43.7)
Obesity	80/577 (13.9)
HSDP x High NO_2-PM_{10} concentrations	280/295 (94.9)
High NO_2-PM_{10} concentrations x Obesity	71/503 (14.1)
HSDP x Obesity	39/238 (16.4)
Venn diagram variables	**Numbers for Venn diagram**
High NO_2-PM_{10} concentrations	646
HSDP	305
Obesity	80
HSDP ∩ High NO_2-PM_{10} concentrations	280
High NO_2-PM_{10} concentrations ∩ Obesity	71
HSDP ∩ Obesity	39
HSDP ∩ High NO_2-PM_{10} concentrations ∩ Obesity	**37**

Figure 4.29 Childhood obesity in relation to high NO_2-PM_{10} concentrations and HSDP



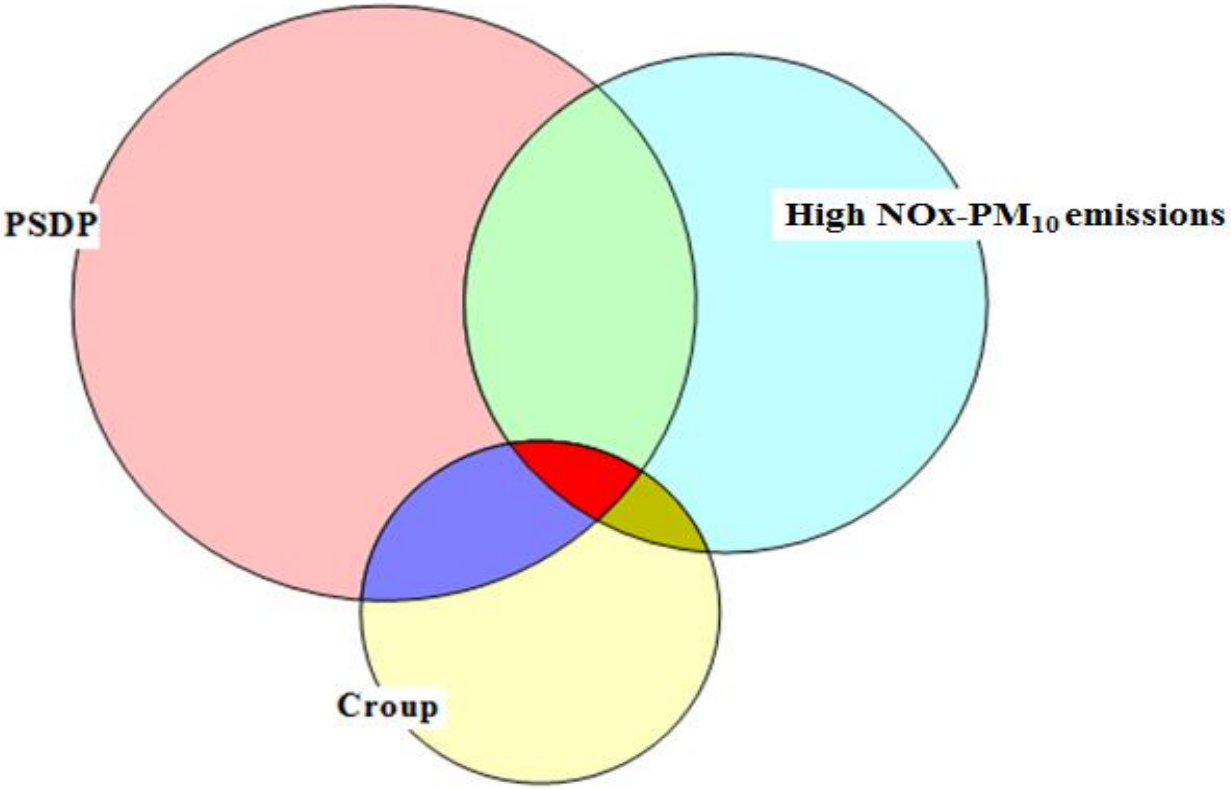
In this analysis, the data used for the Venn diagram are summarised in the table above (Table 4.89) along with the proportional Venn diagram for childhood obesity in relation to high NO_2-PM_{10} concentrations and HSDP which has been illustrated below the table (Figure 4.29). The Venn diagram shows that the proportion of childhood obesity associated with high NO_2-PM_{10} concentrations was higher when compared to that of household member smoking during pregnancy. The proportion of childhood obesity associated with HSDP and high NO_2-PM_{10} concentrations was equal when compared to that of HSDP alone, whereas it was lower when compared to high NO_2-PM_{10} concentrations.

4.10.19 Venn diagram table summary and proportional Venn diagram for childhood croup in relation to combined high NO_2-PM_{10} emissions and PSDP

Table 4.90 Childhood croup in relation to high NO_2-PM_{10} emissions and PSDP

Venn diagram variables	**Prevalence (%)**
High NOx-PM_{10} emissions	142/668 (21.3)
PSDP	202/565 (35.8)
Croup	67/691 (9.7)
PSDP x High NOx-PM_{10} emissions	68/270 (25.2)
High NOx-PM_{10} emissions x Croup	7/138 (5.07)
PSDP x Croup	19/194 (9.80)
Venn diagram variables	**Numbers for Venn diagram**
High NOx-PM_{10} emissions	142
PSDP	305
Croup	67
PSDP ∩ High NOx-PM_{10} emissions	68
High NOx-PM_{10} emissions ∩ Croup	7
PSDP ∩ Croup	19
PSDP ∩ High NOx-PM_{10} emissions ∩ Croup	**1**

Figure 4.30 Childhood croup in relation to high NO_2-PM_{10} emissions and PSDP



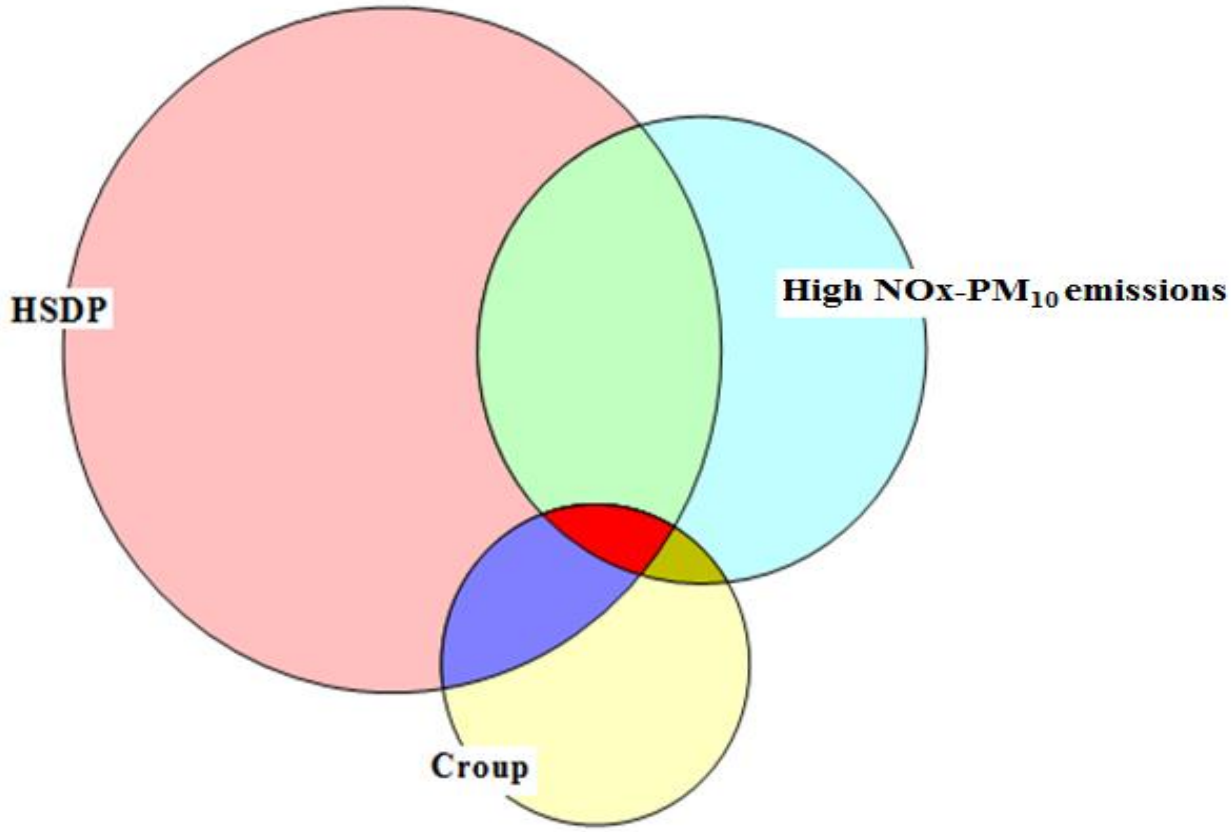
In this analysis, the data used for the Venn diagram are summarised in the table above (Table no 4.90) along with the proportional Venn diagram for childhood croup in relation to high NOx-PM_{10} emissions and PSDP which has been illustrated below the table (Figure 4.30). The Venn diagram shows that the proportion of childhood croup associated with high NOx-PM_{10} emissions was lower when compared to that of paternal smoking during pregnancy. The proportion of childhood croup associated with PSDP and high NOx-PM_{10} emissions was lower when compared to that of PSDP alone, and also when compared to that of high NOx-PM_{10} emissions alone.

4.10.20 Venn diagram table summary and proportional Venn diagram for childhood croup in relation to high NO_2-PM_{10} emissions and HSDP

Table 4.91 Childhood croup in relation to high NO_2-PM_{10} emissions and HSDP

Venn diagram variables	**Prevalence (%)**
High NOx-PM_{10} emissions	142/668 (21.3)
HSDP	305/698 (43.7)
Croup	67/691 (9.7)
HSDP x High NOx-PM_{10} emissions	68/270 (25.2)
High NOx-PM_{10} emissions x Croup	7/138 (5.07)
HSDP x Croup	21/293 (7.17)
Venn diagram variables	**Numbers for Venn diagram**
High NOx-PM_{10} emissions	142
HSDP	305
Croup	67
HSDP ∩ High NOx-PM_{10} emissions	68
High NOx-PM_{10} emissions ∩ Croup	7
HSDP ∩ Croup	21
HSDP ∩ High NOx-PM_{10} emissions ∩ Croup	**2**

Figure 4.31 Childhood croup in relation to high NO_2-PM_{10} emissions and HSDP



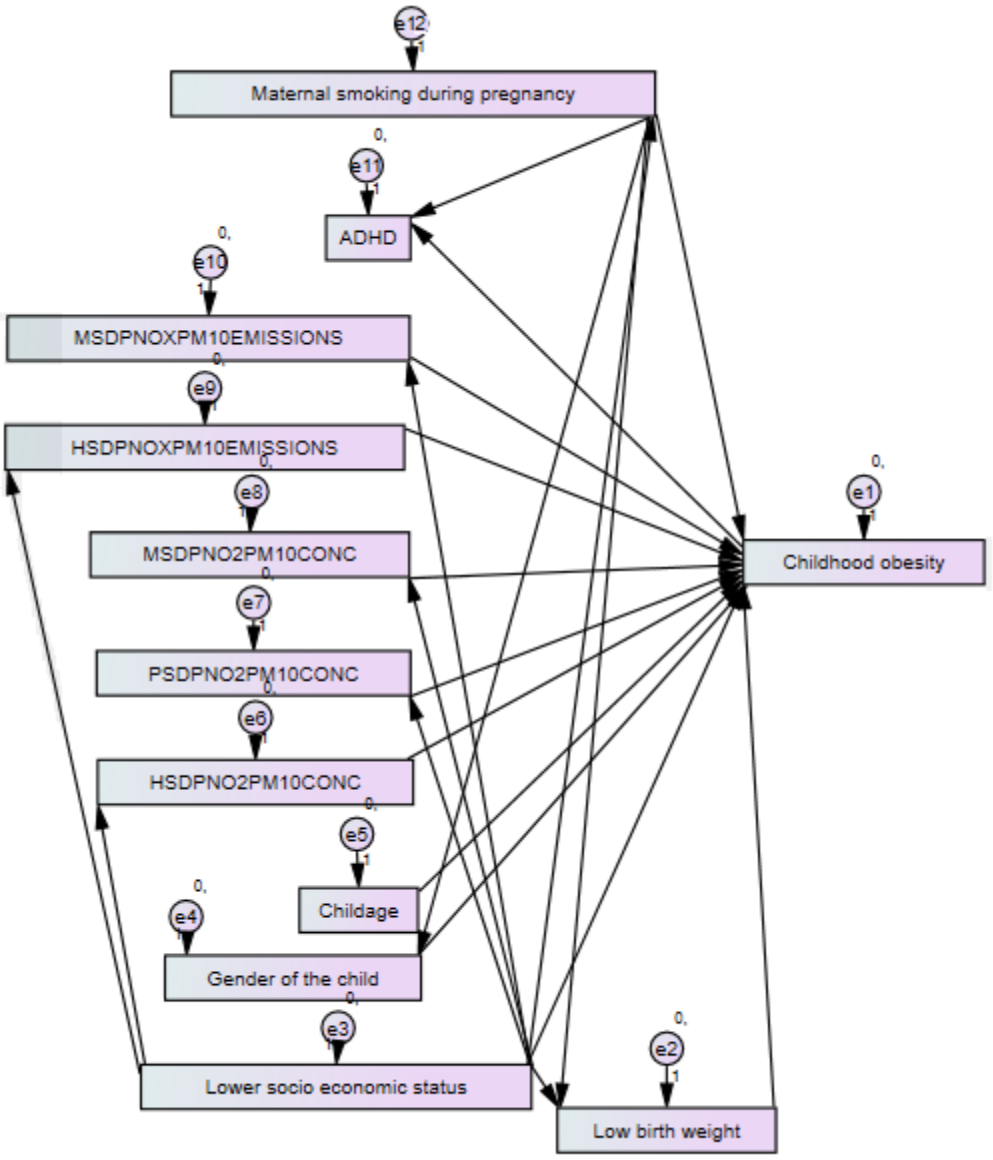
In this analysis, the data used for the Venn diagram are summarised in the table above (Table no 4.91) along with the proportional Venn diagram for childhood croup in relation to high NOx-PM_{10} emissions and HSDP which has been illustrated below the table (Figure 4.31). The Venn diagram shows that the proportion of childhood croup associated with high NOx-PM_{10} emissions was lower when compared to that of household member smoking during pregnancy. The proportion of childhood croup associated with HSDP and high NOx-PM_{10} emissions was lower when compared to that of HSDP alone, and also when compared to that of high NOx-PM_{10} emissions alone.

4.11 Structural equation modelling (SEM) for childhood obesity, ever wheeze, croup and female birth with relevant exposure variables

A trial of basic structural equation modelling was carried out to analyse the data using the SPSS AMOS 20 version and the direct effects of different factors on childhood obesity, ever wheeze symptom, croup and female birth separately. Figures 4.32 – 4.35 illustrate the model tested (Figure 4.32 Childhood obesity ; Figure 4.33 Ever wheeze ; Figure 4.34 Croup ; Figure 4.35 female birth) and tables 4.92 to 4.95 summarise the standardised estimates (Table 4.92 Childhood obesity ; Table 4.93 Ever wheeze ; Table 4.94 Croup ; Table 4.95 female birth). All the SEM models were created based on inclusion of variables which were significant from univariate analysis ($p<0.05$), and which were included in the logistic regression analysis. Although this model appeared to fit, no goodness of fit statistics could be computed suggesting that at least some of the estimated coefficients (associations) might not be reliable. Since this was a trial of SEM analysis for creating models based on the data available further details are not included in results section as there were problems with model fit. Although the model showed some significant associations reliable conclusions could not be drawn.

4.11.1 Structural equation modelling for childhood obesity

Figure 4.32 Structural equation model path diagram for childhood obesity*



***corresponding values included in the table below**

Path coefficient: 1
e: error variance or residual

Table 4.92 Standardised estimates using structural equation modelling for childhood obesity

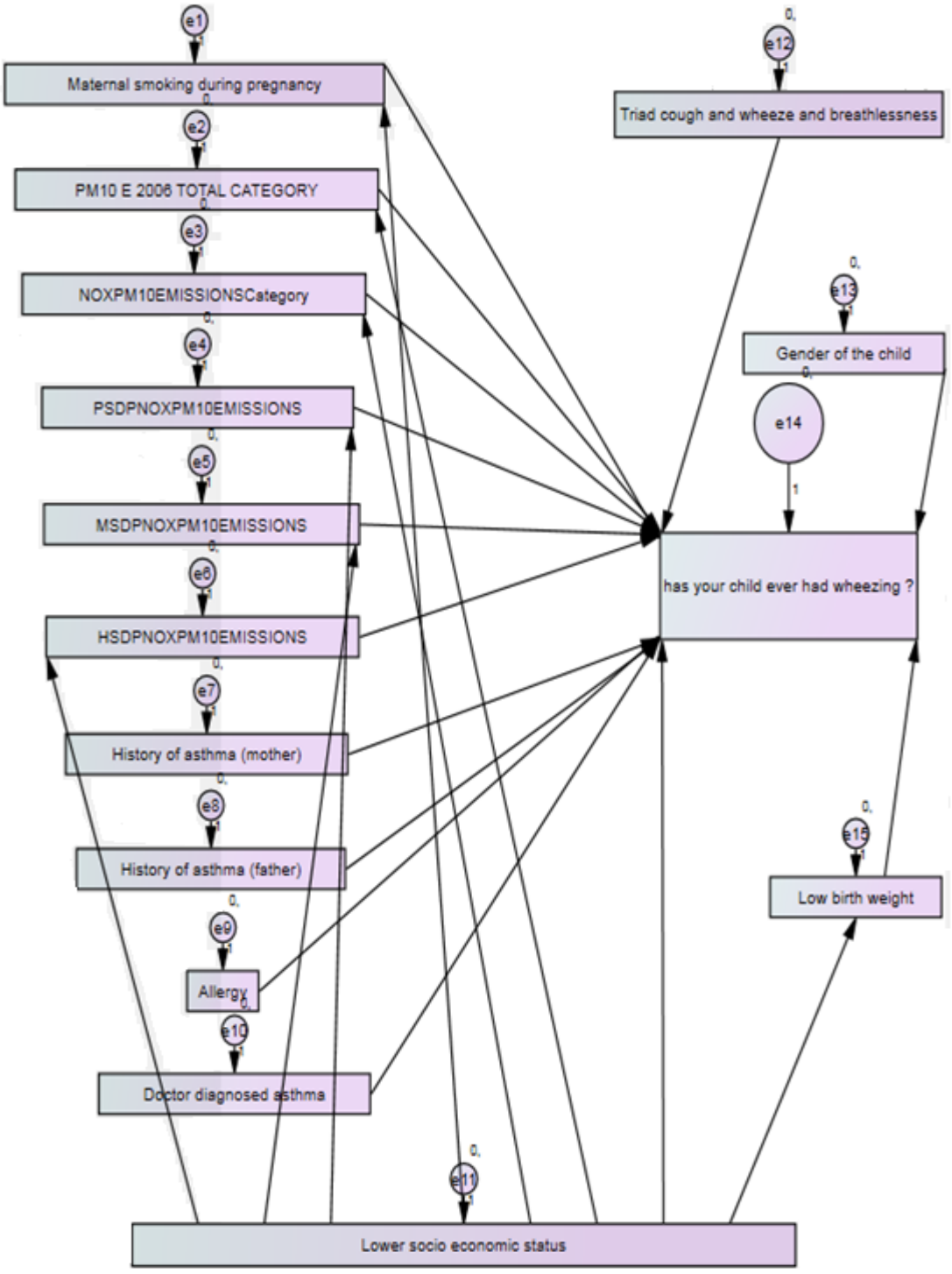
Outcome		Exposure	Estimate	S.E.	C.R.	P value
MSDP	<---	Upperquartile	.195	.062	3.168	.002
HSDPNOXPM10EMISSIONS	<---	Upperquartile	.233	.076	3.060	.002
HSDPNO2PM10CONC	<---	Upperquartile	.877	.045	19.476	***
PSDPNO2PM10CONC	<---	Upperquartile	.840	.065	12.831	***
MSDPNO2PM10CONC	<---	Upperquartile	.926	.063	14.688	***
MSDPNOXPM10EMISSIONS	<---	Upperquartile	.133	.056	2.374	.018
LBW	<---	Upperquartile	-.082	.040	-2.080	.038
LBW	<---	MSDP	.043	.024	1.748	.080
Gender	<---	MSDP	-.166	.043	-3.880	***
BMICLOB	<---	MSDPNOXPM10EMISSIONS	.049	.048	1.007	.314
BMICLOB	<---	MSDPNO2PM10CONC	-.192	.081	-2.364	.018
BMICLOB	<---	PSDPNO2PM10CONC	.127	.077	1.656	.098
BMICLOB	<---	Childage	.009	.006	1.431	.152
BMICLOB	<---	HSDPNO2PM10CONC	.147	.080	1.838	.066
BMICLOB	<---	LBW	.005	.044	.124	.901
BMICLOB	<---	Gender	.019	.023	.823	.410
BMICLOB	<---	Upperquartile	-.056	.135	-.411	.681
BMICLOB	<---	HSDPNOXPM10EMISSIONS	.001	.043	.013	.990
BMICLOB	<---	MSDP	.064	.028	2.316	.021
ADHD	<---	MSDP	.021	.015	1.417	.157
ADHD	<---	BMICLOB	.081	.020	4.033	***

MSDP: Maternal smoking during pregnancy
LBW; Low birthweight
ADHD: Attention Deficit Hyperactivity Disorder
MSDPNOXPM10Emissions: Maternal smoking during pregnancy combined with NOx and PM_{10} emissions
MSDP NO2PM10Conc: Maternal smoking during pregnancy combined with NO_2 and PM_{10} concentrations
PSDP NO2PM10Conc: Paternal smoking during pregnancy combined with NO_2 and PM_{10} concentrations
HSDPNOXPM10Emissions: Household member smoking during pregnancy combined with NOx and PM_{10} emissions
HSDP NO2PM10Conc: Household member smoking during pregnancy combined with NO_2 and PM_{10} concentrations
Upper Quartile: Lower socio economic status (Townsend score 4 to 12)
BMICLOB: Childhood obesity
Child age; Gender: Male
SE: Standardised estimates
CR: Critical ratio
CR>1.96 for regression weight, path significant at 0.05 level

In this analysis, the standardised estimates using structural equation modelling for childhood obesity are summarised in table no 4.92 and the corresponding structural equation model path diagram illustrated above the table (Figure 4.32). Childhood obesity in relation to combined air pollutant category with pregnancy smoking exposure category did not show any significant association, except for obesity in relation to maternal smoking during pregnancy combined with high NO_2-PM_{10} concentrations, eventhough no goodness of fit statistics could be computed.

4.11.2 Structural equation modelling for ever wheeze

Figure 4.33 Structural equation model path diagram for childhood ever wheeze*



***corresponding values included in the table below**

Path coefficient: 1
e: error variance or residual

Table 4.93 Standardised estimates using structural equation modelling for childhood everwheeze

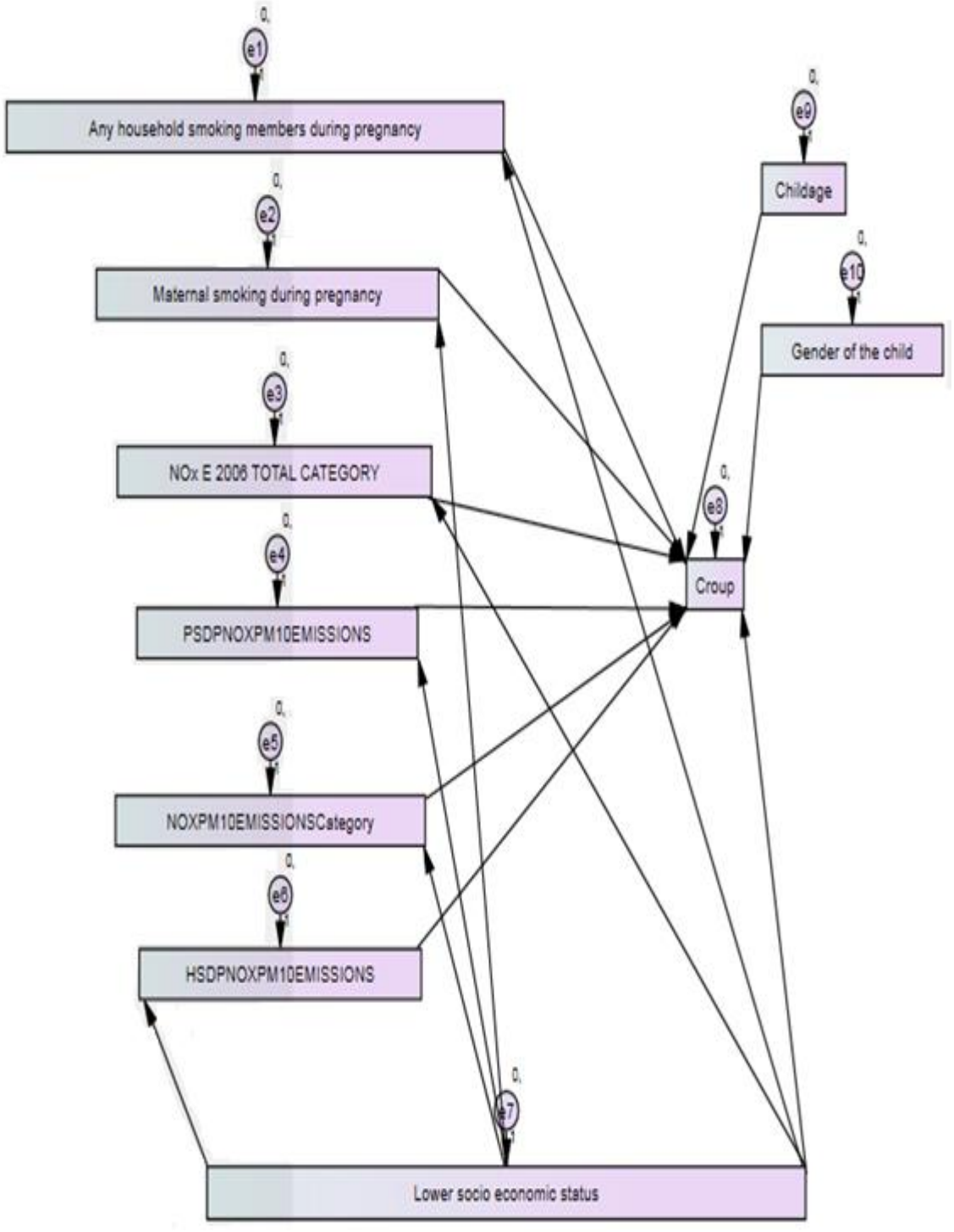
Outcome		Exposure	Estimate	S E	C R	P value
LBW	<---	Upperquartile	-.069	.041	-1.700	.089
MSDP	<---	Upperquartile	.179	.064	2.805	.005
PM102006TOTALCAT	<---	Upperquartile	.282	.060	4.670	***
NOXPM10EMISSIONSCategory	<---	Upperquartile	.282	.060	4.670	***
PSDPNOXPM10EMISSIONS	<---	Upperquartile	.216	.075	2.867	.004
MSDPNOXPM10EMISSIONS	<---	Upperquartile	.168	.057	2.923	.003
HSDPNOXPM10EMISSIONS	<---	Upperquartile	.279	.077	3.621	***
wheezing	<---	triadcwb	.017	.027	.617	.537
wheezing	<---	MSDPNOXPM10EMISSIONS	.007	.053	.138	.891
wheezing	<---	HSDPNOXPM10EMISSIONS	.014	.046	.294	.769
wheezing	<---	LBW	.031	.047	.665	.506
wheezing	<---	NOXPM10EMISSIONSCategory	.029	.032	.900	.368
wheezing	<---	PM102006TOTALCAT	.029	.032	.900	.368
wheezing	<---	PSDPNOXPM10EMISSIONS	.047	.053	.890	.374
wheezing	<---	Upperquartile	-.080	.055	-1.461	.144
wheezing	<---	Allergy	.054	.033	1.643	.100
wheezing	<---	dadasthma	.110	.046	2.405	.016
wheezing	<---	mumasthma	.004	.037	.119	.905
wheezing	<---	DDA	.574	.032	17.682	***
wheezing	<---	Gender	.049	.025	1.985	.047
wheezing	<---	MSDP	.083	.029	2.832	.005

MSDP: Maternal smoking during pregnancy
LBW; Low birthweight
MSDPNOXPM10Emission: Maternal smoking during pregnancy combined with NOx and PM_{10} emission
PSDPNOXPM10Emission: Paternal smoking during pregnancy combined with NOx and PM_{10} emission
HSDPNOXPM10Emission: Household member smoking during pregnancy combined with NOx and PM_{10} emission
PM102006TOTALCAT: High PM10 emission: PM_{10} emission >5 tonnes per annum
NOXPM10EMISSIONSCategory: High NOx-PM_{10} emission: NOx emission >10 and PM_{10} emission > 5 tonnes per annum
Upper Quartile: Lower socio economic status (Townsend score 4 to 12)
Gender: Male
SE: Standardised estimates; CR: Critical ratio
CR>1.96 for regression weight, path significant at 0.05 level

In this analysis, the standardised estimates using structural equation modelling for ever wheeze are summarised in table no 4.93 and the corresponding structural equation model path diagram illustrated above the table (Figure 4.33). Childhood ever wheeze in relation to combined air pollutant category with pregnancy smoking exposure category did not show any significant association, eventhough no goodness of fit statistics could be computed.

4.11.3 Structural equation modelling for croup

Figure 4.34 Structural equation model path diagram for childhood croup*



***corresponding values included in the table below**

Path coefficient: 1
e: error variance or residual

Table 4.94 Standardised estimates using structural equation modelling for childhood croup

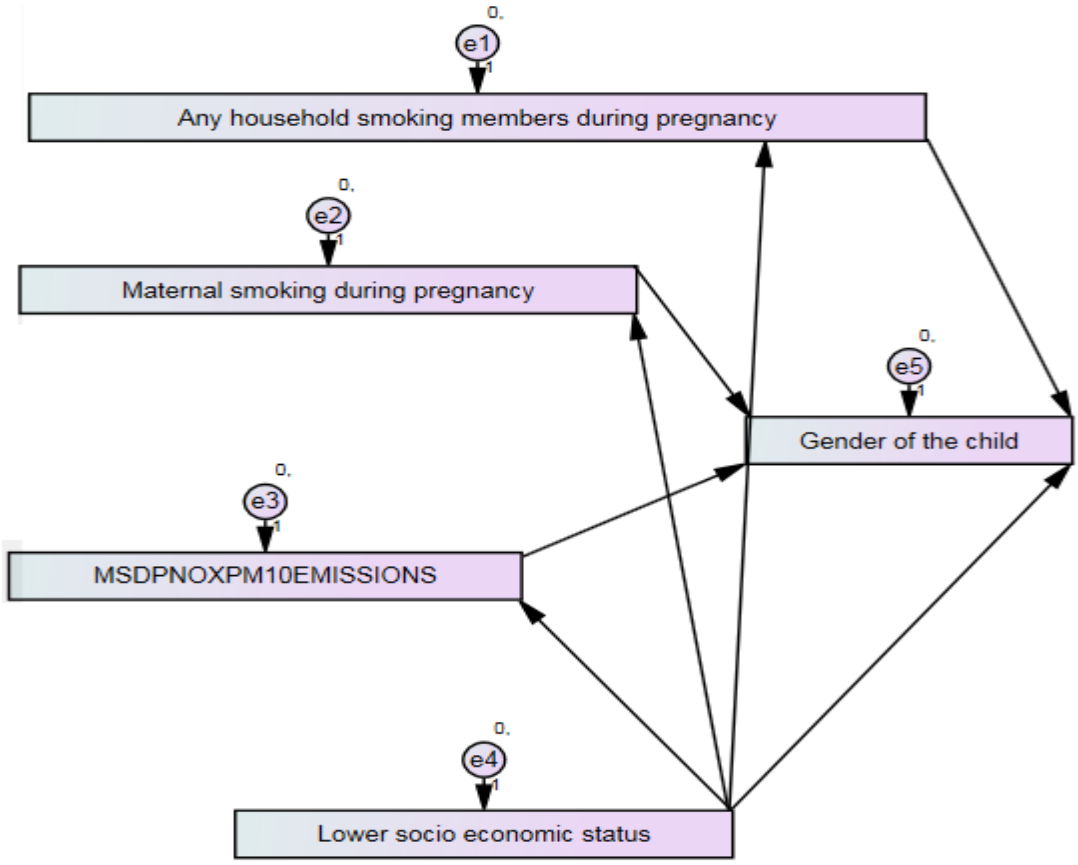
Outcome		Exposure	Estimate	S E	C R	P value
MSDP	<---	Upperquartile	.174	.065	2.695	.007
HSDPNOXPM10EMISSIONS	<---	Upperquartile	.259	.078	3.312	***
NOXPM10EMISSIONSCategory	<---	Upperquartile	.238	.061	3.879	***
PSDPNOXPM10EMISSIONS	<---	Upperquartile	.202	.076	2.653	.008
NOE2006TOTALCAT	<---	Upperquartile	.234	.070	3.343	***
HSDP	<---	Upperquartile	.222	.073	3.029	.002
Croup	<---	Upperquartile	.075	.048	1.555	.120
Croup	<---	HSDPNOXPM10EMISSIONS	.010	.041	.242	.809
Croup	<---	NOXPM10EMISSIONSCategory	-.020	.029	-.703	.482
Croup	<---	PSDPNOXPM10EMISSIONS	-.045	.046	-.970	.332
Croup	<---	NOE2006TOTALCAT	-.057	.025	-2.273	.023
Croup	<---	MSDP	.026	.026	.995	.320
Croup	<---	HSDP	-.051	.023	-2.202	.028
Croup	<---	Childage	-.002	.006	-.264	.792
Croup	<---	Gender	.024	.022	1.075	.282

MSDP: Maternal smoking during pregnancy
HSDP: Household member smoking during pregnancy
LBW; Low birthweight
PSDPNOXPM10Emissions: Paternal smoking during pregnancy combined with NOx and PM_{10} emission
HSDPNOXPM10Emissions: Household member smoking during pregnancy combined with NOx and PM_{10} emission
NOX2006TOTALCAT: High NO_X emission: NO_X emission >10 tonnes per annum
NOXPM10EMISSIONSCategory: High NOx-PM_{10} Emission: NOx emission >10 and PM_{10} emission > 5 tonnes per annum
Upper Quartile: Lower socio economic status (Townsend score 4 to 12)
Gender: male
SE: Standardised estimates
CR: Critical ratio
CR>1.96 for regression weight, path significant at 0.05 level

In this analysis, the standardised estimates using structural equation modelling for childhood croup are summarised in table no 4.94 and the corresponding structural equation model path diagram illustrated above the table (Figure 4.34). Childhood croup in relation to combined air pollutant category with pregnancy smoking exposure category did not show any significant association, eventhough no goodness of fit statistics could be computed.

4.11.4 Structural equation modelling for female birth (male births used in figure and table)

Figure 4.35 Structural equation model path diagram for gender (male)*



***corresponding values included in the table below**

Path coefficient: 1
e: error variance or residual

Table 4.95 Standardised estimates using structural equation modelling for gender (male)

Outcome		Exposure	Estimate	S E	C R	P value
MSDPNOXPM10EMISSIONS	<---	Upperquartile	.135	.059	2.284	.022
MSDP	<---	Upperquartile	.182	.065	2.801	.005
HSDP	<---	Upperquartile	.220	.074	2.978	.003
Gender	<---	MSDP	-.126	.043	-2.917	.004
Gender	<---	HSDP	-.086	.038	-2.270	.023
Gender	<---	MSDPNOXPM10EMISSIONS	.001	.077	.007	.994
Gender	<---	Upperquartile	.013	.075	.178	.859

MSDP: Maternal smoking during pregnancy
HSDP: Household member smoking during pregnancy
MSDPNOXPM10Emissions: Maternal smoking during pregnancy combined with NOx and PM_{10} emissions
Upper Quartile: Lower socio economic status (Townsend score 4 to 12)
Gender: male
SE: Standardised estimates
CR: Critical ratio
CR>1.96 for regression weight, path significant at 0.05 level

In this analysis, the standardised estimates using structural equation modelling for male births are summarised in table no 4.95 and the corresponding structural equation model path diagram illustrated above the table (Figure 4.35). Male births in relation to combined air pollutant category with pregnancy smoking exposure category did not show any significant association, eventhough no goodness of fit statistics could be computed.

4.12. Spatial mapping

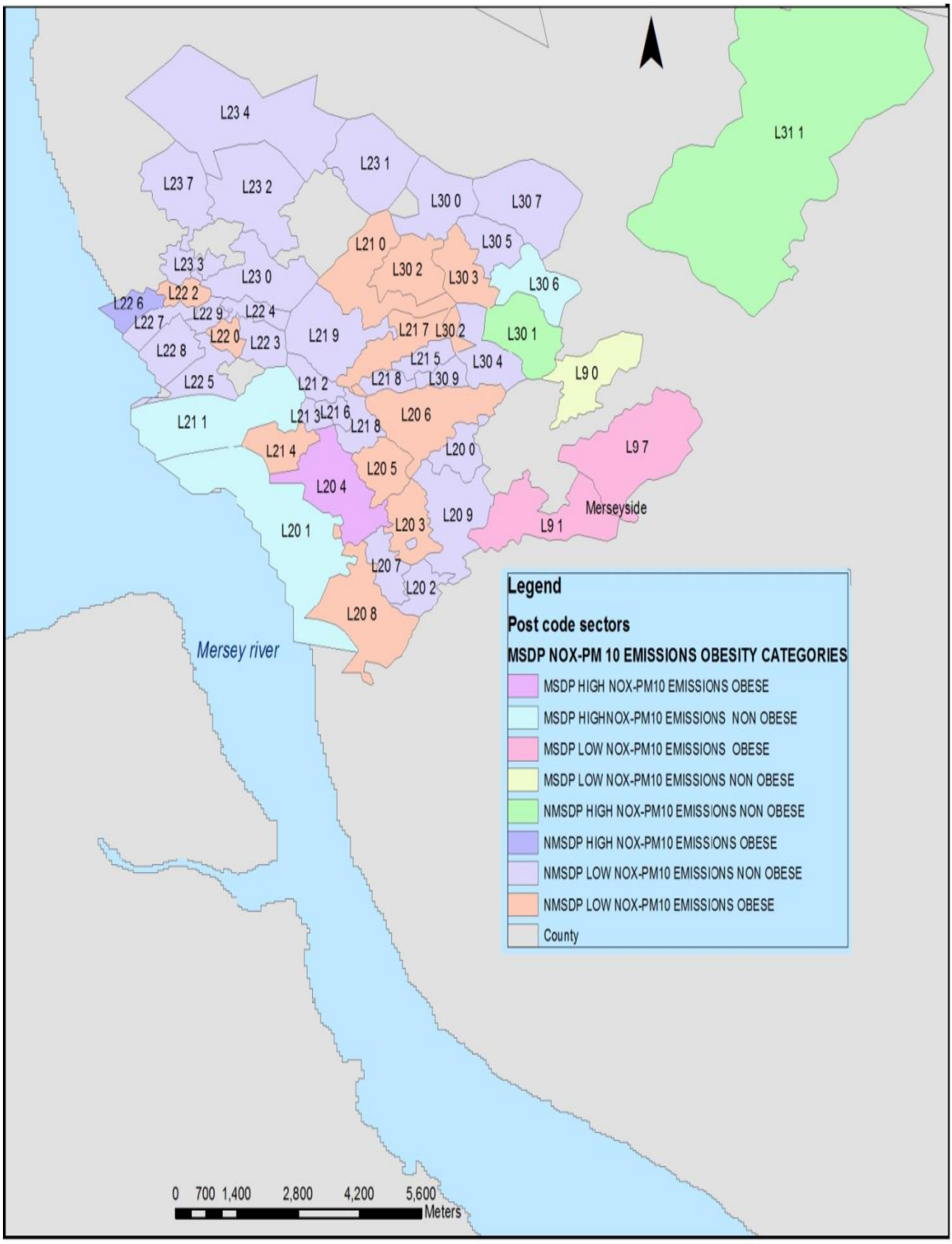
4.12.1 Spatial mapping of childhood obesity and childhood croup in relation to relevant combined air pollution and pregnancy smoking exposure

For the spatial maps, two outcomes - childhood obesity and croup were selected as they both showed clear significant association with combined smoking and air pollution categories after the logistic regression analysis. Modes of childhood obesity in relation to pregnancy smoking and air pollution categories and croup in relation to paternal smoking during the mother's pregnancy and air pollution categories have been used as corresponding spatial modes for spatial mapping illustrated by different colour codes.

4.12.1.1 Spatial mapping of childhood obesity categories in relation to maternal smoking during pregnancy and air pollution

Figure 4.36 illustrates the spatial mapping of childhood obesity categories in relation to combined maternal smoking during pregnancy – NOx-PM_{10} categories for the 10 Liverpool schools. The spatial map shows that obesity associated with combined maternal smoking during pregnancy and high NOx-PM_{10} emissions was higher among school children who resided in postcode sector area L20 4) corresponding to a predominantly lower socio economic area.

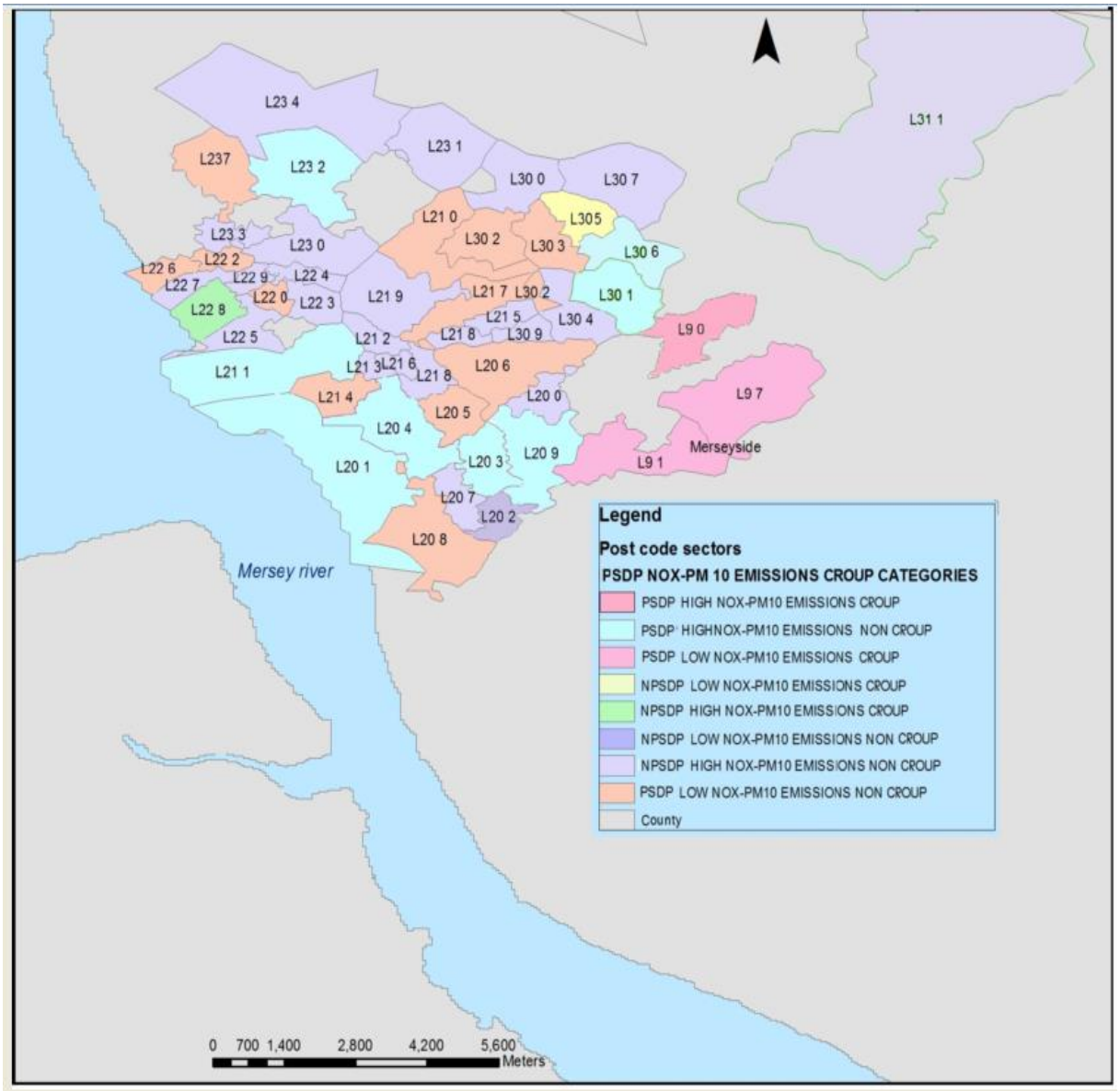
Figure 4.36 Map of obesity, maternal pregnancy smoking and air pollution (Liverpool schools)



4.12.1.2 Spatial mapping of childhood croup categories in relation to paternal smoking during pregnancy and air pollution

Figure 4.37 illustrates the spatial mapping of childhood croup categories in relation to combined paternal smoking during pregnancy – NOx-PM_{10} emission categories for the 10 Liverpool schools. The spatial map shows that croup associated with combined paternal smoking during pregnancy with high NOx-PM_{10} emissions was lower among school children who resided in postcode sectors of south west areas of Liverpool (L20 1, L21 1 and L20 4) which corresponded to predominantly lower socio economic areas.

Figure 4.37 Map of croup, paternal smoking during pregnancy and air pollution (Liverpool schools)



CHAPTER 5

DISCUSSION

5.1 Introduction to discussion

This thesis evaluated birth and child health outcomes of air pollution and smoking exposures during pregnancy by linking health outcomes available in a large community data set with concurrent air pollution data for the same geographic area. This study is unique and relevant as there are no other studies previously conducted, which have looked into multiple birth and child health outcomes in relation to both air pollution and pregnancy smoking exposure among primary school children, after linkage of two different datasets - air pollution data with child health data with common variables from the same area and collected during the same period, even though there has been a previous prospective investigation into the joint obesogenic effects of tobacco smoke and air pollution exposure among school children (McConnell et al, 2015). The findings from that study did provide some insights into possible associations underlying air pollution and pregnancy exposures and adverse child health outcomes and their interactions. The present study was conducted in the Liverpool, Sefton Metropolitan Area. A total of 792 school children aged 5-11 years from ten primary schools from South Sefton, Liverpool were included in this study, which was completed using a parent-completed questionnaire survey. These schools were selected as they were located predominantly in areas of lower socio-economic status and situated within a three mile radius of each other (Rizwan et al, 2004). The presence of air pollution and smoking as a combined exposure could have important public health and clinical impacts, as these exposures are quite common and needs to be considered as part of any policy formulation related to air pollution control and smoking cessation program.

In this chapter, the factors related to assessment of the impact of air pollution exposure, the burden of air pollution and pregnancy smoking exposure in the selected study area, methodological issues, and parental compliance for questionnaire completion along with selection bias, study characteristics, birth and child health outcomes in relation to air pollution and pregnancy smoking exposure have been discussed. There is also discussion about the results obtained from data analysis using different methods along with some of the possible mechanisms underlying the significant association between relevant child health outcomes in relation to combined air pollution and pregnancy smoking exposure. Different analytical methods including visual display of the data in the form of Venn diagrams and spatial mapping based on the results have been discussed which could be used to motivate the public as part of

health promotion activities to take part in smoking cessation and air pollution prevention programs thus helping to provide a better and safe environment for children.

5.2 Factors related to assessment of the impact of air pollution exposures

5.2.1 Establishing the burden of air pollution and pregnancy smoking exposure in the study area

Previous studies of the factors influencing the respiratory health of children in the Sefton area of Merseyside, including airborne dust, were reported twenty years ago (Milligan et al, 1994). The problem of coal air dust pollution in the Liverpool Docks in the 1990's resulting from large scale coal handling activities and increased respiratory symptoms among primary school children attending the schools near to these areas, which led to Merseyside Child Health Surveys have been reported in detail previously (Kelly et al, 1995; Koshy et al, 2010a, Koshy et al, 2010b; Koshy et al, 2011a, Koshy et al, 2011b; Delpisheh et al, 2006b, Delpisheh et al, 2006c; Delpisheh, 2006d, Delpisheh et al, 2007, Delpisheh et al, 2008b; Rizwan et al, 2007; Koshy et al, 2012a, Koshy et al, 2012b). The burden of pregnancy smoking exposures and birth and child health outcomes and trends in heavy smoking in Liverpool have been reported in these studies previously as part of the Merseyside Child health surveys conducted in 1991, 1993, 1998 and 2006 and have already been published in the form of six papers in peer reviewed journals and included as part of my PhD thesis published in 2012 (Koshy et al, 2012b), so will not be discussed here. Although birth and child health outcomes in relation to pregnancy smoking in these areas had been previously assessed, the air pollution indicators or their combined effects with pregnancy smoking exposure on birth and child health outcomes were not included, as it was not available for analysis to establish the overall burden of air pollution and this project helped with further research by extending the analysis to include data on concurrent exposures to additional airborne emissions of air pollutants (NO_x emissions, NO_2 concentrations, PM_{10} emissions and PM_{10} concentrations).

5.3 Methodological issues

5.3.1 Parental compliance for questionnaire completion and selection bias

Parental questionnaires like many other evaluation methods have been very commonly used previously in schools for collection of data. Even though many of the parents may answer the questionnaires promptly, they might just answer some of the questions superficially, in case if it takes a long time to complete. Distribution and collection of questionnaires in schools has been shown to yield good returns compared to postal surveys, even though frequent visits to schools and motivation of parents and school teachers are needed occasionally. The parental questionnaire compliance for the original study which included 15 schools was 30.3% for the 2006 survey, the lowest of all the surveys conducted from 1991 to 2006 as part of the Merseyside Respiratory Child Health Surveys. The previous studies reported parental questionnaire compliances of 92% (1872/2035) in 1991, 87.4% (3746/4288) in 1993, 78.1% (1964/2514) in 1998 and 30.3% (1074/3540) in 2006. However, the average parental questionnaire compliance for the present study, which included only 10 schools was 32.9% (792/2405), which was slightly higher than the compliance reported for 15 schools. The same efforts to enrol child absentees were used for all these surveys, but despite this systematic and considerable effort, good compliance was not maintained. One of the reasons for decrease in compliance for the 2006 survey could be due to reduced concern among the parents after pollution control measures which had been taken by the Health Authority and Borough Council to reduce environmental exposures in the Bootle dock area. Increased frequency of questionnaire surveys occurring in schools related to various audits, monitoring and evaluation activities, lack of available time for employed parents, lengthy questionnaires, changes in population demographics, as well as changing attitudes of respondents to sensitive questions could be some of the other possible reasons. (Iglesias et al, 2000; Rolstad et al, 2011). The possibility of selection bias needs to be considered particularly as compliance with the 2006 survey was lower than in previous surveys and had been showing a progressive decreasing trend from 1991 to 2006. Although the response rate in 2006 was low, the sample still represented the same primary school populations, the same age group of children, and the same monthly survey period (September to December). This should reduce some sources of variations due to seasonal or locational influences, which could act as important confounding factors, when data is collected during different seasons and from different locations.

In view of the increasing constraints on schools and parents it is difficult to achieve high community survey compliance. The same efforts to enrol child absentees were used in all these Merseyside Community Surveys, but despite these efforts, sustained high compliance was not attained. Socio-economic status was uniform across all surveys with little change in ethnic composition. Socio-economic status assessed by mean Townsend scores was comparable with the previous surveys. The same survey instruments, questionnaire and core questions, methodology and schools were used across all the surveys as part of the follow up ruling out the possibility of change in compliance due to methodological differences. Amongst the respondents, changing response rates to the same question provided some insights into questionnaire compliance (Koshy et al, 2012). Parental compliance still remains an emerging problem in school based surveys.

5.3.2 Missing values and school absenteeism

Missing values or responses are a recurrent problem in questionnaire based surveys, which needs to be addressed. Missing values were analysed by complete case analysis. Complete case analysis is the most common method used and excludes all cases in which any of the outputs are missing and uses the available data for analysis and for final results. Even though this method provides an accurate assessment of the results based on the available data and are representative of the original sample, this could also limit the amount of information available for analysis, if the data has got more missing values. The same method for missing values were used in all the previous follow up surveys.

The number of missing values varied by variables. For the 2006 survey, the missing values were highest for food allergy (64.7%), pet allergy (64.4%), hay fever (62.8%), eczema (60.9%), paternal smoking during pregnancy (28.7%) and for computed variable CWB symptom triad (40.1%). The reason for increased missing responses for respiratory and allergic symptoms is not known, but could be due to the parents being reluctant to answer questions regarding respiratory health of their children. Weight and height could not be measured in some children due to school absenteeism. Missing values for asthma defined by the symptom triad could in part relate to missing data for one of the triad symptom categories. The nature of these symptoms might confuse some parents as many of the symptoms such as cough and wheezing that support the diagnosis of asthma in children in young children may not be specific for

asthma as reported by Bush (2007). The lowest number of missing values was recorded for child's sex (0%) and child age (8.7%). Conducting direct interviews of parents and visiting homes or based in school should reduce this inherent problem in self-completed questionnaires, which could be helpful in maximising the response rates.

5.4 Study characteristics

5.4.1 Demographic characteristics

All schools were predominantly located in lower socio economic areas with 91.7% of the children belonging to the lowest socio-economic class. School based surveys conducted in children from lower socio-economic areas allows inclusion of lower socio economic status as one of the confounding factors in analysis of health outcomes. The details of estimation of the Townsend Deprivation Index Scores which were calculated using the Manchester Information and Association Services Systems Software are summarised in the methodology section. The Townsend overall deprivation indices were chosen because their conceptual development is clearly defined and the indices have been demonstrated to be valid (Platt and Ashton, 1991).Despite their limitations they achieve a balance between ease of measurement and conceptual adequacy.

Since the previous analyses of this data by socio-economic status had been based on Townsend scores, the same method for assessment of socio-economic status had to be used in the present study as well. Lower socio-economic status could be associated with increased prevalence of maternal smoking during pregnancy. A longitudinal study on pregnancy from Mater Hospital, University of Queensland, Australia was carried out and data from 8556 pregnant women attending antenatal clinics during the period between 1981 and 1984 were analysed (Najman et al, 1998). This study examined the association between socio-economic status and pregnancy smoking habits and reported that women from lower socio-economic groups more commonly smoked before, during and after pregnancy. However, in this study, the additive effects of air pollution in a lower socio-economic setting have not been considered. It was also reported that young single mothers were more likely to be heavy smokers and more likely to be poor, although women who quit smoking during pregnancy had a more than 50 percent chance of relapsing within six months of the birth, regardless of income level (Najman et al, 1998). It is likely that pregnant women who smoke during pregnancy and who are from

lower socio-economic backgrounds will experience a disproportionate burden of tobacco related mortality and morbidity due to lack of access to smoking cessation programs and medical services, and due to lack of support from the medical and social services when seeking to quit smoking (American Legacy, 2010). Hiscock et al (2012) in their review on socio-economic status and smoking reported that smoking prevalence was higher among pregnant women from lower socio-economic groups, who were less likely to quit due to stronger addiction to tobacco and low motivation to quit. There is less evidence of interventions that work among low socio-economic groups.

5.4.2 Child characteristics

The age distribution of children was consistent with a primary school population with a slight female bias (49:51). 15.3 % of children were reported to have been born preterm which was higher than the prevalence reported in the previous 1998 survey in this school population (12.4%). The size of this difference is substantial ($p = 0.039$) and raises the question whether the 2006 value overestimates the true prevalence. The other possible explanations could be the influence from missing values, definitions used, and changes in survival rate in neonatal unit. Prematurity in this question is likely to indicate that the mother was informed by a doctor (or nurse) that their baby was preterm. It is not possible to validate this response. It does not possibly reflect the proportion of babies admitted to the Neonatal Intensive Care Unit here and probably reflects that the majority were only a few days preterm. The UK national estimate for preterm birth reported in 2007 was 6% (1 in 13 live births), (National UK Birth Statistics, 2007). In Liverpool, a study by Robinson et al (2011), which was part of a retrospective cohort study of 39,873 women in Liverpool, UK, from 2002–2008 reported that 2.6% (n=1036) of pregnancies resulted in preterm birth before 34 weeks at the Liverpool Women's Hospital.

There was a doubling of the low birth weight in 2006 compared to the 1998 survey suggesting some concordance in this reporting (11.4% versus 6.8%). Male babies were more likely to have been reported as being preterm than the female babies, whereas female babies were more likely to have low birthweight than the male babies for the 2006 survey. Although it is uncertain, whether these differences are true findings, it is unlikely birthweight reporting is inaccurate as most mothers try to recall this parameter with relative confidence and maternal birthweight recall has been reported to be of value in epidemiological studies in similar settings (Lule et al 2012). A further reason for these survey differences in birth outcomes could be

selection bias, with varied response rates of mothers from families with different socio-economic profiles.

5.4.3 Anthropometric characteristics

The anthropometric measurements were essential in order to assess the growth patterns of the primary school children in general and also to compare the growth patterns of these children in relation to the pregnancy smoking exposure and air pollution indicator categories and their combinations.The median weight of the children was 28.6 Kg (SD 8.93) which was higher when compared to the 1998 survey 28.4 Kg (SD 8.37). The minimum weight recorded was 15 kgs and maximum weight recorded was 73.20 kgs. 121 children had weight more than 30 kgs and 14 children had weight more than 50 kgs and the highest recorded weights were 64.20 and 73.20 kgs. The child for whom the recorded weight was 73.2 kgs (99.4th BMI centile) was a male child and had a birth weight of 3.64 kgs and height of 152cms. This child who was grossly obese represented the tip of the iceberg of the problem of overweight and obesity among these school children. The mean height of the children was 126.03 cms (SD 14.35) which was lower compared to that for the 1998 survey 127.43 cms (SD 11.66).

The minimum height recorded was 84 cms and maximum height was 191cms. 51 children had heights more than 145 cms and 4 children had height in the range 170 cms and 191 cms. Outliers for weight and height were noted and identified. The extreme values for weight and height recorded could explain the outliers noted in the graph. Increasing child weights and decreasing child heights in 2006, when compared to 1998, emphasises the need to look at the trends for child weight and height in future.

The mean 2006 BMI z- score was higher than the value for the 1998 survey (0.45 versus 0.34), which was higher than mean BMI z scores for Aberdeen primary school children (Crum et al, 2006). Since the BMI z scores were calculated using the Epi info Nutrition 6 software, this facilitated calculation of z scores appropriate for age, weight, height and sex of the child using the CDC guidelines which used 1.04 and 1.64 as cut offs for defining overweight and obesity respectively. Studies have shown that BMI z-scores calculated in this way and used for defining childhood obesity and overweight have been found to be predictive of co-morbidity associated with obesity (Rudolf et al, 2008). Increasing child weights and body mass indices

and decreasing child heights in 2006, when compared to 1998 suggests an increasing trend in childhood obesity and short stature from 1998 to 2006.

5.4.4 Respiratory characteristics

The prevalence of wheezing was 19.3% which was lower than the 1998 survey (29.8%) (Koshy et al, 2010).This may indicate that wheeze in these children is a symptom of asthma and not due to other causes, since there also was a decrease in prevalence of doctor diagnosed asthma in these school children during this same period. The prevalence of breathlessness in the past 12 months was 7.2 % in the survey, and the prevalence of excess cough was 18.7%. The excess cough prevalence was much higher than the value for the 1998 survey (5.1%) (Koshy et al, 2010). This difference may be due to maternal over-reporting reflecting the non-specific nature of this symptom, although why this should occur specifically for the 2006 sample is unclear, even though there have been progressive increase in viral infection induced upper respiratory infections among school children, which might explain the rise in non-specific cough among these children (Bailey & Chang, 2008). The possibility of selection bias also needs to be considered particularly as compliance with the 2006 survey was lower than that of previous surveys and the parents are more likely to answer positively, if cough is present. Asthma was reported as well controlled in 71.4% of the children.

Respiratory symptoms were an important cause of school absenteeism, 47.3% of children were reported as absent from school due to respiratory symptoms in the previous 12 months, and 9.7% had been admitted to hospital due to various respiratory symptoms. Even though respiratory infections are quite common among primary school children, the occurrence of these infections follow a seasonal trend as well which might explain the rise in peaks of respiratory infections during certain periods of the year. Overall 20.3% of the children were reported to experience allergy. Changes in housing in recent decades have led to an increase in the indoor levels of house dust mite allergen which may relate to allergy risk (Rodrigues et al, 2008). The lower prevalence of allergy parallels the decrease in doctor diagnosed asthma, supporting an allergic component as a related cause. The prevalence of maternal asthma was 13.2% and paternal asthma 9.5% in 2006. Maternal asthma prevalence was higher than paternal asthma, which is consistent with other published studies (CDC, 2001).

5.4.5 Parental smoking patterns

Parental smoking patterns for parents and household members of children from fifteen schools and pregnancy smoking data from Liverpool Women's Hospital have been studied previously (Kelly et al, 1995; Kelly et al, 1997; Delpisheh et al, 2006a, Delpisheh et al, 2006b; Delpisheh et al, 2007, Delpisheh et al, 2008; Rizwan et al, 2007, Koshy et al, 2010a, Koshy et al, 2010b; Koshy et al, 2011a, Koshy et al, 2011b;; Koshy et al, 2012a, Koshy et al, 2012b). The present study included parental smoking patterns of parents of children from 10 schools only, which was needed to look at the combined effects of air pollution and pregnancy smoking exposure on birth and child health outcomes. 25.4% of mothers smoked during pregnancy. The prevalence of pregnancy smoking was comparable to findings from other European countries, for example Portugal, in which it was reported that 29% of the mothers smoked during their pregnancy (Simoes et al, 1994). The prevalence of heavy maternal smoking during pregnancy was 6.8% (19/278). Prevalence of pregnancy smoking in Liverpool is one of the highest in the United Kingdom. Delpisheh et al (2006) reported reduction in smoking prevalence in pregnancy in adults during the decade of 1990's, when smoking cessation programs were initiated, but an increase in smoking prevalence occurred in adolescent pregnancies from 40.6% in 2000 to 46.3% in 2003.

The prevalence estimates of paternal smoking and household smoking at the time of pregnancy were 35.8% and 43.7% respectively, reflecting the high prevalence of smoking amongst household members in this study sample. The prevalence of paternal heavy smoking was 7.4% (20/269) suggesting heavy smoking was more prevalent on the paternal side when compared to the mothers who smoked heavily during pregnancy, which was quite similar to the paternal and maternal smoking prevalence patterns during pregnancy. The prevalence of smoking by grandfathers and grandmothers during the period of pregnancy was 31.1% and 40.6% respectively which was consistent with the high prevalence estimates for their adult children. These chronic family patterns of pregnancy smoking reflect a generalised cycle of smoking exposures.

5.4.6 Air pollution patterns

Four air pollutants were available and included in the present study- NOx emissions, PM_{10} emissions, NO_2 concentrations and PM_{10} concentrations. The median NOx emissions was

1.08 tonnes per annum and median PM_{10} emissions was 0.04 tonnes per annum. Higher median emissions for NOx and PM_{10} were reported in the L20 1 Bootle, where the air pollution used to be high due to heavy traffic congestion and delivery of goods (Sefton Air Quality Report, 2010) suggesting increased release of air pollutants into the air in this area from traffic. The median NO_2 and PM_{10} concentrations were 22.15 and 15.65 micrograms per m3 respectively indicating the concentration of these air pollutants present in the air, which were reported to be higher in the L20 8 and L20 9 postcode sector areas. Even though the emissions and concentrations of air pollution indicators gives us a clear picture of the air pollution in a specific area, the possible health effects from these air pollution indicators depends on the period of exposure, mode of travel, particles composition and the type of pollutant. Travelling in areas near to roads with traffic congestion may contribute to a substantial fraction of the total daily exposure, especially for those inside vehicles, if they are exposed to air pollutants released from exhaust emissions from neighbouring vehicles (Barett et al, 2008; Dons et al, 2012). Even though the level of concentrations of air pollutants in a specific area depends on the emissions, especially when released from traffic sources, it could vary during different periods depending on periods of heavy traffic and the level of exhaust fumes released which is further influenced by the wind direction and seasonal changes. A comparison of these emissions and concentrations between 2006 and 2008 were carried out to assess these trends. There was increase in the prevalence of NOx emissions from 68.6% in 2006 to 72.8% in 2008 and also for PM_{10} emissions from 21.3% in 2006 to 22.8% in 2008. This suggests an increase in the release of NOx emissions and PM_{10} emissions possibly from increase in the traffic releasing exhaust fumes in this area. This might also suggest an increasing trend in the air pollution prevalence in these areas, which needs to be assessed in future to confirm if the increasing trend is real or a transient rise. There was same increasing trend for high NOx-PM_{10} emissions as well, which increased from 20.8% in 2006 to 22.3% in 2008, emphasising the need for more measures to tackle the increasing air pollution problem in this area, after assessing the present situation and conducting future studies to assess the air pollution problem. The trend in NO_2 concentrations and PM_{10} concentrations could not be assessed as the concentrations data for 2008 was not available.

5.4.7 Combined air pollution and pregnancy smoking patterns

The prevalence of air pollutant categories combined with pregnancy smoking categories were also determined for the purpose of comparing the difference in occurrence of

birth and child health outcomes when assessed in relation to these categories. There was increased prevalence of high NOx emissions when combined separately with HSDP (61.6%) compared to the same categories when combined with PSDP (52.7%) and MSDP (41.9%) respectively. There was a similar decreasing trend, when high PM_{10} emissions was combined with HSDP, PSDP and MSDP respectively (18.6%, 15.0%, 10.8%). The combination categories for high NO_2 and high PM_{10} also showed a similar decreasing trend when combined with HSDP, PSDP and MSDP respectively (95.1%, 94.6%, 91.4% for NO_2 concentrations and 87.8%, 83.6%, 77.4% for PM_{10} concentrations). The higher prevalence noted for these air pollution indicators with HSDP, when compared to MSDP and PSDP suggests the role of other household members in the family and the influence of their smoking habits which seems to be more pronounced, when compared to MSDP and PSDP, even though mothers and fathers who smoke during pregnancy are also included in the household members who smoked during pregnancy. The least prevalence noted among mothers might be explained by the fact that pregnant women tend to stay and spend more time at home compared to the other two groups who are more exposed to air pollution. The prevalence of high NOx emissions combined with heavy maternal smoking was higher when compared to the prevalence of high NOx emissions with heavy paternal smoking. In contrast, the prevalence was higher for high PM_{10} emissions combined with heavy paternal smoking when compared to the combination with heavy maternal smoking. For the concentrations data, the prevalence of high NO_2 concentrations and high PM_{10} concentrations separately combined with heavy maternal smoking was higher when compared to the separate combination with heavy paternal smoking. This shows that the prevalence of different air pollution indicators combined with heavy maternal smoking were in general higher when compared to the combination with paternal smoking except for the combination with high PM_{10} emissions, which could not be explained. Similar trends were noted with high NO_2 concentrations and high PM_{10} concentrations which showed higher prevalence when combined with heavy maternal smoking compared to the combination with heavy paternal smoking, suggesting higher prevalence of air pollution indicators when combined with heavy maternal smoking prevalence, even though the sample size was limited for the combined air pollution and heavy smoking categories. Furthermore, air pollution can be compared to increased exposure to particulate matter at different levels, but the difference lies in the fact that air pollution is involuntary and affects wider population whereas smoking is voluntary and affects the smoking person, and the unborn baby especially in pregnancy and the others surrounding the smoking person. The patterns of air pollution and smoking and their

combined defects also depends on the level of air pollutants exposed to and the number of cigarettes smoked.

5.5 Air pollution exposure and pregnancy smoking exposure and birth and child health outcomes

5.5.1 Air pollution exposure and birth and child health outcomes

Air pollution exposure and birth and child health outcomes were assessed only in the 10 Liverpool schools as data on air pollution exposure was available only for these schools. There was significantly decreased risk of hay fever and croup associated with high NOx emissions, however it is difficult to explain the underlying mechanisms for decrease in these childhood conditions in relation to air pollution. High NO_2 concentrations were significantly associated with decreased risk of preterm birth, which was also observed with combined high NO_2 and PM_{10} concentrations, even though the decreased risk was more significant with the combined group, suggesting a possible additive effect on the preterm births from high PM_{10} concentrations. The significant association of preterm birth with low NO_2 concentration is unexpected and could be a chance finding. This is because if this association was real, it should have also shown an association with the birthweight. The preterm birth status was based on maternal recall and needs to be assessed, how this variable differs from the preterm status data collected from hospital records. It may also reflect the low denominator numbers in some of the comparator groups, but also need to consider the other risk factors for preterm birth before coming to a final conclusion.

Because its primary source is traffic emissions, NO_2 is considered to be a valid proxy for exposure to air pollution from traffic suggesting the possibility of higher traffic congestion and resulting air pollution in these areas. Comparisons between studies on air pollution caused by traffic sources are difficult because of variation in exposure measurement methods, as well as study differences leading to misclassification of exposure assessment, and birth outcomes.

High PM_{10} emissions were associated with increased symptom prevalence of ever wheeze. These findings are consistent with results from other epidemiological studies which have reported significant associations of adverse respiratory symptoms including wheeze in children with air pollution from particulate matter and road traffic (Janssen et al, 2003;

McConnell et al, 2006; Nicolai et al, 2003; Venn et al, 2005; Brunekreef et al, 2009; Venn et al, 2005). High PM_{10} concentrations were associated with increased childhood obesity, even though the association was not highly significant (p=0.047).

Although increased wheeze was the only respiratory symptom associated with increased PM_{10} emissions and combined high NOx and high PM_{10} emissions in the present study, there was no association between doctor diagnosed asthma and increased PM_{10} exposure. Exposure to traffic pollution with increased exacerbation of doctor diagnosed asthma has been reported in children (McConell, 2006). Molter et al (2015), who reported a meta-analysis of data collected, using validated parental questionnaires on asthma and current wheeze prevalence at 4-5 years and 8-10 years in relation to air pollution, showed there were no significant associations between childhood asthma prevalence among 8-10 years old children and exposure to high PM_{10} and NO_x emissions. In the present study, using the same ISAAC study questions on asthma and wheeze as in the Molter study the results showed no association between air pollution and doctor diagnosed asthma, but there was a positive association with wheeze in relation to high PM_{10} emissions. The Air Quality guidelines (2005) reported that even relatively low concentrations of air pollutants resulted in a range of adverse health effects and the adverse health outcomes were more prevalent in homes where biomass fuels and coal were used for cooking and heating. Significant effects have been seen between increased PM_{10} mass concentrations (PM with aerodynamic diameter < 10 μm) and increased visits for asthma and prescriptions for inhalers (Chimonas and Gessner, 2007).

There are several studies including a small number of meta-analyses and multi-national studies which have been published on childhood asthma and air pollution exposure (Pattenden et al, 2006; Anderson et al 2010; Anderson et al 2013; Nishimura et al 2013; Gasana et al, 2012). A borderline significant increase in asthma prevalence associated with NO_2 or NOx exposure has been reported in a meta-analysis of published studies reported by Gasana et al, (2012). A recent meta-analysis of multi-community studies showed no evidence of an association between community levels of NO_2 or PM_{10} concentrations and asthma prevalence among children (Anderson et al, 2013). There have been no evidence from remaining studies suggesting an association of asthma or wheeze among children exposed to air pollution (Anderson et al, 2010; Nishimura et al, 2013). Nevertheless, these studies did not examine exacerbations of asthma symptoms in children with pre-existing asthma. Anderson et al (2010) in their study also used parental questionnaires, which was similar across each cohort and

reported that there was a good agreement between maternal recall response with the response noted in medical records during early childhood, which also had been reported previously (D'Souza-Vazirani et al, 2005). Unfortunately, Anderson et al (2010) in their study did not adjust for epidemics, impact of vaccinations or frequency of infections, as these data was not available across all cohorts. There is growing evidence of association of indoor air pollution including second hand smoke and biomass use and outdoor air pollution as risk factors for respiratory infections in children (da Costa et al, 2004; Brauer et al, 2006; Leonardi et al, 2000). Particulate material in the atmosphere is a complex mixture of particles of different sizes and composition arising from a range of sources including emissions from man's activity, including fuel combustion, grit and dust from various industrial processes, wind lift from ground, emissions from motor vehicle exhausts and particles formed in the atmosphere by chemical reactions (Department of Health, 1992). There is evidence that particulate air pollution even at the relatively low concentration prevalent in United Kingdom cities may be responsible for adverse effects on health (Anderson et al 2004). The main associations are with respiratory and cardiac effects, especially in older people, and it seems likely that the effects of particles are to cause exacerbation of already existing disease in vulnerable people. Seaton et al (1994) have proposed that the pathogenicity of the particles is based not on the mass inhaled but rather on their numbers, and thus the surface area available to react with epithelial and inflammatory cells in the lung.

In the present study, in general, air pollution was associated with ever wheeze among children, especially with high PM_{10} emissions with combined high NOx. Interestingly, there was a decreased risk of croup with high NOx emissions and with combined high NOx and high PM_{10} emissions, which could not be explained. There was no association of air pollution with doctor diagnosed asthma in the present study. McCormack et al (2008) in their study, which investigated the in-home PM on asthma morbidity among a cohort of 150 children in the 2-6 years age group from Baltimore, Maryland, reported that for every 10-μg/m3 increase in PM measured indoors, there was a 7% increase in days of wheezing severe enough to limit speech and a 4% increase in days on which rescue medication was needed, even after adjustment for potential confounders such as other respiratory problems and lower socio-economic status. A study in urban and rural areas in Switzerland concluded that the incidence and duration of respiratory symptom episodes in children aged 0 to 5 years were associated with total suspended particles in air and that the duration of symptoms may be associated with nitrous and nitric oxide concentration (Braun Farhlander, 1992). It has been reported that

asymptomatic children living in highly polluted industrial areas have increased bronchial responsiveness compared to children from non-polluted areas (Wang et al, 1997; Molfino et al 1992). In contrast, Ware et al (1986) showed no association between bronchial responsiveness and air pollution. The different findings in the present study are consistent with several recent longitudinal studies in Europe and Southern California which reported associations between residential traffic and asthma incidence (Gauderman et al, 2005; McConnell et al, 2006). It is interesting to note that in the present study, NO_2, a secondary product of traffic emissions, had stronger correlations with wheeze suggesting a toxin or chemical related underlying mechanism affecting the airways.

Current evidence has not yet convincingly demonstrated that high indoor NO_2 concentrations contribute to the risk of developing asthma, because NO_2 concentrations seem to be similar in homes of children with and without asthma (Diette et al, 2007; Hoek et al, 1984; Institute of Medicine Committee on the Assessment of Asthma and Indoor Air, 2000). Studies done in subjects with asthma have suggested that higher indoor NO_2 concentrations lead to increased asthma symptoms; however, results have not been consistent across subpopulations (Belanger et al, 2006; Garrett et al, 1998; Hasselblad et al, 1992; Kattan et al, 2007; Nitschke et al, 2006; Shima and Adachi, 2000; Smith et al, 2000). Increased symptoms and bronchodilator usage have been associated with increased levels of PM_{10} pollution (Roemer et al, 1993). Buchdahl et al (1996) reported that acute wheezing episodes in children were associated with increased levels of summer haze (with high SO_2 and O_3 content). Conversely Hoek and Brunekreef (1995) in a study from Netherlands did not show a relationship between reporting of symptoms and wide fluctuations in PM_{10} concentrations (11-135 microgram/m_3 or O_3 (14-114ppb) levels. In general, consistent association between respiratory symptoms and PM_{10} concentrations have been shown which suggests that there could be a possible association between the particle concentration and the chemical composition (Osunsanya et al, 2001).

Studies have showed that a small fraction of distally deposited particles from air pollution migrate through alveolar tissue directly into the lymphatic circulation (Dockery and Pope, 1994). Damage to the respiratory tract may occur if the rate of deposition exceeds the rate of removal, or if particles are chemically reactive. Large particles may cause bronchitis, whereas small particles might lead to alveolitis and small airways disease. The clearance mechanism may be altered by exposure to pollutants, high concentrations of SO2, and

decreasing action of cilia and during such exposures particulate matter may become fixed more easily in the airway (Salathe et al, 1997). Studying the effects of air pollution on health is complicated as the actions and effects of individual pollutants may differ from the effects of a combination of pollutants. Then pollutants may act in combination in three ways, additive, antagonistically or synergistically (Department of Health, 1992; Salathe et al, 1997). Also there may be considerable variation in responses noticed within and between individuals. The mechanisms underlying the effects of particles on human health are not fully understood. Non-specific irritation of the airway may cause inflammation which could result in increased bronchial activity (Landau, 1995). Interaction between pollutants and aeroallergens may be synergistic, such that when particles deposit on airway epithelium the passage of antigen across the epithelium may be enhanced. Genetic studies examining pathways likely to mediate effects of air pollution also strengthen the causal inference that traffic-related pollutants may cause asthma. It has been shown that the risk of asthma associated with traffic exposure and with traffic-related ambient PM was modifiable in a predictable way by functional gene variants in pathways associated with asthma, including inflammation and airway remodelling (Salam et al, 2007a) and oxidative stress (Islam et al, 2008; Salam et al, 2007b). Atmospheric emission may affect the incidence of respiratory symptoms by altering respiratory immune mechanisms and increasing mucus secretions predisposing to virus or bacterial infections.

5.5.2 Comparing means of air pollutants and assessment of growth parameters in relation to air pollution and smoking

There was increased mean NOx emissions and PM_{10} emissions in areas with increased bronchitis and ever wheeze by comparing means method and decreased mean NOx emissions in areas with increased croup. This shows that the increase in mean NOx and PM_{10} emissions were associated with increased respiratory problems in this area in general, even though there was no significant association noted with doctor diagnosed asthma. There was increased average NO_2 and PM_{10} concentrations in areas with increased prevalence of childhood obesity. This could indicate causality due to airborne toxins acting directly or indirectly producing pregnancy metabolic changes and intrauterine effects predisposing to later childhood obesity. It is possible that difference in the chemical composition of particulate matter in different areas might contribute to the different effects in different areas of the same region. The mean concentrations of NO_2 were also higher in areas with higher prevalence of maternal smoking during pregnancy, higher prevalence of household smoking during pregnancy, lower socio-

economic status and decreased prevalence of breast feeding. Mean concentrations of PM_{10} were also higher in lower socio-economic areas and in areas with more female children. The fact that they were lower in areas with a higher prevalence of breast feeding could be related to selection bias.

Comparison of growth parameters including child weight, child height, birthweight, body mass index, weight for age z-score and height for age z scores in school children in relation to different combinations of air pollution and pregnancy smoking indicators were carried out in individual and combined group categories. Eventhough, there were variations in different growth parameters in relation to different individual and combined air pollution and pregnancy smoking group categories there were no significant associations identified except for mean birthweight which was lower with high NO_2-PM_{10} concentrations when combined with maternal smoking during pregnancy.

Studies have reported growth changes in children in relation to air pollution and smoking (Jerett et al, 2014, Koshy et al, 2012). Jerett et al (2014) conducted a prospective cohort study of 4550 children across 13 communities in Southern California and showed that traffic pollution was positively associated with increase in the BMI in 5-11 year old children even after adjusting for confounding factors and that there was a positive association with traffic density as well. The confounding factors included asthma status of the child, the language used to complete the questionnaire (Spanish or English), child exposure to second-hand smoke in the home, parental education, the number of fast food outlets within 500 m of the child's home, greenness around the home as measured by the normalized difference vegetation index, and the number of active recreational programs for children offered within 5 km of the home, and traffic density at 150 m. They did not identify the responsible components of the traffic mixture, and there was lack of information on dietary habits of the children. Koshy et al (2010) reported that children whose mothers had only passive cigarette smoke exposure during their pregnancy also had lower mean height for age z-scores compared to those of parents who were non-smokers ($P < 0.01$), which was also noted among children born to mothers who smoked during pregnancy. Mean BMI z-score was also higher in maternal smoker categories compared to parental non-smokers, but this difference was significant only for children of mothers who alone were household smokers.

5.5.3 Interaction analysis

Interaction analysis was carried out in order to show the significant interactions between the exposure factors which is essential in order to completely understand the association between the different exposure and outcomes, since many of the exposure factors could have confounding effects, when looking at their effects on birth and child health outcomes. High NOx emissions were significantly higher in lower socio-economic areas, which might be explained by the close proximity to traffic and industrial sources and there was decreased prevalence of breast feeding. It was also associated with increased maternal smoking during pregnancy, high NO_2 concentrations, and high PM_{10} concentrations. There was increased mean NOx and PM_{10} emissions noted in relation to increased prevalence of maternal smoking during pregnancy, paternal smoking during pregnancy and household smoking during pregnancy using the comparing means method which might be related to the smoking habits of the population in the same area correlating with the amount of toxic particles present in air and their differing composition from traffic and/or smoking exposures. High NO_2 concentrations were significantly associated with lower socio-economic status, high PM_{10} concentrations, high NOx emissions and heavy paternal smoking. High PM_{10} concentrations were significantly associated with increased lower socio-economic status, household smoking during pregnancy, and maternal smoking during pregnancy, high NOx emissions, high NO_2 concentrations, heavy paternal smoking and decreased prevalence of breast feeding in the present study. In general, there was significantly increased lower socio economic status associated with maternal smoking during pregnancy, household member during pregnancy, high NOx and high PM_{10} emissions and high PM_{10} concentrations and decreased lower socioeconomic status with increased breast feeding and increased heavy paternal smoking.

The present study showed air pollution exposure occurred more frequently among children from lower socio-economic areas. The close proximity of households living near to the polluted areas and high dense traffic areas might contribute to higher air pollution in these areas. The adverse effect of air pollution on child health outcomes may be influenced by disadvantaged living conditions. This may contribute to a modification effect due to less access to nutritious food, fresh leafy vegetables, fruits and fish, and reduced intake of essential antioxidants, polyunsaturated fatty acids and vitamins which may offer protection against the adverse consequences of particle exposure (Romieu et al, 2005). Children from disadvantaged groups have been found to be more highly exposed to some air pollutants (Sexton et al, 1993).

Many inner-city households use gas stoves, an important source of indoor NO_2 concentrations, and many of these stoves are unvented (Breysse et al, 2005; Diette et al, 2007), but the information on the presence of gas stoves in household was not available in the present study.The use of household gas stoves have reported to increase the indoor NO_2 levels more than the outdoor NO_2 irrespective of the season as reported by Dėdelė A & Miškinytė A (2016) in their study, which looked into the seasonal variation of indoor and outdoor air quality of nitrogen dioxide in homes with gas and electric stoves. Increased outdoor NO2 levels could be more related to the degree of urbanisation and traffic in the location of residence as reported by Esplugues A et al (2010).

Epidemiologic studies have linked proximity to busy roads, where the air pollution is higher, with adverse health outcomes, including respiratory symptoms, asthma, adverse birth outcomes, and cardiopulmonary mortality (Brunekreef et al, 1997; Hoek et al, 2002; Wilhelm and Ritz 2003). Methods for estimating exposures to traffic pollutants have included neighbourhood- or school-based estimates of traffic (Brunekreef et al, 1997; Kim et al, 2004), distance to freeways or busy roads (Gauderman et al, 2005), presence of a busy road within a given buffer (area of land designated for environmental protection) (Venn et al, 2001), and traffic density within a given radius (English et al, 1999; Wilhelm and Ritz, 2003).

Recently, studies have started to examine the role of SES in the vulnerability of sub-populations to outdoor air pollution especially to particulate matter, although the results remain inconsistent with varied associations (O'Neill et al, 2003). Studies reported from the Scandinavian region have shown variations in personal exposure to particles and other pollutants by education and occupation (Rotko et al, 2000, 2001). Similarly, another study from United States indicated differences in exposure to air pollutants especially with gaseous pollutants when stratified by education, income status, minority and education, and it was also related to the age of the home and exposure to indoor dust containing chemicals like lead from the painted surfaces (Pellizzari et al, 1999). Zeka et al (2006) reported that individual-level education was inversely related to the risk of mortality when associated with PM_{10}. In their study, which was carried out as a case-crossover study of 20 US cities, it was reported that PM_{10} related, out-hospital deaths were more common than in-hospital deaths and suggesting the need for emphasis on population characteristics to identify the greater chances of exposure and susceptibility. Modification of air particle effects by socio-economic status was reported by another cohort study from Hamilton, Ontario, (Finkelstein et al, 2003; Jerrett et al, 2004).

In contrast, Gouveia and Fletcher (2000) observed a larger effect of air pollution in areas of higher SES level, whereas Bateson and Schwartz (2004) found no indication whether susceptibility to air pollution was modified by group-level SES measures.

SES factors such as educational attainment may modify the health effects of outdoor air pollution in several pathways. Children coming from households with lower socio-economic status could be more prone to air pollution–related health hazards since they are more likely to have higher prevalence of pre-existing diseases. This in turn could confer a greater risk of dying when associated with air pollution exposure, and they are more likely to receive inferior medical treatment for pre-existing diseases (Chaix et al, 2006). In the Public Health and Air Pollution in Asia (PAPA) study, Kan et al (2008) reported stronger associations between pollutants [PM_{10} (PM with aerodynamic diameter < 10 μm) SO2, NO_2, ozone (O3)] and daily respiratory mortality among women, elderly, and in children from lower socio-economic areas. Air pollution in huge concentrations such as Sao Paulo, Brazil has been associated with respiratory ill health in 12 to 13 year old children with evidence that socio-economic conditions aggravated the problems (Sobral et al, 1989).

Young children and those with lower SES may be at particular risk. For example, Smith et al (2000) identified an association between NO_2 concentrations and increased risk of asthma symptoms in individuals < 14 years of age, but not in older individuals, except for a marginal increased risk of cough in subjects 35–49 years of age. In a cross-sectional analysis, Belanger et al, (2006) found that indoor NO_2 exposure was associated with increased incidence and frequency of chest tightness and wheezing, but only in individuals living in multifamily housing units, which was an indicator of lower SES.

Although various hypotheses propose air pollution exposure effects on health are greater in people with lower socio-economic status (SES) (O'Neill et al. 2003), findings remain inconsistent: some studies found evidence of effect modification (Finkelstein et al, 2003; Jerrett et al, 2004; Krewski et al, 2005; Zeka et al, 2006), whereas others did not (Bateson and Schwartz 2004; Cakmak et al, 2006; Samet et al, 2000; Zanobetti and Schwartz 2000). Moreover, most of these studies were conducted in developed countries, with a small number conducted in Asia (Health Effects Institute 2004). There is need for conduct of similar studies in cities in developing countries, where characteristics of outdoor air pollution (for example-

air pollution level, mixture, and transport of pollutants), meteorological conditions, and socio-demographic patterns may differ from those in North America and Europe.

5.5.4 Combinations of smoking and airborne pollution exposures and birth and child health outcomes:

Different combinations of individual and combined air pollutant categories were carried out with different categories of pregnancy smoking exposure to assess the birth and child health outcomes in relation to these categories. The air pollution categories included NOx emissions, NO_2 concentrations, PM_{10} emissions, PM_{10} concentrations, combined NO_X-PM_{10} emissions which were combined with pregnancy smoking categories (MSDP, PSDP and HSDP) separately. The reason for analysis of individual and combined categories of air pollution with pregnancy smoking categories was to facilitate the comparison of birth and child health outcomes occurrence between individual and combined air pollution-pregnancy smoking groups. Univariate analysis showed different associations of birth and child health outcomes with combined individual air pollution and pregnancy smoking categories: For high NOx emissions, there was significantly increased ever wheeze, childhood obesity and female birth, when combined with MSDP; decreased croup and eczema and increased ever wheeze and obesity when combined with PSDP and increased female birth and childhood obesity and decreased croup, when combined with HSDP respectively. Even though, the increased childhood obesity with high NOx emissions categories combined with three categories of pregnancy smoking suggests probably a common toxin mediated mechanism underlying the association of obesity with these categories, it's difficult to determine the exact mechanism due to interactions between the toxins in smoke and varied composition of chemical toxins in air pollutants. For high PM_{10} emissions, there was significantly increased ever wheeze, childhood obesity and female birth, when combined with MSDP; increased ever wheeze and bronchitis, when combined with PSDP and increased ever wheeze and childhood obesity and decreased croup when combined with HSDP. There was increased ever wheeze with high PM_{10} emissions combined with three categories of pregnancy smoking suggesting a possible allergic mediated and inflammatory response to particles present in smoke and air pollutants, with possible contribution from toxins present in cigarette smoke and air pollutants as well. There was significantly decreased preterm births in relation to combined high NO_2 concentrations and MSDP, whereas there was no significant association of any of the birth or child health

outcomes in relation to combined NO_2 concentration with PSDP or HSDP. For high PM_{10} concentrations, there was significantly increased childhood obesity and female births, when combined with MSDP; decreased low birthweight, when combined with PSDP and increased female births when combined with HSDP. The association of increased female births with combined high PM_{10} concentrations with MSDP or HSDP suggests a possible toxin related particles present in tobacco smoke and air pollutant particles, even though the hormonal changes resulting from exposure to toxic particles might be one of the mechanisms.

For combined high NOx and high PM_{10} emissions, there was significantly increased ever wheeze, obesity and female births, when combined with MSDP, whereas for combined high NO_2 concentrations and high PM_{10} concentrations, there was significantly decreased preterm birth when combined with MSDP. The difference in significant increase or decrease in birth and child health outcomes with different emissions indicators with MSDP could suggest possibly different mechanisms underlying the associations. However, for combined high NOx and high PM_{10} emissions, there was significantly increased ever wheeze, bronchitis and decreased croup and eczema, when combined with PSDP, whereas for combined high NO_2 concentrations and high PM_{10} concentrations, with PSDP did not show any significant association. The change in outcomes with PSDP could suggest the variation in the level of exposure to different air pollutants for fathers when compared to mothers. For combined high NOx and high PM_{10} emissions, there was significantly increased ever wheeze and obesity and decreased croup and hay fever when combined with HSDP, whereas for combined high NO_2 concentrations and high PM_{10} concentrations, there was significantly decreased preterm birth when combined with HSDP.

The univariate analysis of birth and child health outcomes in relation to individual (NOx emissions, PM_{10} emissions, NO_2 concentrations and PM_{10} concentrations) and combined air pollution indicators (combined NOx-PM_{10} emissions and combined NO_2-PM_{10} concentrations) combined with dose related pregnancy smoking categories (heavy maternal smokers and heavy paternal smokers) were also carried out, even though the numbers available for the analysis was quite smaller due to subcategories. This analysis was needed to compare the birth and child health outcomes in relation to different combinations of air pollution and pregnancy smoking dose based categories with that of birth and child health outcomes in relation to individual and combined air pollution indicators combined with pregnancy smoking categories without dose response. Significant associations were only observed with combined high NOx emissions +

heavy maternal smoking during pregnancy (increased ever wheeze and doctor diagnosed asthma); combined high PM_{10} emissions + heavy paternal smoking during pregnancy (increased breathlessness); combined high PM_{10} + heavy paternal smoking during pregnancy (increased overweight); combined high NOx-PM_{10} emissions + heavy paternal smoking during pregnancy (increased breathlessness) and combined high NO_2-PM_{10} concentrations + heavy paternal smoking during pregnancy (increased overweight).

Logistic regression analysis by backward stepwise regression method demonstrated that there was significant independent association of childhood obesity with high NOx-PM_{10} emissions, high NOx emissions + MSDP, high NOx emissions + PSDP, high NOx emissions + HSDP and high PM_{10} + MSDP, whereas there was increased significant independent association of childhood ever wheeze with DDA, high NOx emissions + PSDP and high PM_{10} emissions + PSDP after adjusting for relevant confounding factors. However, a complete logistic regression analysis needs to consider other important factors like nutritional factors including dietary habits, genetic factors and hormonal factors for analysis related to obesity and other factors related to wheezing including viral infections induced wheeze and other common causes of wheeze. The regression analysis also showed significant independent association of decreased croup with high NOx emissions, high NOx emissions + PSDP, high NOx emissions +HSDP, high NOx –PM_{10} emissions + HSDP and high NOx–PM_{10} emissions + PSDP after adjusting for relevant confounding factors. Obviously there was decreased croup in relation to different combination of air pollution and pregnancy smoking categories, however the confounding factors for this analysis was quite limited. Logistic regression analysis for female births demonstrated that there was significant independent associations of increased female births with high PM_{10} concentrations + MSDP, high PM_{10} concentrations + HSDP, high NOx emissions, + MSDP, after adjusting for confounding factors. Many other environmental factors including hormonal change inducing agents like pesticides and other environmental toxins, could also have influence on the gender of the baby, when the mother is exposed to these factors during pregnancy through food chain or through different cosmetic products.

Here in this section below, two main outcomes-childhood obesity and croup which had significant associations with combined pregnancy smoking and air pollution exposure after adjusting for confounding factors have been discussed in detail with more emphasis on childhood obesity along with possible underlying mechanisms because of its public health importance.

5.5.4.1 Combined air pollution–pregnancy smoking and childhood obesity

The present study contributes evidence that on combined exposures to air pollution and pregnancy smoking exposure there is increased development of childhood obesity suggesting possible synergistic effects. Increased risk of adverse birth and child health outcomes in relation to combined high NOx and PM_{10} emissions with maternal smoking during pregnancy may be explained by the additive effects of both sources of air pollution acting directly through toxin induced mechanisms and through potential in utero effects of these toxins. There was significant independent association of childhood obesity with combined high NO_X-PM_{10} emissions and maternal smoking during pregnancy after adjusting for confounding factors in the present study. The exact mechanism underlying this association is not known. Many of the toxins present in tobacco smoke are similar to the toxins present in polluted air which are released from different sources including industries and traffic. Similar results on the development of childhood obesity in relation to air pollution and smoking exposure has been reported by McConell et al (2015) as part of the southern California children's health study, whereas some other studies reported increase in childhood obesity correlated with increase in air pollution alone (Jerret et al 2014; Li et al 2015). Air pollution resulting from toxic mass of pollutants released from cigarette smoking by household members results in non-smokers including pregnant women and children being exposed to pollutant concentrations that are many times higher than the levels normally permitted outdoors based on the existing environmental guidelines and standards of air quality (Nelson et al, 1998).

1 Possible mechanisms inducing childhood obesity associated with **air pollution exposure and pregnancy smoking exposure**

1a Intra-uterine effects of toxins from smoking and air pollution and childhood obesity

The amount of toxins from smoking as well as air pollution could have multiplicate adverse fetal effects leading to growth restriction. This effect is likely to be influenced by the number of cigarettes smoked during pregnancy or the amount of air pollutants, especially oxides of nitrogen and PM_{10} present (Levin & Dunn-Meynell, 1997). The role of natural barriers including the gut, nasal and lung epithelium and the blood brain barrier and their break down could facilitate the passage of air pollutants including toxins from smoke into the blood

stream and brain resulting in neuro inflammation (Brockmeyer et al, 2016). Even though the toxins released from air pollution and smoking exposure could enter the body through these barrier breakdown mechanisms, there is difficulty in measuring the amount of toxins absorbed and its composition due to the varied composition of the source of air pollution and toxins released from different types of smoking sources, which could play a role in determining the detrimental effects on children's health. Metabolic and neuroendocrine dysfunction may result from exposure to endocrine disrupting chemicals released from tobacco smoke and air pollution exposure such as persistent organic pollutants, phthalates, and bisphenol (Grun and Blumberg, 2009 and Thayer et al, 2012). Exposure to these chemicals also known as 'obesogens' during the developmental periods of life potentially plays a major role in endocrine dysfunction leading to later child obesity (Diamanti-Kandarakis et al., 2009).

There is evidence for a direct relationship between birthweight and subsequent attained BMI later in childhood with mechanisms related to chronic changes in the proportion of fat and lean body mass, central nervous system appetite control, as well as pancreatic structure and function (Okens & Gillman, 2003). Barker (1990) observed that there was a relationship between birthweight, as well as weight at one year of age, with adult morbidity and mortality due to coronary artery disease, cardiovascular disease, hypertension, and non-insulin dependent disease and renal disease. These adverse effects were greatest among babies with growth restriction and were least among premature babies (Leon et al, 2000). Growth restriction followed by postnatal over-feeding and a sudden upward shift in the growth curve to higher centiles is generally considered as an important risk factor for early onset adult diseases including cardiovascular diseases, type 2 diabetes, dyslipidaemic disorders, arterial hypertension and obesity (Simeoni & Zetterstrom, 2007). Catch-up growth may occur at any growth stage, but it is more often seen following severe growth restriction (Karlberg et al 1995). Mechanisms which regulate and signal early catch-up growth in the postnatal period may influence the extent of catch-up growth (Ong et al, 2000). This could be related to the mechanism of differentially methylated DNA in cord blood resulting from an intra-uterine mechanism (Sharp et al, 2015). Exposure to toxins from air pollution and smoking could result in genetic changes followed by postnatal events (Tang et al, 2006). The development of respiratory diseases in children from exposure to air pollution and pregnancy smoking exposure could also be influenced by the exposure during development of lungs especially in younger children (Duijts et al, 2012). There is a need for replication of this work using larger studies, and assessment of causality with methods such as two-step Mendelian randomization, which

is a strategy used for establishing the causal role of epigenetic processes in pathways to disease. However, all children living near to air pollution areas or living in homes with increased smoking exposure may not develop health problems, whereas some children are more susceptible to the untoward effects of air pollution and pregnancy smoking exposure. This could suggest that there could be many unknown underlying mechanisms operating, when toxins from air pollution and from pregnancy smoking exposure gets mixed with each other resulting in difference in the birth and child health outcomes, which varies with different levels of exposure and the composition of the toxins released and exposed to, in different environmental conditions, which further depends on the changes in climate including indoor temperature and humidity. Even though measurement of air pollution and pregnancy smoking exposure indicators in epidemiological studies depends on source of the air pollution and the health status of the exposed children, it is possible to use some methods like direct measurement of air in residences (Nieuwenhuijsen et al, 2008), which seems to be more reliable and by real time measurement of air pollutants or biomarkers using samplers like breath analysers.

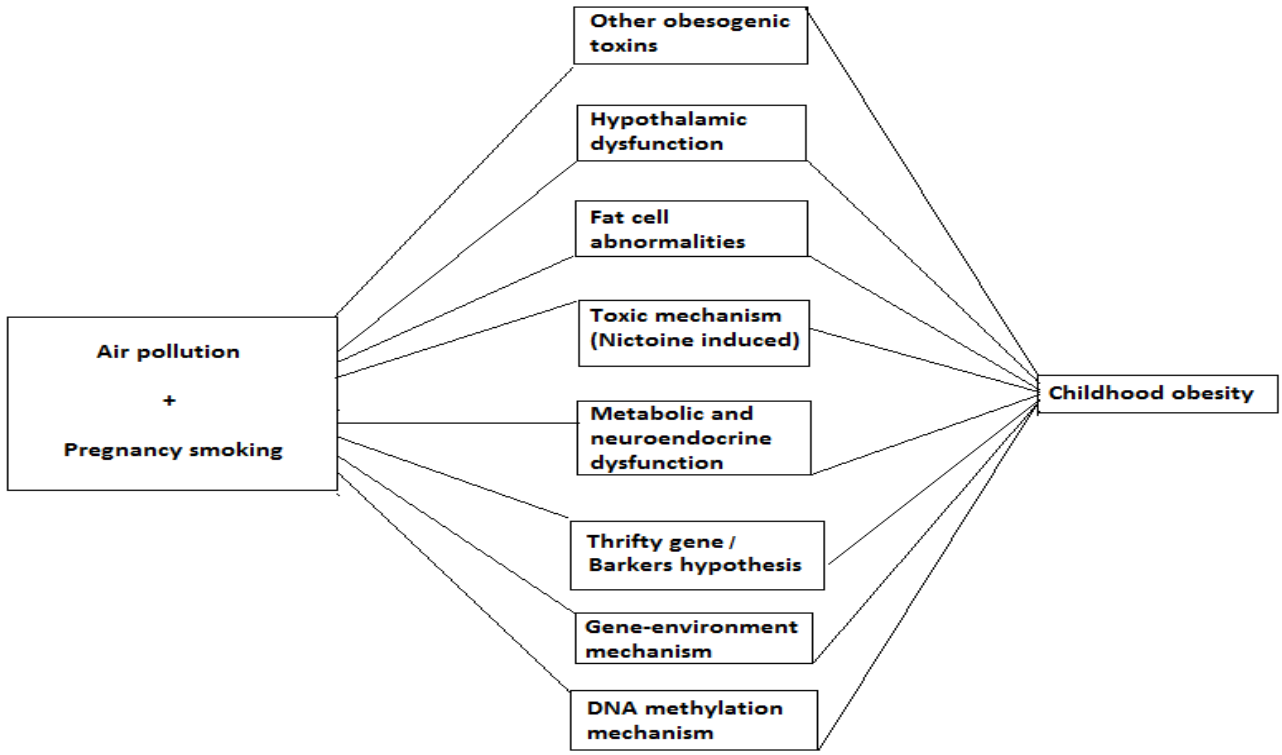
1b Other mechanisms

Even though there has been research on biomarkers for child health outcomes in relation to pregnancy smoking exposure, there is need for further research into the specific biomarkers of obesogenic exposures including polycyclic aromatic hydrocarbons (PAHs) and phthalates and their epigenetic effects (Liu et al 2008; Grun and Blumberg, 2009; Sharp et al 2015). Air pollutants and cigarette smoke contains chemical and toxins with endocrine-disrupting properties (Weitzman et al, 2005; Xie et al, 2010). Environmental tobacco smoke plays an important role in the relationship between PAHs and childhood obesity, because cigarette smoke itself is one of main sources of exposure to PAHs (Ding et al, 2005). Exposure to toxins with endocrine disrupting properties released from air pollution and pregnancy smoking, earlier in life could bring about changes resulting in diabetic pathophysiology among overweight and obese children. Toledo et al (2016) in their cross sectional study, which examined the relationship between air pollution and metabolic outcomes by performing the metabolic profiling among 429 overweight and obese African-American children living in urban Los Angeles, California reported that there was elevated air pollution exposure which in turn was associated with a metabolic profile corresponding to increased risk for type 2 diabetes. However, in this study even though the study showed that Ambient Air Pollution (AAP) (NO_2,

$PM_{2.5}$, and O_3) and traffic related air pollution (TRAP) exposure were associated with adverse effects on the glucose metabolism independent of the body fat, emphasis was given to prior year exposure to air pollution, when examining the effect of these air pollutants on diabetes related pathophysiology in overweight and obese minority children. There was 25.0% higher fasting insulin ($p<0.001$), 8.3% lower insulin sensitivity ($p < 0.001$), 14.7% higher acute insulin response to glucose ($p = 0.001$) with $PM_{2.5}$ and interestingly, similar effects were observed for NO_2 concentration as well. There could also be cardiovascular consequences among obese children which might result from systemic low grade inflammation as shown by C- reactive protein measurements (Tzoulaki et al, 2008).

Fetal malnutrition resulting from maternal smoking may also influence expression or function of insulin receptors, increasing the risk of subsequent insulin resistant type 2 diabetes and obesity (Lindahl et al, 2002). This is plausible because low birthweight, which has long been linked with maternal smoking, increases risk of insulin resistance and type 2 diabetes. Dose-response associations of pregnancy smoking with birth and childhood health outcomes have been shown by others. Von kries (2002) and Toschke et al (2003) observed a dose-dependent association between childhood overweight or obesity and maternal smoking during pregnancy which could not be explained by a wide range of confounders suggesting that intrauterine exposure to inhaled smoke products were responsible, rather than lifestyle factors associated with maternal smoking. The interaction between Poly Aromatic Hydrocarbons (PAHs) and Environmental Tobacco Smoke (ETS) is a biologically interesting phenomenon that reflects the synergistic interactions between chemicals in cigarette smoke and PAHs. However, almost all previous experimental and human studies on possible obesogens have focused on individual chemicals. The present analysis, to the investigators knowledge, is the first human study showing a strong synergistic interaction between 2 suspected obesogens. RCP and RCPCH Joint Air Pollution Report (2016) mentions about the role of chemicals from air pollution and their implications in the development of obesity, which could act as a vicious circle, as obese people are more sensitive to air pollution and more vulnerable among those exposed and living in most deprived communities due to poor housing conditions and indoor air quality, the stress of living on a low income, and limited access to healthy food and/or green spaces. Underlying mechanisms potentially involved with childhood obesity resulting from interactions between different toxins released from exposure to air pollution and pregnancy smoking are emphasised (Figure 5.1).

Figure 5.1 Possible mechanisms for childhood obesity associated with **pregnancy smoking and air pollution exposure**



5.5.4.2 Combined air pollution-pregnancy smoking and childhood croup

There was decreased croup prevalence associated with high NOx-PM_{10} emissions + PSDP, and high NOx-PM_{10} emissions + HSDP. Mechanisms to explain this association are unknown. Macintyre et al (2014) as part of the European Study of Cohorts for Air Pollution Effects (ESCAPE) project, investigated traffic-related air pollution as a risk factor for respiratory infections including croup during early childhood. The association between air pollution and croup was examined and a meta-analysis was conducted of the combined estimated effects in 10 European birth cohorts from Sweden (1), Italy (1), Germany (2), UK (1), Netherlands (1) and Spain (4). Parent reporting croup during early childhood (n=9101) was assessed in relation to annual average pollutant levels [nitrogen dioxide (NO_2), nitrogen oxide (NOx), particulate matter ≤ 2.5 μm (PM2.5), PM_{10}, PM2.5–10 (coarse PM)]. The results, even though not significant showed that the combined adjusted odds ratio was decreased for croup during the second year of life in relation to NO_2 (OR = 0.92; 95% CI: 0.78, 1.09), and NOx (OR = 0.92; 95% CI: 0.78, 1.08), as observed in the present study, which showed a significant

decrease in adjusted odds ratio for croup in relation to high NOx emissions (OR = 0.43; 95% CI: 0.24, 0.79) after adjusting for household member smoking during pregnancy and NOx and PM_{10} emissions. But Macintyre et al (2014), in their study used models which were adjusted for municipality, sex, older siblings, breastfeeding at 6 months, atopy of either parent, any child-care reported during follow-up, maternal smoking during pregnancy, any environmental tobacco smoke in the child's home reported during follow-up, visible mould or dampness in the home, use of gas stove, birth season, lower parental socio-economic status. The main strength of that study was its harmonised approach to study design, health assessment, exposure assessment and statistical analysis of child health outcomes in relation to air pollution exposure. One of the limitations was that it only analysed the outdoor air pollution exposure, excluding indoor air pollution as it was not feasible enough to reliably estimate indoor exposure individually for each of more than 10,000 children included in this study.

5.6 Disease indicators

5.6.1 Prevalence surveillance and spatial mapping

Prevalence surveillance using spatial mapping of birth and child health outcomes in relation to pregnancy smoking and air pollution (based on results from logistic regression) was conducted. For spatial mapping childhood obesity and croup were selected as the main outcomes as only these two variables showed clear significant association with combined smoking and air pollution categories in the logistic regression analysis. The availability of geographic information systems (GIS) facilitated creation of spatial maps for health data and has greatly expanded different techniques of presenting data from various health projects. The purpose of spatial mapping was to explore the significant child health outcomes associations in relation to pregnancy smoking exposure and air pollution in different socio-economic areas.

Spatial mapping showed that obesity associated with combined maternal smoking during pregnancy with high NOx-PM_{10} emissions was higher among school children who resided in postcode sectors of south and south east of Liverpool corresponding to a predominantly lower socio-economic area. Increased prevalence of maternal smoking during pregnancy in lower socio-economic areas has been previously shown by cross sectional surveys conducted in Merseyside schools based on parental questionnaires (Koshy et al, 2011; Koshy et al, 2012).

Spatial mapping of childhood croup categories in relation to combined paternal smoking during pregnancy – NOx-PM_{10} emission categories showed that there was low prevalence of croup associated with combined paternal smoking during pregnancy and high NOx-PM_{10} emissions among school children who resided in postcode sectors of south west areas of Liverpool which corresponded to predominantly lower socio-economic areas.

Spatial maps on childhood obesity and croup in relation to pregnancy smoking and air pollution are useful as maps for indicating the possible areas of increased or decreased prevalence of these outcomes in relation to these exposures in different areas of the study location and indicating the prevalence by colour coding. This is potentially relevant for preventive measures by targeting specific areas and improving localised health promotion efforts in areas with higher prevalence of these outcomes. It also identifies target areas for conducting studies into related risk factors and mechanisms.

5.6.2 Venn diagrams for profiling

Proportional Venn diagrams have been used in visual display of the interactions between pregnancy smoking, air pollution and birth and child health. The Venn diagram for childhood obesity in relation to maternal smoking during pregnancy and high NOx-PM_{10} emissions showed that there were nine children who had childhood obesity illustrated by the overlapping area between the three circles. Venn diagrams analysis of data also showed that there was one child with croup associated with high NOx-PM_{10} emissions + PSDP, compared to two children with croup associated with high NOx-PM_{10} emissions + HSDP.

Venn diagrams created based on interaction of childhood obesity with different categories of air pollution and pregnancy smoking showed that childhood obesity was more if individual air pollution categories were combined with household smoking during pregnancy and maternal smoking pregnancy, when compared to paternal smoking during pregnancy suggesting a possible association with household members who were frequently present at home or direct effect of maternal smoking exposure during pregnancy period.

A clear cut distinct overlap area for obesity in relation to different combinations of single and combined air pollutant categories was noted with high NOx emissions and MSDP, high PM10 emissions and MSDP, high NOx emissions and PSDP, high PM10 emissions and

PSDP, high NOx emissions and HSDP, high PM10 emissions and HSDP, high NOx-PM10 emissions and MSDP, high NOx-PM10 emissions with PSDP, and with high NOx-PM10 emissions with HSDP. Maximum overlap for obesity was noted with high NOx emissions and MSDP and with high NOx emissions and HSDP. The areas of overlap for childhood obesity helps in identifying and differentiating obesity as a public health problem in selected post code sector areas in relation to air pollution and pregnancy smoking exposure, even though it does not take into consideration the other confounding risk factors for obesity.

Venn diagrams may be useful in future for displaying complex interactions between multiple exposure and outcomes. There is a need to consider the possibility of assessing how the impact of confounding factors could also be displayed along with these variables. The inclusion of option for adding risk factors also in Venn diagrams software along with assessment option for significance of associations could be helpful in future for generating more representative and proportional Venn diagrams. Even though Venn diagrams are quite useful, highly efficient and easy to use and provides a better visualisation of relationships between different health outcomes in relation to different exposures, it could appear difficult to interpret, when multi-circle Venn diagrams are created using numerous variables at the same time from complex datasets especially when creating proportional Venn diagrams. However, it seems to be one of the best and simplest methods, which could be used for visual illustration of data for the purpose of explaining health outcomes and associations and for health promotion and motivation among poorer communities and in educating the public about the ill effects of air pollution and smoking in pregnancy.

Venn diagrams created using software Venn Painter and Venn Plex could be used for producing more complex Venn diagrams (Lin et al, 2016; Cai et al, 2013). They are more useful as a comprehensive tool for identifying genes and the relationships between them or gene families in comparative analysis, which could be applied in exploring the association between different genes involved in childhood obesity resulting from exposure to air pollution and pregnancy smoking. Venn diagram software like BioVenn does not need to be downloaded and could be used as a web based alternative tool for creating proportional Venn diagrams irrespective of the operating system (Hulsen et al, 2008) compared to the other software mentioned above which needs to be downloaded and varies with different operating systems.

5.6.3 Population attributable risk for significant birth and child health outcomes after logistic regression

The PAR for childhood obesity associated with high NOx-PM_{10} emissions + MSDP was 44.4% (4.85%-77.97). This association might be explained by the additive effects of smoking and air pollution. The PAR for childhood croup associated with high NOx-PM_{10} emissions + PSDP was -42.86 (-5.26- -44.93) and with high NOx-PM_{10} emissions + HSDP was -45.87 (-55.05- -12.49). The underlying mechanism is unknown, although it is of interest that oxides of nitrogen, especially inhaled nitric oxide (iNO), have been used as the mainstay of treatment in cardio -respiratory conditions like persistent pulmonary hypertension (PPHN) in near-term and term infants and has reduced the need for Extra Corporeal membrane Oxygenation (ECMO) (Nair et al, 2014; Clark et al, 2000; Roberts et al, 1997).

5.6.4 Structural Equation Modelling for significant birth and child health outcomes

Reliable conclusions could not be drawn from structural equation modelling technique using this method as no goodness of fit statistics could be computed suggesting that at least some of the estimated coefficients (associations) were not be reliable. This may indicate that the model parameters were unsuitable or the proposed interactions were not adequately taking into consideration unknown parameters. Structural equation modelling analysis showed a clear potential association between the lower socio economic status and air pollution and also with smoking.This needs to be explored further in future and larger sample sizes will need to be considered for improving the fit and to provide sufficient statistical power and precise estimates.

5.7 Multiple statistical testing using different methods

Multiple statistical testing using different methods helps to confirm and to check for consistency of the significant associations across the results, which could also be helpful in explaining the positive findings and giving more strength to the final conclusions. For example, in the present study, the birth and child health outcomes were assessed in relation to individual and combined categories of air pollution exposure and air pollution combined with pregnancy smoking exposure using different methods. These methods included univariate analysis, comparison of means emissions for NOx and PM_{10} and concentrations for NO_2 and PM_{10} in

relation to high and low prevalence of birth and child health outcomes, assessment of growth characteristics in relation to these exposure categories, backward stepwise logistic regression, population attributable risk and Venn diagrams. The present study did show consistency in the significant associations for some of the outcomes (for example: childhood obesity and croup) in relation to similar individual categories for air pollution and with combined air pollution and pregnancy smoking exposure categories across the results obtained by using these different statistical methods as mentioned above, and also even after adjustment for confounding factors.

Results showing p values less than 0.05 suggest that the significance is unlikely due to chance and the significant results are further strengthened if the consistency remains the same and uniform across the different tests conducted. However multiple statistical tests increases the possibility of false positive findings. There is need to focus on the study quality and actual effect size along with supporting data from other studies rather than concentrating on the statistical significance of the study alone. Various approaches for correcting overall alpha error (occurs when the null hypothesis is true, but is rejected) include Bonferroni, Holm and Hochberg methods and the latter two are more superior as a first choice with large number of tests. The Hochberg procedure is useful to control false significant results among significant results.

5.8 Air pollutants and mixed interactions

There are interactions amongst air pollutants that change the toxicity of the mixture. These occur at the level of physicochemical interactions in air as well at the biological level. In developing air quality policies the following issues can be considered: the little evidence from health studies that the mixture of air pollutants results in significantly more health effects (synergy) than would be expected based on the information for the single pollutants. This is partly due to insufficient data and methodological limitations. The present study is one of the very few epidemiologic studies that have examined the mixed interactions between air pollutants and tobacco smoke at different levels. The existence of such pollutant mixtures makes it often difficult, in uncontrolled settings, to determine either independent or synergistic effects of ambient air pollutants. There is some evidence of potential interactions amongst pollutants with high temperatures. It is mainly the smaller sized particles, less than 2.5 microns that may cause most health problems (Mahoney, 2004). Changing the air pollution mixture due to changing fuels may under certain conditions lead to more harmful emissions. RCP and

RCPCH Joint Air Pollution Report (2016) reports that air pollution plays a key role in the process of climatic change and that measures taken to decrease air pollution could help in slow down the overheating of our planet.

5.9 Traffic related air pollution

With advances in technology resulting in the establishment of manufacturing industries and rapid proliferation in motor vehicles on roads resulting in busy traffic and emissions both in developing and developed countries, a spectrum of atmospheric pollutants has emerged including ozone, oxides of Nitrogen (NOx) and particulate matter. It may be challenging to adopt an air pollution free environment and a smoke free policy, because of the increase in the amount of traffic over the past few years, which still depend on fuels which releases lots of gases and particles into the atmosphere along with changing patterns of industrialisation and smoking sources. This is an emerging problem especially in developing countries where there is increased population growth along with limited living places leading to overcrowding and more family members exposed to indoor air pollution from smoking households' particularly pregnant women and children.

Brunekreef and Holgate (2002) in a review on "Air pollution and Health" which included studies from Europe, US and Canada stated adverse health effects occured even at lower air pollutant concentrations and that the health effects of air pollution will need to receive much scientific and regulatory interest for years to come. Traffic-related pollutant levels may also be considerably higher during the morning hours, especially when temperature variations occur largely during winter months (Kim et al, 2002; Ning et al, 2007). There is need for keeping air pollutant emissions from traffic to a minimum. Newer technological solutions such as the use of catalytic converters, oxidation catalysts, carbon canisters, exhaust gas re-circulation and electric transport might be helpful in providing clean air for the next few years, but do not provide a long term solution to transport related urban air pollution. Educating, encouraging and motivating the public to use vehicles with less air pollutant emissions and further development of less harmful alternative fuels, combined with effective management systems to reduce traffic and control traffic related air pollution are useful measures to tackle this problem in future. The next chapter provides a conclusion and includes related sections on future research, strength and limitations.

CHAPTER 6

CONCLUSION, FUTURE RESEARCH AND IMPLICATIONS

6.1 Summary and key conclusions

Combined high NOx-PM_{10} emissions and maternal smoking during pregnancy was associated with increased risk of childhood obesity. There was a decreased croup with combined exposure to air pollution and paternal smoking during pregnancy, or combined air pollution and household smoking during pregnancy. To conclude, combined pregnancy smoking and air pollution was associated with increased childhood obesity and decreased croup. Interventions to reduce air pollution and smoking during pregnancy could lead to better health outcomes among mothers and children in this area.

6.2 What this thesis adds

This study provides evidence that there could be an association between air pollution and pregnancy smoking exposure and birth and child health outcomes especially childhood obesity. It also provides some insight into possible mechanisms underlying these associations. It emphasises the need for control measures to be taken to protect children from adverse effects of pregnancy smoking and air pollution and the need for continued surveillance to address these public health problems using linked and combined data sets. This thesis has identified innovative ways to monitor these effects and has explored the possibility of the potential use of Venn diagrams for illustration of birth and child health outcomes in relation to pregnancy smoking and environmental pollution exposures in future which could be used as a preventive public health intervention tool. This study has also identified the method of linking of two different datasets with common variables for assessing outcomes in relation to exposures, which could be tried at a national level.

6.3 Preventive measures for air pollution and smoking in pregnancy

Long-term improvement in air quality could have significant positive clinical effects on child health. There are two complementary aspects to any air quality management programme. The long-term control involves measuring levels of air pollution, identifying its sources and then implementing measures to control and monitor its effectiveness (Mahoney, 2004; Mahoney, 2008). The Local Councils in UK have the right to introduce extra controls on emissions, if air quality problems are detected in air pollution prone areas. If the air quality

falls below required standards, the Council will declare this area as an Air Quality Management area and will develop an action plan to work towards achieving compliance and for improvements in future. In Liverpool, Sefton council has a statutory duty to undertake regular review and assessments of air quality across the borough to determine whether or not objectives set by the Government in the National Air Quality strategy will be complied with. Stewart et al (2015) suggested that future smoking cessation programs should pay more attention to addressing socio-demographic and cultural factors that influence the behaviour of maternal smokers. Although pregnancy is a critical period which offers multiple windows of opportunity for smoking cessation interventions, actions for smoking cessation in pregnancy can be difficult to design. The Ewles and Simnet model of health promotion planning and evaluation is a suitable model for a smoking cessation program (Ewles and Simnet, 2003). This makes use of the medical, behavioural change, educational, empowerment and social change approaches to motivate pregnant women to stop smoking (Naidoo et al, 2004). There is a need to start with the woman's knowledge and concerns about pregnancy smoking. Prioritisation of prevention strategies in young adolescent girls is critical, especially in the Liverpool area which experiences high adolescent smoking prevalence. Broader initiatives also play an important role such as Local councils in the UK establishing wider smoke control areas.

Royal College of Physicians and Royal College of Paediatrics and Child health recommendations for action and research includes the need for everyone accepting the responsibility for reducing air pollution and proposes fourteen steps to achieve this goal, which includes acting now and thinking in the long term, educating professionals and public, promoting alternatives to cars fuelled by petrol and diesel, putting onus on the polluters, monitoring air pollution effectively, acting to protect the public health when air pollution levels are high, tackling inequality, protecting those most at risk, leading by example in the NHS for research, defining the economic impact of air pollution, quantifying the relationship between indoor air pollution and health, determining how the global trends are affecting air quality and developing newer technologies to improve air pollution monitoring (RCP and RCPCH Air Pollution Report, 2016).

The Liverpool's air quality has been reported as an "invisible public health emergency", and a new city two week festival called 'VENT' in collaboration with the University of Liverpool as part of the 'Liverpool Air project' was conducted between February 22nd and 5th March 2016 to raise the awareness of air quality among local population of Liverpool as part

of the programme which included free art, events and public forums based on the impact of long term exposure to microscopic man made pollution, their causes and related issues (VENT, 2016). The main aim of this project, funded by the European Cultural Foundation Research and Development grant and Granada Foundation and the Arts Council Grants was to create five new cultural products or public health works to facilitate public engagement on air quality issues in Liverpool.

6.4 Future work

6.4.1 Observations informing future work

Since obesity was higher among children born to mothers who smoked during pregnancy and were exposed to air pollution, there is need for basic research on underlying mechanisms. A challenge relates to the multiple factors which may impact on obesity risk. There is a need for future studies to consider important risk factors for childhood obesity such as nutritional factors, physical activity and maternal risk factors including obesity and diabetes.

6.4.2 Possible future research

There is need for better understanding into the mechanisms underlying the associations between air pollution and child health and the potentially additive interactions between pregnancy smoking and air pollution, including assessment of the long-term effects later in childhood. Improved co-ordination of survey data on environmental exposures and health at a national level is required. One approach would be to develop a generic model for analysing this type of data which might be replicable and suitable for future analyses and surveys. Different data health sets including NHS health data, pollution and climate data could be linked based on identical variables including post-code or post-code sectors and could be analysed on a national scale. For example, the Clinical Practice Research datalink (CPRD), a governmental not for profit research service jointly funded by the NHS National Institute for Health Research (NIHR) and the Medicines and Healthcare products Regulatory Agency (MHRA), a part of the Department of Health, have been providing anonymised primary care records for public health research since 1987, which has resulted in nearly 1700 publications and have led to improvement in drug safety, best practice and clinical guidelines (CPRD, 2016). Health data from previous years needs to be linked to air pollution data for more extended and detailed

analysis in order to obtain better results. Analysis of data after inclusion of other air pollutants such as SO_2, $PM_{2.5}$, CO and ozone would be helpful in addressing the confounding effects of these pollutants on various child health outcomes.

There is need for separate analysis of adult health from NHS records in relation to air pollution for comparison with child health data and calculation of PAR estimates. Logistic regression analysis and seasonality trends of air pollution in relation to health outcomes require analysis using advanced spatial mapping techniques. Collaboration with St Helens PCT/Environmental Research team who are exploring the climatic changes on air pollution and co-ordination of survey data on environmental exposures and health is a currently relevant initiative. Inclusion of more complex health data sets with multiple health outcomes could be useful in achieving better results.

Use of more advanced forms of Venn diagrams for diagrammatic representation of associations might be useful for analysis of complex datasets related to air pollution and health outcomes. There is need for similar studies on a national and international basis in future in order to make use of the unused datasets, which could be linked with related datasets for assessing health outcomes. Focussing on air pollution and child health outcomes in countries such as China and India, where air pollution rates are high are useful, and conducting systematic / Cochrane reviews and meta-analysis and comparing emissions from different countries are required. There is need to include data from different periods to compare the trends of birth and child health outcomes in relation to smoking habits and air pollution and considering the changes with different seasons on a regular basis in order to proximate control measures to current data.

The next survey as part of the Merseyside Community Child Health Surveys is due and needs to be carried out for assessing the trends of childhood asthma and obesity in relation to pregnancy smoking. It would be useful to use LUR (Land Use Regression) models, a commonly used method in air pollution epidemiology, which analyses the relationship between measured pollutant concentrations and surrounding land use characteristics. Future studies may need to include genetic data, to establish insight into the gene-environment interactions among children who are at increased risk of health problems from smoking or exposure to air pollutants.

6.4.3 Factors to consider when assessing the impact of air pollution

There is need to focus on understanding the associations between air quality and meteorological conditions and variations of emissions and concentrations and the transport and transformation of air pollutants in different regions in UK. There is also need for more specific indicators of air pollution and host biomarkers, which could help in diagnosing child health problems specifically caused by air pollutants by linking outcomes to specific biomarkers. Different sources of air pollution and their compounding effects on child health should be considered, to avoid confounding effects of different pollutants, varied composition as well as cigarette smoke. The presence of many sources of air pollution makes this a substantial challenge. Toxins released from smoking contribute to toxins present in air, which further influence the gas composition especially in confined spaces.

6.5 Benefits of this research

The results and information from this study would be helpful in motivating and creating awareness among local population especially pregnant women about the detrimental effects and combined risks from air pollution and smoking on their unborn child. Measures taken to reduce air pollution and maternal smoking would help to reduce related neonatal, infant and maternal morbidity and improve long-term health outcomes. The Venn diagrams might be considered for use in health promotion as part of smoking cessation programs in antenatal clinics in this area. Prevention of smoking early in pregnancy, especially heavy smoking, is an immediate public health challenge for young women living in the Merseyside area. Maternal smoking and air pollution forms an ideal target for intervention and there is a need for more resources for existing smoking cessation campaigns and air pollution control programs in order to achieve higher cure rates and substantially diminish current levels of smoking and air pollution related child health problems. This approach could increase the ability of public health professionals to identify early warnings of risks and to understand the potential effectiveness of interventions through innovative preventive health measures based on visual schematic outlines of risk profiles (eg, use of Venn diagrams). Immunological studies in children exposed to air borne dust pollution might be beneficial, especially in relation to allergic disease. The utility of annual data sources for linkage with on-going air pollution monitoring and evaluation activities in the Sefton area will need to be established.

6.6 Limitations of the study

A limitation of this study is its cross-sectional design, which does not help to establish the direction of the air pollution for associations with health outcomes. These also depend on duration and level of exposure which was not definitively established in the present study. There is no ideal definition for outcome variables such as obesity, asthma, and smoking and air pollution, and while the present study adopted ISAAC questions within its questionnaire format, it was not possible to harmonise definitions with other reports providing air pollution or pregnancy smoking data. This limits study comparability. Asthma and ADHD status were defined by questions based on reported doctor diagnosis and were not validated by family doctor, or hospital clinical data. Another limitation is that although some confounding factors were adequately controlled, absence of some relevant factors amongst the different datasets limited optimal statistical analysis. There is a possibility of survival bias, a form of selection bias in cross sectional studies like this because it is possible as it does not consider several other risk factors which could influence the outcomes. There is also the issue of the presence of unknown air pollution indicators. In the context of childhood obesity, potential unmeasured confounders included the detailed nutritional status (other than height and weight) and dietary intakes of children, as well as details on physical activity, energy expenditure, parental control, and parental obesity. Quantification of smoking habits in pregnancy was not possible and parental smoking habits may be misclassified if not supported by quantitative evidence of smoking exposure using cotinine estimations especially when combined with air pollution exposure in assessing the effects on birth and child health outcomes. The consistency in the significance of the selected outcomes in relation to the exposures across different methods of analysis suggest that the associations could be true and not chance findings.

Differences in the methodology, sample size, uniformity and types of surveys used in data analysis could add to difficulties when combining datasets. Seasonal differences in air pollution were not measured, as temporal data on climatic changes was not available for the 2006 survey. Conducting the study at the same time and same area could be helpful in reducing the differences in two datasets with regards to influencing factors such as climatic changes and socio-economic status. Decreasing compliance seems to be an emerging and always a challenging problem with cross-sectional surveys, which remains a persistent challenge in future and is an underlying limitation of the present analysis.

6.7 Strengths of the study

This study was unique as this project facilitated linkage of two different datasets with related variables from the same area in Liverpool for research on child health outcomes in relation to pregnancy smoking and air pollution exposures. The data contained an extensive range of variables including data on child health, birth outcomes, and maternal characteristics as well as detailed information on socio-economic and other relevant characteristics. This allowed analysis to explore associations between the exposure and outcomes using different methods. The application of post-code sectors to match data on air pollution and child health was possible as both studies were conducted in the same area of Sefton and during the same period and year.

6.8 Key recommendations

*There is need for creating awareness among the public about the adverse health effects of air pollution and pregnancy smoking on birth and child health outcomes and how this can be prevented by following simple measures. Health education and health promotion and community participation would be useful to combat the adverse effects at an early stage.

*Traffic related air pollution needs to be controlled through measures such as emissions restrictions, promoting the use of public transit and zero emission vehicles and limiting traffic with higher pollution from residential and school areas.

*There is need for measures to be taken to control air pollution by using alternative forms of fuel such as electricity. Stricter policies and regulations against improper use of fuel should be implemented especially in industrialized countries and highly polluted areas.

*Strengthening interventions to reduce fetal exposure to tobacco smoke during pregnancy is important, through improved education of parents and adolescents about the wider hazardous health effects of smoking on children's health and their interactions and health risks later in life.

*Creating awareness of the importance of healthy home atmosphere, free from air pollution or smoking exposure is essential along with the need for active involvement of household members in providing a clean pollution and smoke free environment for children and family members.

REFERENCES

AIRVIRO (2014) Air Quality Management Web based system available at http://www.smhi.se/ airviro accessed on 6th April 2016

Allen R, Larson T, Sheppard L, Wallace L, Liu LJ. Use of real-time light scattering data to estimate the contribution of infiltrated and indoor-generated particles to indoor air. *Environ Sci Technol* 2003; **37**:3484-3492

Amann M, Bertok I, Cofala J, Gyarfas F, Heyes C, Klimont Z et al. Baseline scenarios for the Clean Air for Europe (CAFE) Programme. Final report for the study on development of the baseline and policy scenarios and integrated assessment modeling framework for the Clean Air or Europe (CAFE) Programme-Lot 2005

American Legacy. Tobacco control in low SES populations. American Legacy Foundation 2010

Anderson HR, Ruggles R, Strachan DP, Austin JB, Burr M, Jeffs D, et al. Trends in prevalence of symptoms of asthma, hay fever, and eczema in 12-14 year olds in the British Isles, 1995-2002: questionnaire survey. *BMJ* 2004; **328**: 1052-3

Anderson HR, Favarato G, Atkinson R. Long-term exposure to outdoor air pollution and the prevalence of asthma: meta-analysis of multi-community prevalence studies. *Air Qual Atmos Health* 2013; **6**: 57–68.

Anderson HR, Ruggles R, Pandey KD, et al. Ambient particulate pollution and the world-wide prevalence of asthma, rhino-conjunctivitis and eczema in children: phase one of the International Study of Asthma and Allergies in Childhood (ISAAC). *Occup Environ Med* 2010; **67**: 293–300

Annesi-Maesano I, Moreau D, Caillaud D, Lavaud F, Le Moullec Y, Taytard A, et al. Residential proximity fine particles related to allergic sensitization and asthma in primary school children. *Resp Med* 2007; **101**:1721–1729

Anto JM, Sunyer J, Rodriguez-Roisin R, Suarez-Cervera M, Vazquez L, and the Toxico-epidemiological Committee. Community outbreaks of asthma associated with inhalation of soybean dust. *N Engl J Med* 1989; **320**: 1097-1102

Asher MI, Martinez F, Keil U, Anderson HR, Beasley R, Crane J, et al. International Study of Asthma and Allergies in Childhood (ISAAC): *Eur Respir J* 1995; **8**:483-91

Asmussen I. Ultrastructure of the villi and fetal capillaries in placentas from smoking and non-smoking mothers. *Br J Obstet Gynaecol* 1980; **87**:239-45

Atkinson AB. How should we measure poverty? Some conceptual issues. Discussion Paper No. 82, ESRC Programme on taxation, incentives and distribution of income. London School of Economics; 1985a

Atkinson AB. On the measurement of poverty. Discussion Paper No. 90, ESRC Programme on Taxation, Incentives and the Distribution of Income. London School of Economics. 1985b

Ayres J. Cardiovascular disease and air pollution: a report by the committee on the medical effects of air pollutants. UK Department of Health 2006

Bailey EJ, Chang A. In children with prolonged cough, does treatment with antibiotics have a better effect on cough resolution than no treatment?: Part A: Evidence-based answer and summary. *Paediatrics & Child Health*. 2008; **13**:512-513.

Banerjee A, Mondal NK, Das D, Ray MR. Neutrophilic inflammatory response and oxidative stress in premenopausal women chronically exposed to indoor air pollution from biomass burning. *Inflammation* 2012; **35**: 671–683

Barker DJ. The fetal origins and infant origins of adult disease. *BMJ* 1990; **301**:111-12

Barreto M, Villa MP, Martella S, Ronchetti F, Darder MT, Falasca C et al. Exhaled nitric oxide in asthmatic and non-asthmatic children: influence of type of allergen sensitization and exposure to tobacco smoke. *Pediatr Allergy Immunol* 2001; **12**:247–256

Basu R, Woodruff TJ, Parker JD, Saulnier L, Schoendorf KC. Comparing exposure metrics in the relationship between $PM_{2.5}$ and birth weight in California. *J Expo Anal Environ Epidemiol* 2004; **14**:391–396

Bateson TF, Schwartz J. Who is sensitive to the effects of particulate air pollution on mortality? A case-crossover analysis of effect modifiers. *Epidemiology* 2004; **15**:143–149

Beelen R, Hoek G, Houthuijs D, Van den-Brandt PA, Goldbohm RA, Fischer P et al. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. *Occup Environ Med* 2009; **66**: 243–50

Bekö, G, Morrison, GC, Weschler, CJ et al. Measurements of dermal uptake of nicotine directly from air and clothing. in Proceedings of Indoor Air 2016., 241

Belanger K, Gent JF, Triche EW, Bracken MB, Leaderer BP. Association of indoor nitrogen dioxide exposure with respiratory symptoms in children with asthma. *Am J Respir Crit Care Med* 2006; **173**:297–303

Bellinger DC. Neurological and behavioral consequences of childhood lead exposure. *PLoS Med* 2008; **5**:115

Bellettiere J, Hughes SC, Liles S., Boman-Davis M, Klepeis N, Blumberg E et al. Developing and selecting auditory warnings for real-time behavioral intervention. *Am. J. Public Health Res* 2014; 2:232–238. doi: 10.12691/ajphr-2-6-3

Bentayeb M, Billionnet C, Baiz N, Derbez M, Kirchner S, Annesi-Maesano I. Higher prevalence of breathlessness in elderly exposed to indoor aldehydes and VOCs in a representative sample of French dwellings. *Respir Med* 2013; **107**:1598-607

Birkett S. Clean air London. Breathtaking breach of NO_2 annual legal limit in eight days available at http://cleanair.london/legal/breathtaking-breach-of-no2-annual-legal-limit-in-eight-days/#sthash.eBViMFS1.dpuf accessed on 12th March 2016

Björkstén F, Haahtela T, Hannuksela M. Assay of allergen preparations using direct RAST titration and skin tests. *Allergy* 1980; **35**:233-4

Blaiss M. Current concepts and therapeutic strategies for allergic rhinitis in school-age children. *Clin Ther* 2004; **26**:1876–1889

Bluestone CD, Klein JO. Otitis media with effusion, atelectasis, and Eustachian tube dysfunction. In: Bluestone CD, Stool SE, editors. *Pediatric Otolaryngology* Philadelphia: WB. Saunders 1983; 356–512

Bollati V, Baccarelli A. Environmental epigenetics. *Heredity* 2010; **105**:105–12.

Brauer M, Gehring U, Brunekreef B, de Jongste J, Gerritsen J, Rovers M, et al. Traffic-related air pollution and otitis media. *Environ Health Perspect* 2006; **114**:1414–1418

Braun-Fahrlander C, Ackermann-Liebrich U, Schwartz J, Gnehm HP, Rutishauser M, Wanner HU. Air pollution and respiratory symptoms in pre-school children. *Am Rev Respir Dis* 1992; **145**:42–47

Brauer M, Hoek G, Smit HA, de Jongste JC, Gerritsen J, Postma DS et al. Air pollution and development of asthma, allergy and infections in a birth cohort. *Eur Respir J* 2007; **29**:879–888

Breysse PN, Buckley TJ, Williams D, Beck CM, Jo SJ, Merriman B et al. Indoor exposures to air pollutants and allergens in the homes of asthmatic children in inner-city Baltimore. *Environ Res* 2005; **98**:167–176

Brockmeyer S, D'Angiulli A. . How air pollution alters brain development: the role of neuro-inflammation. Transl Neurosci 2016 **21**; **7**:24-30

Brook RD, Rajagopalan S, Pope CA, Brook JR, Bhatnagar A, Diez-Roux AV et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 2010; **121**: 2331–2378

Brunekreef B, Holgate ST. Air pollution and health. *Lancet* 2002; **360**:1233–1242

Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiol* 1997; **8**:298–303

Brunekreef B, Stewart AW, Anderson HR, Lai CK, Strachan DP, Pearce N. Self-reported truck traffic on the street of residence and symptoms of asthma and allergic disease: a global relationship in ISAAC Phase 3. *Environ Health Perspect* 2009; **117**:1791–1798

Buchdahl R, Parker A, Stebbings T, Babiker A. Association between air pollution and acute childhood wheezy episodes: prospective observational study. *BMJ* 1996; **312**:661-5

Bundred P, Kitchiner D, Buchan I. Prevalence of overweight and obese children between 1989 and 1998: Population based series of cross sectional studies. *BMJ* 2001; **322**:326-8

Burnett RT, Smith-Doiron D, Stieb D, Cakmak S, Brook JR. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. *Arch Environ Health* 1999; **54**:130–139

Butz AM, Matsui EC, Breysse P, Curtin-Brosnan J, Eggleston P, Diette G, et al. A randomized trial of air cleaners and a health coach to improve indoor air quality for inner-city children with asthma and secondhand smoke exposure. *Arch Pediatr Adolesc Med* 2011; **165:741**–748

Burr ML. Pollution: Does it cause asthma? *Arch Dis Child* 1995; **72**:377–379

Cadle SH, Mulawa P, Hunsanger EC, *et al.* Light-duty motor vehicle exhaust particulate matter measurement in the Denver, Colorado area. *J Air Waste Manage Assoc* 1999; **49:164**–74

Cai H, Chen H, Yi T, Daimon CM, Boyle JP, Peers C, Maudsley S, Martin B. VennPlex-a novel
Venn diagram program for comparing and visualizing datasets with differentially regulated data points. *PLoS One* 2013; **8**: e53388

Cakmak S, Dales RE, Judek S. Do gender, education, and income modify the effect of air pollution gases on cardiac disease? *J Occup Environ Med* 2006; **48**: 89–94

Calderón-Garcidueñas L, Reed W, Maronpot RR, et al. Brain inflammation and Alzheimer's-like pathology in individuals exposed to severe air pollution. *Toxicol Pathol* 2004; **32**: 650–658

Castano-Vinyals G, D'Errico A, Malats N, Kogevinas M. Biomarkers of exposure to polycyclic aromatic hydrocarbons from environmental air pollution. *Occup Environ Med* 2004; **61**:e12

Cesaroni G, Forastriere F, Stafoggia M, Andersen ZJ, Badaloni C, Beelen R, et al. Long-term exposure to ambient air pollution and incidence of acute coronary events-Analysis of eleven European cohorts from the ESCAPE Project. *BMJ* 2014; **348**: f7421

Centers for Disease Control and Prevention (CDC). *MMWR* 2001; **50**: 682-686

CERC : Air Quality Modelling and Source Apportionment Report: Air Quality Modelling for Sefton Final Report 2010

Chaix B, Gustafsson S, Jerrett M, et al. Children's exposure to nitrogen dioxide in Sweden: investigating environmental injustice in an egalitarian country. *J Epidemiol Community Health* 2006; **60**:234-241

Chastang J, Baïz N, Cadwallader JS, Robert S, Dywer J, Charpin DA, et al. Postnatal environmental tobacco smoke exposure related to behavioral problems in children. *PLoS ONE* 2015; **10**:8 e0133604.

Chauhan AJ, Sebastian LJ. Air pollution and infection in respiratory illness. *Brit Med Bulletin* 2003; **68**: 95–112

Cheng TM. Taiwan's new national health insurance program: genesis and experience so far. *Health Aff* 2003; **22**: 61-7

Chen W, Guo XB. Advance in research on ambient particulate matter and cardiovascular diseases. *J Environ Health* 2005; **22**: 488–491

Chen XK, Cheng HM, Luo HL. Index assessment of airborne VOCs pollution in automobile for transporting passengers. *Huan Jing Ke Xue* 2013; **34**:4599-604

Chimonas MAR, Gessner BD. Airborne particulate matter from primarily geologic, non-industrial sources at levels below National Ambient Air Quality Standards is associated with outpatient visits for asthma and quick-relief medication prescriptions among children less than 20 years old enrolled in Medicaid in Anchorage, Alaska. *Environ Research* 2007; **103**:397–404

Choudhuri S, Cui Y, Klaassen CD. Molecular targets of epigenetic regulation and effectors of environmental influences. *Toxicol Appl Pharmacol* 2010; **245**: 378–93

Ciccone G, Forastiere F, Agabiti N, et al. Road traffic and adverse respiratory effects in children. SIDRIA Collaborative Group. *Occup Environ Med* 1998; **55**:771–778

Clark RH, Kueser TJ, Walker MW, Southgate WM, Huckaby JL, Perez JA, et al. Low-dose nitric oxide therapy for persistent pulmonary hypertension of the newborn. Clinical Inhaled Nitric Oxide Research Group. *N Engl J Med* 2000; **342**:469–74

COMEAP Committee on the Medical Effects of Air Pollutants (2015) available at https://www.gov.uk/government/groups/committee-on-the-medical-effects-of-air-pollutants-comeap accessed on 9th October 2015

Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. Health effects of outdoor air pollution. *Am J Respir Crit Care Med* 1996; **153**: 3–50

Cooper C, David FC, Alley 1986. Air Pollution Control: A Design Approach. Prospect Heights, Waveland Press.

Connolly G, Carpenter CM, Travers MJ, Cummings KM, Hyland A, Mulcahy M, Clancy L. How smokefree laws improve air quality: A global study of Irish pubs. *Nicotine Tob Res* 2009; **11:**600–605

Coronas MV, Horn RC, Ducatti A, Rocha JV, Vargas VM.Mutagenic activity of airborne particulate matter in a petrochemical industrial area. *Mutat Res* 2008; **650**: 196-201

Cortés J, González CM, Morales L, Abalos M, Abad E, Aristizábal BH. PCDD/PCDF and dl-PCB in the ambient air of a tropical Andean city: passive and active sampling measurements near industrial and vehicular pollution sources. *Sci Total Environ* 2014; **1**:491-492

Clinical Practice Research Datalink (CPRD) 2017 Medicines and Health care products Regulatory agency available at https://www.cprd.com/intro.asp accessed on 24-01-2017

Crum EJ, McDougall CM. Changing prevalence of overweight and obesity in primary schoolchildren: a glimmer of hope? *SMJ* 2006; **51**: 34-36

Da Costa JL, Navarro A, Neves JB, Martin M. Household wood and charcoal smoke increases risk of otitis media in childhood in Maputo. *Int J Epidemiol* 2004; **33**:573–578

Dėdelė A, Miškinytė A. Seasonal variation of indoor and outdoor air quality of nitrogen dioxide in homes with gas and electric stoves *Environ Sci Pollut Res* 2016;23:17784–17792

Daly A, Zannetti P. An introduction to air pollution - definitions, classifications, and history. Chapter 1 of Ambient Air Pollution. The Arab School for Science and Technology and Enviro Comp Institute: Freemont, CA, USA 2007

Danesh J, Gault S, Semmence et al. Postcodes as useful markers of social class: population based study in 26000 British households. *BMJ* 1999; **318**: 843-4

Datson H & Birch WJ. The development of a novel method for directional dust monitoring Environmental Monitoring and Assessment 2007; **124**:301-308
DEFRA (2006). A Review of the UK Urban Network for the measurement of black smoke, SO2 and NO_2.

DEFRA (2009a). The UK Air Quality Strategy 2000 available at http://www.air-quality.org.uk/20.php accessed on 16th January 2014

DEFRA (2009). Local Air Quality Management Technical Guidance (LAQM)

DEFRA (2008). Diffusion Tubes for Ambient NO_2 monitoring: Practical Guidance for Laboratories and Users. AEA Technology Report.

Delfino RJ, Staimer N, Gillen D, Tjoa T, Sioutas C, Fung K, et al. Personal and ambient air pollution is associated with increased exhaled nitric oxide in children with asthma. *Environ Health Perspect* 2006; **114**: 1736–1743

Delfino RJ, Gong H, Linn WS, Hu Y, Pellizzari ED. Respiratory symptoms and peak expiratory flow in children with asthma in relation to volatile organic compounds in exhaled breath and ambient air. *J Expo Anal Environ Epidemiol* 2003a; **13**:348–363

Delfino RJ, Gong H, Jr, Linn WS, Pellizzari ED, Hu Y. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. *Environ Health Perspect* 2003b; **111**:647–656

Delpisheh A, Attia E, Drammond S, Brabin BJ. Adolescent smoking in pregnancy and birth outcomes. *Eur J Public Health* 2006a; **16**:168-72

Delpisheh A, Topping J, Reyad M, Tang A, Brabin BJ. Smoking exposure in pregnancy: use of salivary cotinine in monitoring. *Brit J Midwif* 2007; **15**: 216–20

Delpisheh, A, Brabin L, Attia E, Brabin BJ. 'Pregnancy late in life: A hospital-based study of birth outcomes'. *J Women's Health* 2008a; **17**: 965-970

Delpisheh A, Brabin L, Brabin BJ. Pregnancy, smoking and birth outcomes. *Womens Health* 2006b; **2**: 389-403

Delpisheh A, Brabin L, Drummond S, Brabin BJ. Prenatal smoking exposure and asymmetric fetal growth restriction. *Ann Hum Biol* 2008c; **35**: 573-83

Delpisheh A, Brabin L, Topping J, Reyad M, Tang AW, Brabin BJ. A case-control study of CYP1A1, GSTT1 and GSTM1 gene polymorphisms, pregnancy smoking and fetal growth restriction. *Eur J Obstet Gyn R B* 2009; **143**: 38-42

Delpisheh A, Kelly Y, Brabin BJ. Passive cigarette smoke exposure in primary school children in Liverpool. *Public health* 2006c; **120**: 65-9

Delpisheh A, Kelly Y, Rizwan S, Attia E, Drammond S, Brabin BJ. Population attributable risk for adverse pregnancy outcomes related to smoking in adolescents and adults. *Public health* 2007; **121**: 861-8

Delpisheh A, Kelly Y, Rizwan S, Brabin BJ. Socio-economic status, smoking during pregnancy and birth outcomes: an analysis of cross-sectional community studies in Liverpool (1993-2001). *J Child Health Care* 2006d; **10**: 140-8

Delpisheh A, Kelly Y, Rizwan S, Brabin BJ. Salivary cotinine, doctor-diagnosed asthma and respiratory symptoms in primary schoolchildren. *Matern Child Health* J 2008b; **12**: 188-93

Demissie K, Liu S, Wen SW, Marcoux S, Kramer MS. Maternal asthma and pregnancy outcomes: a retrospective cohort study. *Am J Obstet Gynecol* 2001; **184**: 90–96

Dewan N, Brabin B, Wood L, Dramond S, Cooper C. The effects of smoking on birthweight-for-gestational-age curves in teenage and adult primigravidae. *Public health* 2003; **117:**31-5

Devalia JL, Rusznak C, Herdman MJ, Trigg CJ, Tarraf H, Davies RJ. Effect of nitrogen dioxide and sulphur dioxide on airway response of mild asthmatic patients to allergen inhalation. *Lancet* 1994; **344**:1668–1671.

Diamanti-Kandarakis E, Bourguignon JP, Giudice LC, Hauser R, Prins GS, Soto AM et al. Endocrine-disrupting chemicals: an Endocrine Society scientific statement. *Endocr Rev* 2009; **30**: 293-342

Diette GB, Hansel NN, Buckley TJ, Curtin-Brosnan J, Eggleston PA, Matsui EC et al. Home indoor pollutant exposures among inner-city children with and without asthma. *Environ Health Perspect* 2007; **115**:1665–1669

Diette GB, McCormack MC, Hansel NN, Breysse PN, Matsui EC. Environmental issues in managing asthma. *Respir Care* 2008; **53**: 602–615

Ding YS, Trommel JS, Yan XJ, Ashley D, Watson CH. Determination of 14 polycyclic aromatic hydrocarbons in mainstream smoke from domestic cigarettes. *Environ Sci Technol* 2005; **39**: 471-8

Djordjevic MV, Stellman SD, Zang E. Doses of nicotine and lung coarcinogens delivered to cigarette smokers. *J Natl Cancer Inst* 2000; **92**:106-111

Dockery DW, Ware JH, Ferris BG Jr, Speizer FE, Cook NR, Herman SM. Change in pulmonary function in children associated with air pollution episodes. *J Air Pollut Control Assoc* 1982; **32**: 937-42

Dockery DW, Pope CA. Acute respiratory effects of particulate air pollution. *Ann Rev Public Health* 1994; **15**: 107-32

Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG. 1989. Effects of inhalable particles on respiratory health of children. *Am Rev Respir Dis* 1989; **139**:587–594

Dodge R, Solomon P, Moyers J, Hayes C. A longitudinal study of children exposed to sulfur oxides. *Am J Epidemiol* 1985; **121**: 720-36

Donoso LA, Kim D, Frost A, Callahan A, Hageman G. The role of inflammation in the pathogenesis of age-related macular degeneration. *Surv Ophthalmol* 2006; **51**, 137–152

D'Souza-Vazirani D, Minkovitz CS, Strobino DM. Validity of maternal report of acute health care use for children younger than 3 years. *Arch Pediatr Adolesc Med* 2005; **159**: 167–172

Du Y, Xu X, Chu M, Guo Y, Wang J. Air particulate matter and cardiovascular disease: the epidemiological, biomedical and clinical evidence. *J Thorac Dis*. 2016; **8:**8-19

Duhme H, Weiland SK, Keil U, Kraemer B, Schmid M, Stender M, et al. The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents. *Epidemiol* 1996; **7**: 578–582

Durham S. ABC of allergies, Summer hay fever. *BMJ* 1998; **316**: 843-845

Eakin MN, Rand CS, Borrelli B, Bilderback A, Hovell M, Riekert KA. Effectiveness of motivational interviewing to reduce head start children's secondhand smoke exposure. A randomized clinical trial. *Am J Respir Crit Care Med* 2014; **189:**1530–1537

Eder W, Ege MJ, von Mutius E. The asthma epidemic. *N Engl J Med* 2006; **355**: 2226–2235

Edward AWF. Cogwheels of the Mind: the story of Venn diagrams, Johns Hopkins University Press, Baltimore and London 2004

Ellegard A. Cooking fuel smoke and respiratory symptoms among women in low income areas in Maputo. *Envir Health Perspect* 1996; **104**:980–985

Elliott P, Wartenberg D. Spatial Epidemiology: Current Approaches and Future Challenges. *Envir Health Perspect* 2004; **112**:998-1006

English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L. Examining associations between childhood asthma and traffic flow using a geographic information system. *Environ Health Perspect* 1999; **107**:761–767

Environmental Protection Agency (US) Indoor air quality: basic information: carbon monoxide (CO) available at http://www.epa.gov/iaq/co.html accessed on 21st October 2015

Environmental Protection Agency. Air Quality Index (AQI): a guide to air quality and your health (2009) available at http:// airnow.gov/index. cfm?action= aqibasics. aqi accessed on 21st October 2015

Environmental Protection Agency. National Ambient Air Quality Standards. Office of Air and Radiation 2008 available at http://www.epa.gov/air/criteria.html. accessed on 18th Jan 2016

Ernst A, Zibrak JD. Carbon monoxide poisoning. *N Eng J Med* 1998; **339**:1603–8

ESCAPE (European Study of Cohorts for Air Pollution Effects) 2009. Home page-avaiable at http://www.escapeproject.eu/ accessed on 22 November 2014

Esplugues A, Ballester F, Estarlich M, Llop S, Fuentes V, Mantilla E, Iñiguez C. Indoor and outdoor concentrations and determinants of NO2 in a cohort of 1-year-old children in Valencia, Spain. *Indoor Air* 2010; **20**:213-23
Ewles and Simnett. Promoting health: a practical guide. 5th ed. Edinbugh: Balliere Tindall, 2003

Ezzaine Z. The impact of air pollution on low birth weight and infant mortality. *Reviews on Environ Health* 2013; **28**:107–115

Faulk C, Dolinoy DC. Timing is everything: the when and how of environmentally induced changes in the epigenome of animals. *Epigenetics* 2011; **6:**791–7

Feleszko W, Ruszczyński M, Jaworska J, *et al*. Environmental tobacco smoke exposure and risk of allergic sensitisation in children: a systematic review and meta-analysis. *Arch Dis Child* 2014; **99:**985-992

Finkelstein MM, Jerrett M, DeLuca P, Finkelstein N, Verma DK, Chapman K, et al. Relation between income, air pollution and mortality: a cohort study. *CMAJ* 2003; **169**: 397–402

Fonken LK, Xu X, Weil ZM, et al. Air pollution impairs cognition, provokes depressive-like behaviors and alters hippocampal cytokine expression and morphology. *Mol Psych* 2011; **16**:987–995

Franklin PJ, Turner SW, Le Souef PN, Stick SM. Exhaled nitric oxide and asthma: complex interactions between atopy, airway responsiveness, and symptoms in a community population of children. *Thorax* 2003; **58**:1048–1052

Freire C, Ramos R, Puertas R, et al. Association of traffic-related air pollution with cognitive development in children. *J Epidemiol Commun H* 2010; **64**: 223–228

Friend K, Levy DT. Reductions in smoking prevalence and cigarette consumption associated with mass-media campaigns. *Health Educ Res* 2002; **17**:85-98

Frischer TM, Kuehr J, Pullwitt A, Meinert R, Forster J, Studnicka M et al. Ambient ozone causes upper airways inflammation in children. *Am Rev Respir Dis* 1993; **148**: 961-4

Fuertes E, Standl M, Cyrys J et al. A longitudinal analysis of associations between traffic-related air pollution with asthma, allergies and sensitization in the GINI plus and LISA plus birth cohorts. *Peer J* 2013; **1**:193

Gaga EO, Döğeroğlu T, Ozden O, Ari A, Yay OD, Altuğ H et al. Evaluation of air quality by passive and active sampling in an urban city in Turkey: current status and spatial analysis of air pollution exposure. *Environ Sci Pollut Res Int* 2012; **19**:3579-96

Gao Y, Chan EYY, Li L et al. Chronic effects of ambient air pollution on respiratory morbidities among Chinese children: a cross-sectional study in Hong Kong. *BMC Public Health* 2014; **14**: 105

Garrett MH, Hooper MA, Hooper BM, Abramson MJ. Respiratory symptoms in children and indoor exposure to nitrogen dioxide and gas stoves. *Am J Respir Crit Care Med* 1998; **158**: 891–895

Gasana J, Dillikar D, Mendy A, et al. Motor vehicle air pollution and asthma in children: a meta-analysis. *Environ Res* 2012; **117**: 36–45

Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, et al. Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiol* 2005; **16**: 737–743

Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, et al. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med* 2000; **162**: 1383–1390

Gent JF, Triche EW, Holford TR, Belanger K, Bracken MB, Beckett WS, et al. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA* 2003; **290**: 1859–1867

Gilboa A, Silberberg A. In situ rheological characterization of epithelial mucus. *Biorheology* 1976; **13**:59-65

Gillespie-Bennett J, Pierse N, Wickens K, Crane J, Howden-Chapman P. The respiratory health effects of nitrogen dioxide in children with asthma. *Eur Respir J* 2011; **38**:303–309

Gilthrope MS. The importance of normalisation in the construction of deprivation indices. *J Epidemiol Commun H* 1995; **492**: S45-S50

Gouveia N, Fletcher T.Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status. *J Epidemiol Commun H* 2000; **54**:750–755

Greater London Authority. London Atmospheric Emissions Inventory 2003. London, United Kingdom 2006

Grün F, Blumberg B: Endocrine disrupters as obesogens. *Mol Cell Endocrinol* 2009, **304**:19-29

Guxens M, Aguilera I, Ballester F, et al. Prenatal exposure to residential air pollution and infant mental development: modulation by antioxidants and detoxification factors. INMA (Infancia Medio Ambiente) Project. *Environ Health Perspect* 2011; **120**:144–149

Hajat S, Haines A, Atkinson RW, Bremner SA, Anderson HR, Emberlin J. Association between air pollution and daily consultations with general practioners for allergic rhinitis in London, United Kingdom. *Am J Epidemiol* 2001; **153**:704–714

Hansel NN, Breysse PN, McCormack MC, Matsui EC, Curtin-Brosnan J, Williams DL. A longitudinal study of indoor nitrogen dioxide levels and respiratory symptoms in inner-city children with Asthma. *Environ Health Perspect* 2008; **116**: 1428–1432

Hansen AM, Raaschou-Nielsen O, Knudsen LE. Urinary 1-hydroxypyrene in children living in city and rural residences in Denmark. *Sci Total Environ* 2005; **347**:98–105

Harutyunyan A, Movsisyan N, Petrosyan V, Petrosyan D, Stillman F. Reducing children's exposure to secondhand smoke at home: A randomized trial. *Pediatrics*. 2013; **132:**1071–1080

Hasselblad V, Eddy DM, Kotchmar DJ. Synthesis of environmental evidence: nitrogen dioxide epidemiology studies. *J Air Waste Manage Assoc* 1992; **42**:662–671

Health Canada (1995). Indoor air quality in office buildings: A technical guide, Health Canada, Ottawa.

Health Effects Institute. Assessing Health Impact of Air Quality Regulations: Concepts and Methods for Accountability Research. Boston, MA: Health Effects Institute 2003.

Health Effects Institute. Health effects of outdoor air pollution in developing countries of Asia: A literature review. Boston: Health Effects Institute 2004

HEI (2010) Traffic-related Air Pollution: A Critical review of the literature on emissions, Exposure, and Health Effects. Special Report 17. Health Effects Institute, Boston, MA.

Heinrich J, Wichmann HE. Traffic related pollutants in Europe and their effect on allergic disease. *Curr Opin Allergy Clin Immunol* 2004; **4**:341–348

Heikkinen T, Thint M, Chonmaitree T. Prevalence of various respiratory viruses in the middle ear during acute otitis media. *N Engl J Med* 1999; **340**:260–264

Heinrich J, Hoelscher B, Frye C, Meyer I, Pitz M, Cyrys J, et al. Improved air quality in reunified Germany and decreases in respiratory symptoms. *Epidemiol* 2002; **13**:394–401

Heinrich J, Raghuyamshi VS. Air pollution and otitis media: a review of the evidence from epidemiologic studies. *Curr Allergy Asthma Rep* 2004; **4**:302–309

Henderson DW. Venn diagrams for more than four classes. *American Mathematical Monthly* 1963; **70**: 424–6

Hendrie GA, Coveney J, Cox DN. Defining the complexity of childhood obesity and related behaviours within the family environment using structural equation modelling. *Public Health Nutr* 2012:**15**; 48-57

Herceg Z, Vaissière T. Epigenetic mechanisms and cancer: an interface between the environment and the genome. *Epigenetics* 2011; **6**:804–19

Herr CE, Nieden AZ, Stilianakis NI, Eikmann TF. Health effects associated with exposure to residential organic dust. *Am J Ind Med* 2004; **46**:381-5

Hiscock R, Linda Bauld, Amanda, Fidler JA, Marcus M. Socioeconomic status and smoking: a review. *Annals of the New York Academy of Sciences* 2012; **1248**:107-123

HMSO 1995b Particles. Department of the Environment Expert Panel on Air Quality Standards, HMSO, London

Hoek G, Brunekreef B. Effect of photochemical air pollution on acute respiratory symptoms in children. *Am J Respir Crit Care Med* 1995; **151**:27-32

Hoek G, Brunekreef B, Meijer R, Scholten A, Boleij J. Indoor nitrogen dioxide pollution and respiratory symptoms of schoolchildren. *Int Arch Occup Environ Health* 1984; **55**:79–86

Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. *Lancet* 2002; **360**:1203–1209

Hoffmann D; Hoffmann I (1998) "Chemistry and toxicity" in cigars: Health effects and trends, smoking and tobacco control monograph 9, National Institute of Health, National Cancer Institute, Bethesda

Hoffmann B, Moebus S, Mohlenkamp S, Stang A, Lehmann N, Dragano N et al. Residential exposure to traffic is associated with coronary atherosclerosis. *Circulation* 2007; **116**:489–496

Holguin F, Flores S, Ross Z, Cortez M, Molina M, Molina L, et al. Traffic-related exposures, airway function, inflammation, and respiratory symptoms in children. Am *J Respir Crit Care Med* 2007; **176**:1236–1242

Horak F Jr, Studnicka M, Gartner C et al. Particulate matter and lung function growth in children: a 3-yr follow-up study in Austrian school children. *Eur Respir J* 2002; **19**:838-45

Hovell M. Fresh Air. National Heart, Lung, and Blood Institute, National Institutes of Health, and Department of Health and Human Services; Bethesda, Maryland, USA: 2011. Grant R01 HL103684-02

Hou L, Wang D, Baccarelli A. Environmental chemicals and microRNAs. *Mutat Res* 2011; **714**:105–12

Huang H, Jasimah CW, Radzi WM, Jenatabadi HS. Family environment and childhood obesity: A new framework with structural equation modelling. *Int J Environ Res Public Health*. 2017; **14**: 181

Hulsen T, de Vlieg J, Alkema W. BioVenn - a web application for the comparison and visualization of biological lists using area-proportional Venn diagrams. *BMC Genomics* 2008; **9**:488

Hwang BF, Jaakkola JJK, Lee YL, Lin YC, Leon Guo Y. Relation between air pollution and allergic rhinitis in Taiwanese school children. *Resp Res* 2006;**7**: 23

Iglesias C, Torgerson D. Does length of questionnaire matter? A randomised trial of response rates to a mailed questionnaire. J Health Serv Res Policy 2000:**5**; 219–221

INEMAR 2010 Regional inventory of emissions into the atmosphere; 3rd edition.

Institute of Medicine Committee on the Assessment of Asthma and Indoor Air. Clearing the Air: Asthma and Indoor Air Exposures. Washington, DC: National Academy Press 2000

Invernizzi G1, Ruprecht A, Mazza R, Rossetti E, Sasco A, Nardini S, Boffi R. Particulate matter from tobacco versus diesel car exhaust: an educational perspective. *Tob Control* 2004; **13:219**-21

Ishizaki T, Koizumi K, Ikemori R, Ishiyama Y, Kushibiki E. Studies of prevalence of Japanese cedar pollinosis among the residents in a densely cultivated area. *Ann Allergy* 1987; **58**:265-70

Islam T, McConnell R, Gauderman WJ, Avol E, Peters JM, Gilliland FD. Ozone, oxidant defence genes, and risk of asthma during adolescence. *Am J Respir Crit Care Med* 2008; **177**:388–395

James LR (2007) "Exposure to Second hand Smoke," Chapter 9 in Ott, Steinemann, and Wallace, eds., Exposure Analysis, CRC-Press, Taylor & Francis Group, Boca Raton, FL

Janghorbani M, Piraei E. Association between air pollution and preterm birth among neonates born in Isfahan. *Iran J Res Med Sci* 2013; **18**: 875-81

Janssen NAH, Brunekreef B, Van Vliet P, Aarts F, Meliefste K, Harssema H et al. The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyper-responsiveness, and respiratory symptoms in Dutch schoolchildren. *Environ Health Perspect* 2003; **111**:1512–1518

Jerrett M, Burnett RT, Ma R, Pope CA, Krewski D, Newbold KB et al. Spatial analysis of air pollution and mortality in Los Angeles. *Epidemiol* 2005; **16**:727–736

Jerrett M, Burnett RT, Brook J, Kanaroglou P, Giovis C, Finkelstein N, et al. Do socioeconomic characteristics modify the short term association between air pollution and mortality? Evidence from a zonal time series in Hamilton, Canada. *J Epidemiol Commun Health* 2004; **58**:31–40

Johnson DL, Ambrose SH, Bassett TJ, Bowen ML, Crummey DE, Isaacson JS, "Meanings of environmental terms". *J Environ Qual* 1997; **26**: 581–589

Jörres R, Magnussen H. Airways response of asthmatics after a 30 min exposure, at resting ventilation, to 0.25 ppm NO_2 or 0.5 ppm SO2. *Eur Respir J* 1990; **3**:132-7

Jung CR, Lin YT, Hwang BF. Air pollution and newly diagnostic autism spectrum disorders: a population-based cohort study in Taiwan. *PLoS One* 2013; **8**: e75510

Just J, Segala C, Sahraoui F, Priol G, Grimfeld A, Neukirch F. Short-term health effects of particulate and photochemical air pollution in asthmatic children. *Eur Respir J* 2002; **20**:899–906

Kan H, London SJ, Chen G, Zhang Y, Song G, Zhao N et al. Season, sex, age, and education as modifiers of the effects of outdoor air pollution on daily mortality in Shanghai, China: the Public Health and Air Pollution in Asia (PAPA) study. *Environ Health Perspect* 2008; **116**:1183-1188

Karmaus W, Kuehr J, Kruse H. Infections and atopic disorders in childhood and organochlorine exposure. *Arch Environ Health* 2001; **56**:485–492

Karlberg J, Albertsson Wikland K. Growth in full-term small-for-gestational-age infants: from birth to final height. *Pediatr Res* 1995; **38**: 733-9

Kattan M, Gergen PJ, Eggleston P, Visness CM, Mitchell HE. Health effects of indoor nitrogen dioxide and passive smoking on urban asthmatic children. *J Allergy Clin Immunol* 2007; **120**:618–624

Kawanishi M, Watanabe T, Hagio S, Ogo S, Shimohara C, Jouchi R et al. Genotoxicity of 3,6-dinitrobenzopyrene, a novel mutagen in ambient air and surface soil, in mammalian cells in vitro and in vivo. *Mutagenesis* 2009; **24**:279-284

Kelishadi R, Hashemi M et al. Effect of particulate air pollution and passive smoking on surrogate biomarkers of endothelial dysfunction in healthy children. *Paediatr Int Child Health* 2014; **34**: 165-169

Kelly YJ, Brabin BJ, Milligan PJ, Reid JA, Heaf D, Pearson MG. Clinical significance of cough and wheeze in the diagnosis of asthma. *Arch Dis Child* 1996; **75**:489–493

Kelly YJ, Brabin BJ, Milligan P, Heaf DP, Reid J, Pearson MG. Maternal asthma, premature birth, and the risk of respiratory morbidity in school children in Merseyside. *Thorax* 1995; **50**:525-30

Kelly YJ (1997). Risk factors for respiratory morbidity in primary school children in Merseyside. PhD thesis, University of Liverpool.

Kim S, Shen S, Sioutas C. Size distribution and diurnal and seasonal trends of ultrafine particles in source and receptor sites of the Los Angeles basin. *J Air Waste Manag Assoc* 2002; **52**:297–307

Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, Ostro B. Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. *Am J Respir Crit Care Med* 2004; **170**:520–526

Kim CS, Alexis NE, Rappold AG, Kehrl H, Hazucha MJ, Lay JC, et al. Lung function and inflammatory responses in healthy young adults exposed to 0.06 ppm ozone for 6.6 hours. *Am J Respir Crit Care Med* 2011; **183**:1215–21

Kim HO, Kim JH, Cho SI, Chung BY, Ahn IS, Lee CH et al. Improvement of atopic dermatitis severity after reducing indoor air pollutants. *Ann Dermatol* 2013; **25**:292-7

Kharitonov SA, Robbins RA, Yates D, Keatings V, Barnes PJ. Acute and chronic effects of cigarette smoking on exhaled nitric oxide. *Am J Respir Crit Care Med* 1995; **152**:609–612

Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, Switzer P et al. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. *J Expo Anal Environ Epidemiol* 2001; **11**:231–252

Klepeis N, Hughes SC, Edwards RD, Allen T, Johnson M, Chowdhury Z. Promoting smoke-free homes: A novel behavioral intervention using real-time audio-visual feedback on airborne particle levels. *PLoS ONE*. 2013; **8** doi: 10.1371/journal.pone.0073251

Klepeis NE, Ott WR, Switzer P. Real-time measurement of outdoor tobacco smoke particles. *J Air and Waste Manag Assoc* 2007; **57**:522-534.

Koenig JQ, Covert DS, Hanley QS, Van Belle G, Pierson WE. Prior exposure to ozone potentiates subsequent response to sulfur dioxide in adolescent asthmatic subjects. *Am Rev Respir Dis* 1990; **141**:377-80

Koenig JQ, Pierson WE. Air pollutants and the respiratory system: toxicity and pharmacologic interventions. *J Toxicol Clin Toxicol* 1991; **29**:401-11

Koenig JQ, Mar TF, Allen RW, Jansen K, Lumley T, Sullivan JH et al. Pulmonary effects of indoor- and outdoor-generated particles in children with asthma. *Environ Health Perspect* 2005; **113**:499–503

Koltai PJ. Effects of air pollution on the upper respiratory tract of children. *J Otolaryngol Head Neck Surgery* 1994; **111**: 9-11

Kopp MV, Ulmer C, Ihorst G, Seydewistz HH, Frischer T, Forster J, et al. Upper airway inflammation in children exposued to ambient ozone and potential signs of adaptation. *Eur Respir J* 1999; **14**:854–861

Koshy G, Akrouf KA, Kelly Y, Delpisheh A, Brabin BJ. Asthma in children in relation to preterm birth and fetal growth restriction. *Matern Child Health J* 2013; **17**:1119-29

Koshy G, Delphisheh A, Brabin L, Attia E, Brabin BJ: Parental smoking and increased likelihood of female births. *Ann Hum Biol* 2010a, **37**:789–800

Koshy G, Brabin BJ. Parental compliance-an emerging problem in Liverpool community child health surveys 1991-2006. *BMC Med Res Methodol* 2012a; **12**:53

Koshy G, Delpisheh A, Brabin BJ. Trends in prevalence of childhood and parental asthma in Merseyside, 1991-2006. *J Public health* 2010b; **32**:488-95

Koshy G, Delpisheh A, Brabin BJ. Childhood obesity and parental smoking as risk factors for childhood ADHD in Liverpool children. *Atten Defic Hyperact Disord* 2011a; **3**:21-8

Koshy G, Delpisheh A, Brabin BJ. Dose response association of pregnancy cigarette smoke exposure, childhood stature, overweight and obesity. *Eur J Public health* 2011b; **21**: 286-91

Koshy G. Pregnancy Smoking, Child health and Nutrition. PhD thesis. Amsterdam University 2012b

Koren HS. Associations between criteria air pollutants and asthma. *Environ Health Perspect* 1995; **103**: 235–242

Kramer U, Koch T, Ranft U, Ring J, Behrendt H. Traffic-related air pollution is associated with atopy in children living in urban areas. *Epidemiol* 2000; **11**:64–70

Krewski D, Burnett RT, Goldberg M, Hoover K, Siemiatycki J, Abrahamowicz M, et al. Reanalysis of the Harvard Six Cities Study, part II: sensitivity analysis. *Inhal Toxicol* 2005; **17**:343–353

Kunzli N. The public health relevance of air pollution abatement. *Eur Respir J* 2002; **20**:198–209

Landau LI. Major provoking factors and their chemical assessment. Smoke and air pollution in childhood asthma and other wheezing disorders, Chapman & Hall, London 1995; 251-259

Landrigan PJ, Kimmel CA, Correa A, Eskenazi B. Children's health and the environment: public health issues and challenges for risk assessment. *Environ Health Perspect* 2004; **112**: 257-265

Lanphear BP, Hornung RW, Khoury J, Yolton K, Lierl M, Kalkbrenner A. Effects of HEPA air cleaners on unscheduled asthma visits and asthma symptoms for children exposed to secondhand tobacco smoke. *Pediatrics* 2011; **127**:93–101

Lee YL, Shaw CK, Su HJ, Lai JS, Ko YC, Huang SL, et al. Climate, traffic-related air pollutants and allergic rhinitis prevalence in middle-school children in Taiwan. *Eur Respir J* 2003; **21**:964–970

Leonardi GS, Houthuijs D, Steerenberg PA, Fletcher T, Armstrong B, Antova T et al. Immune biomarkers in relation to exposure to particulate matter: a cross-sectional survey in 17 cities of Central Europe. *Inhal Toxicol* 2000; **12**:1–14

Lebowitz MD. Airway responses of children to environmental irritants. *Pediatr Pulmonol* 1985; **1**: 235-6

Leon DA, Johansson M, Rasmussen F. Gestational age and growth rate of fetal mass are inversely associated with systolic blood pressure in young adults: an epidemiologic study of 165,136 Swedish men aged 18 years. *Am J Epidemiol* 2000; **152**:597–604

Levin BE, Dunn-Meynell AA The effect of cigarette smoking during pregnancy on fetal growth. *N Engl J Med* 1997; **125**: 267-71

Lewis GW, Ulph DT. Poverty, Inequality and Welfare. *The Economic Journal* 1988, **98**: 117-131.

Lippmann M, Chen LC, Gordon T, Ito K, Thurston GD. National Particle Component Toxicity (NPACT) Initiative: integrated epidemiologic and toxicologic studies of the health effects of particulate matter components. *Res Rep Health Eff Inst* 2013; **177**:5-13

Lindahl HE, Nordberg A. Smoking during pregnancy: A way to transfer the addiction to the next generation? *Respiration* 2002; **69**: 289-93

Lin M, Chen Y, Burnett RT, Villeneuve PJ, Krewski D. The influence of ambient coarse particulate matter on asthma hospitalization in children: case-crossover and time-series analyses. *Environ Health Perspect* 2002; **110**: 575-81

Li M, Qian Z, Vaughn M, Boutwell B, Ward P, Lu T, et al. Sex-specific difference of the association between ambient air pollution and the prevalence of obesity in Chinese adults from a high pollution range area: 33 communities Chinese health study. *Atmos Environ* 2015; **117**: 227–233

Lin G, Chai J, Yuan S, Mai C, Cai L Murphy RW, Zhou W, Luo J. Venn Painter: A tool for the comparison and identification of candidate genes based on venn diagrams. *PLoS One* 2016; **27**; **11**:e0154315

Lioy PJ, Avdenko M, Harkov R, Atherholt T, Daisey JM. A pilot indoor-outdoor study of organic particulate matter and particulate mutagenicity. *J Air Pollut Control Assoc* 1985; **35**: 653-7

Liu C, Flexeder C, Fuertes E, Cyrys J, Bauer CP, Koletzko S et al. Effects of air pollution on exhaled nitric oxide in children: Results from the GINIplus and LISAplus studies. *Int J Hyg Environ Health* 2014; **217**:483-491

Liu L, Poon R, Chen L, Frescura AM, Montuschi P, Ciabattoni G, Wheeler A, Dales R. Acute effects of air pollution on pulmonary function, airway inflammation, and oxidative stress in asthmatic children. *Environ Health Perspect* 2009; **117**: 668-74

Liverpool City Council Air Quality Report (2012) Air Quality updating and screening assessment for Liverpool City Council available at https://www.engageliverpool.com/wp-content /uploads /2015/ 08/Air-Quality-Assessment-Liverpool-2012.pdf accessed on 6th March 2016.

Long CM, Suh HH, Koutrakis P. Characterization of indoor particle sources using continuous mass and size monitors. *J Air Waste Manag Assoc* 2000; **50**:1236–1250.

Long W, Zhong T, Zhang B. China: The Issue of Residential Air Conditioning. 2007 available at http://www.iifiir. org/en/doc/ 1056. pdf. accessed on 21st October 2015

Lule SA, Webb EL, Ndibazza J, Nampijja M, Muhangi L, Akello F. Maternal recall of birthweight and birth size in Entebbe, Uganda. *Trop Med Internatio Health*, 2012; **17**: 1465–1469

MacIntyre EA, Gehring U, Mölter A, Fuertes E, Klümper C, Krämer U et al . Air pollution and respiratory infections during early childhood: an analysis of 10 European birth cohorts within the ESCAPE project. *Environ Health Perspect* 2014 **122**:107–113

MacIntyre EA, Karr CJ, Koehoorn M, Demers PA, Tamburic L, Lencar C. Residential air pollution and otitis media during the first two years of life. *Epidemiol* 2011; **22**:81-9

Mahoney G (2004) Aspects of Air Quality Management. PhD Thesis

Mahoney G (2008). Involving the community and schools in air quality monitoring: Beacon day- Open day presentation.

Mar TF, Jansen K, Shepherd K, Lumley T, Larson TV, Koenig JQ. Exhaled nitric oxide in children with asthma and short-term $PM_{2.5}$ exposure in Seattle. *Environ Health Perspect* 2005; **113**:1791–1794

Mazur B. Peak expiratory flow values in children relative to the degree of atmospheric air pollution. *Acta Paediatr* 1995; **84**:203–205

Malerbi FK, Martins LC, Saldiva PH, Braga AL, Ambient levels of air pollution induce clinical worsening of blepharitis. *Environ Res* 2012, **112**, 199–203

Mann JK, Balmes JR, Bruckner TA et al. Short-term effects of air pollution on wheeze in asthmatic children in Fresno, California. *Environ Health Perspect* 2010; **118**: 1497–1502

Martin P, Heavner DL, Nelson PR, Maiolo KC, Risner CH, Simmons PS, Morgan WT, Ogden MW. Environmental tobacco smoke (ETS): a market cigarette study. *Environ Int* 1997; **23**: 75–90

McCormack MC, Breysse PN, Hansel NN, Matsui EC, Tonorezos ES, Curtin-Brosnan J. Common household activities are associated with elevated particulate matter concentrations in bedrooms of inner-city Baltimore pre-school children. *Environ Res* 2008; **106**:148–155

McConnell R, Berhane K, Gilliland F, London SJ, Vora H, Avol E, et al. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect* 1999; **107**: 757–760

McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F et al. Traffic, susceptibility, and childhood asthma. *Environ Health Perspect* 2006; **114**:766–772

McConnell R, Shen E, Gilliland FD, Jerrett M, Wolch J, Chang CC, et al. A longitudinal cohort study of body mass index and childhood exposure to second hand tobacco smoke and air pollution: the southern California children's health study. *Environ Health Persp.* 2015; **123**:360–366

Mehdi P, Vijayaraj N, and Youping G. Venn - A web application for comparing gene lists using Venn diagrams. *Bioinformation* 2007; **1**: 420–2

Melia RJ, Florey CD, Chinn S, Morris RW, Goldstein BD, John HH, Clark D. Investigations into the relations between respiratory illness in children, gas cooking and nitrogen dioxide in the U.K. *Tokai J Exp Clin Med* 1985;**10**:375-8

Melia RJ, Florey CV, Morris RW, Goldstein BD, John HH, Clark D. Childhood respiratory illness and the home environment. Association between respiratory illness and nitrogen dioxide, temperature and relative humidity. *Int J Epidemiol* 1982; **11**:164-9.

Merseyside Atmospheric Emissions Inventory (2006) Sefton Council Environmental Protection Agency

Merseyside Atmospheric Emissions Inventory (2008) Sefton Council Environmental Protection Agency

Milligan P, Brabin BJ, Smith M, Benjamin C, Dunne E, Pearson M. Respiratory morbidity in Merseyside schoolchildren exposed to coal dust and air pollution. *Arch Dis Child* 1994; **70**:305–12

Milligan PJ, Brabin BJ, Kelly YJ, Pearson MG, Mahoney G, Dunne E, et al. Association of spatial distribution of childhood respiratory morbidity with environmental dust pollution. *J Toxico Enviro H* Part A 1998; **55**:169-84

Miyamoto T. Epidemiology of pollution induced airway disease in Japan. *Allergy* 1997; **52**:30-4

Molfino NA, Slutsky AS, Zamel N. The effects of air pollution on allergic bronchial responsiveness. *Clin Exp Allergy* 1992; **22**: 667-72

Mölter A, Simpson A, Berdel D, Brunekreef B, Custovic A, Cyrys J et al. A multicentre study of air pollution exposure and childhood asthma prevalence: the ESCAPE project. *Eur Respir J* 2015; **45**: 610-24

Morgan WJ, Crain EF, Gruchalla RS, O'Connor GT, Kattan M, Evans R. Results of a home-based environmental intervention among urban children with asthma. *N Engl J Med* 2004; **351**:1068–1080

Morgenstern V, Zutavern A, Cyrys J, Brockow I, Gehring U, Koletzko S, et al. Respiratory health and individual estimated exposure to traffic-related air pollutants in a cohort of young children. *Occup Environ Med* 2007; **64**:8–16

Morgenstern V, Zutavern A, Cyrys J, Brockow I, Koletzko S, Kramer U, et al. Atopic diseases, allergic sensitization, and exposure to traffic-related air pollution in children. *Am J Respir Crit Care Med* 2008; **177**:1331–1337

Morrow NL. The industrial production and use of 1, 3-butadiene. *Environmental Health Perspectives* 1990; **86**:7-8.

Mortimer KM, Tager IB, Dockery DW, Neas LM, Redline S. The effect of ozone on inner-city children with asthma: identification of susceptible subgroups. *Am J Respir Crit Care Med* 2000; **162**:1838-45

Motts JA, Shirley DL, Silbergeld EK et al. Novel biomarkers of mercury-induced autoimmune dysfunction: a cross-sectional study in Amazonian Brazil. *Environ Res* 2014; **132**; 12–18

Mutius, VE, Fritzsch C, Weiland SK, Roll G, Magnussen H. Prevalence of asthma and allergic disorders among children in united Germany: A descriptive comparison. *Br Med J* 1992; **305**:1395–1399

Myrick RH (1996) Air Quality Monitoring in Alberta 1994. Summary Report available at http://www.assembly.ab.ca/lao/library/egovdocs /alen/1994/659 72_94.pdf accessed on 28 September 2010

Nabet C, Ancel PY, Burguet A, Kaminski M. Smoking during pregnancy and preterm birth according to obstetric history: French National Perinatal Surveys. *Paediatr Perinat Epidemiol* 2005; **19**: 88–96

Naidoo B, Warm D, Quigley R, Taylor L. Smoking and Public Health: A review of interventions to increase smoking cessation, reduce smoking initiation and prevent further uptake of smoking, Health Development Agency, London 2004

Nair J, Lakshminrusimha S. Update on PPHN: Mechanisms and treatment. *Semin Perinatol* 2014; **38**: 78–91

Najman JM, Lanyon A, Andersen M, Williams G, Bor W, O'Callaghan M. Socioeconomic status and maternal cigarette smoking before, during and after a pregnancy. *Aust N Zealand J Public Health* 1998; **22**:60-66

Nardini S, Bertoletti R, Rastelli V et al. The influence of personal tobacco smoking on the clinical practice of Italian chest physicians. *Eur Respir J* 1998; **12**:1450–3
National UK birth statistics (2007) available at http://www.statistics. gov.uk/pdfdir /preterm 0507.pdf accessed on 8th October 2015

National Cancer Institute 2001. Risks associated with smoking cigarettes with low machine-measured yields of tar and nicotine. Smoking and Tobacco Control Monograph No. 13. NIH Pub. No. 02-5047 Bethesda, Maryland: National Institutes of Health

NCHS. National Center for Health Statistics, United States. Hyattsville (2007)

Negrisoli J, Nascimento LFC. Atmospheric pollutants and hospital admissions due to pneumonia in children. *Rev Paul Pediatr* 2013; **31**: 501–506

Nelson PR, Kelly SP, Conrad FW. Studies of environmental tobacco smoke generated by different cigarettes. *J Air Waste Manage Assoc* 1998; **48**: 336–44

Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner C, et al. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *Eur Respir J* 2003; **21**:956–963

Nieuwenhuijsen M, K. Katsouyanni, G. Touloumi, L. Jarup, N. Best, D. Wartenberg, V. Gallo, P. Vineis. Special study designs and analyses in environmental epidemiology. Environmental

Epidemiology. Study Methods and Applications, Oxford University Press Inc, New York, United States 2008, pp 171–225

Nitta H, Sato T, Nakai S, Maeda K, Aoki S, Ono M. Respiratory health associated with exposure to automobile exhaust. I. Results of cross-sectional studies in 1979, 1982, and 1983. *Arch Environ Health* 1993; **48**: 53–58

Ning Z, Geller MD, Moore KF, Sheesley R, Schauer JJ, Sioutas C. Daily variation in chemical characteristics of urban ultrafine aerosols and inference of their sources. *Environ Sci Technol* 2007; **41**: 6000–6006

Nishimura KK, Galanter JM, Roth LA, et al. Early life air pollution and asthma risk in minority children: the GALA II & SAGE II studies. *Am J Respir Crit Care Med* 2013; **188**: 309–318

Nitschke M, Pilotto LS, Attewell RG, Smith BJ, Pisaniello D, Martin J et al. A cohort study of indoor nitrogen dioxide and house dust mite exposure in asthmatic children. *J Occup Environ Med* 2006; **48**:462–469

Novotny TE, Zhao F. Consumption and production waste: another externality of tobacco use. *Tob Control* 1999; **8**: 75-80

Oberg M, Jaakola MS., Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: A retrospective analysis of data from 192 countries. *Lancet* 2011; **377**:139–146

Oftedal B, Brunekreef B, Nystad W, Nafstad P. Residential outdoor air pollution and allergen sensitization in school children in Oslo Norway. *Clin Exp Allergy* 2007; **37**:1632–1640

Oftedal B, Nystad W, Brunekreef B, Nafstad P. Long-term traffic-related exposures and asthma onset in schoolchildren in Oslo, Norway. *Environ Health Perspect* 2009; **117**:839–844

Ohashi Y, Nakai Y, Furuya H, Esaki Y, Ikeoka H, Kato S, et al. Mucociliary disease of the middle ear during experimental otitis media with effusion induced by bacterial endotoxin. *Ann Otol Rhinol Laryngol* 1989a; **98**:479–484

Ohashi Y, Nakai Y, Ikeoka H, Koshimo H, Esaki Y. Acute effects f sulfur dioxide exposure on middle ear mucosa. *Ann Otol Rhinol Laryngol* 1989b; **98**:301–307

Oken E, Gillman MW. Fetal origins of obesity. *Obes Res* 2003; **11**: 496-506

Okuno, T. Thermal effect of infra-red radiation on the eye: A study based on a model. *Ann Occup Hyg* 1991; **35**: 1–12

Ong KKL, Ahmed LM, Emmett PM, Preece MA, Dunger DB. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. *BMJ* 2000; **320**:967

O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, et al. Health, wealth, and air pollution: advancing theory and methods. *Environ Health Perspect* 2003; **111**:1861–1870.

Ostro B, Lipsett M, Mann J, Braxton-Owens H, White M. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiol* 2001; **12**:200–208

Ostro B. Fine particulate air pollution and mortality in two Southern California Counties. *Environ Res* 1995; **70**:98-104

Osunsanya T, Prescott G, Seaton A. Acute respiratory effects of particles: mass or number? *Occup Environ Med* 2001; **58**:154-9

Ott WR, Klepeis NE, Switzer P. Analytical solutions to compartmental indoor air quality models with application to environmental tobacco smoke concentrations measured in a house. *J Air Waste Manage Assoc* 2003; **53**: 918-936

Parkes MJ, Lara-Mun˜oz JP, Izzo PN, Spyer KM. Responses of ventral respiratory neurones in the rat to vagus stimulation and the functional division of expiration. *J Physiol* (Lond) 1999; **476**:131–139

Parker J, Woodruff T, Basu R, Schoendorf K. Air pollution and birth weight among term infants in California. *Pediatrics* 2005; **115**:121–128

Patricia S, Floros J. Genetic variant associations of human SP-Aand SP-D with acute and chronic lung injury. *Front Biosci* 2012; **17**:407-429

Pattenden S, Hoek G, Braun-Fahrla¨nder C et al. NO_2 and children's respiratory symptoms in the PATY study. *Occup Environ Med* 2006; **63**: 828–835

Pellizzari ED, Perritt RL, Clayton CA. National Human Exposure Assessment Survey (NHEXAS): exploratory survey of exposure among population subgroups in EPA Region V. *J Expo Anal Environ Epidemiol* 1999; **9**:49–55

Peluso M, Srivatanakul P, Munnia A, Jedpiyawongse A, Meunier A, Sangrajrang S, DNA adduct formation among workers in a Thai industrial estate and nearby residents. *Sci Tot Environ* 2008; **389**: 283-288

Perera F, Hemminki K, Jedrychowski W, Whyatt R, Campbell U, Hsu Y. In utero DNA damage from environmental pollution is associated with somatic gene mutation in newborns. *Cancer Epidemiol Biomarkers Prev* 2002; **11**: 1134–1137

Peters A, Von Klot S, Heier M, et al. Exposure to traffic and the onset of myocardial infarction. *N Engl J Med* 2004; **351**:1721–1730

Peters A, Dockery DW, Heinrich J, Wichmann HE. Medication use modifies the health effects of particulate sulfate air pollution in children with asthma. *Environ Health Perspect* 1997; **105**: 430–435

Peters A, Goldstein IF, Beyer U, Franke K, Heinrich J, Dockery DW, Spengler JD, Wichmann HE. Acute health effects of exposure to high levels of air pollution in Eastern Europe. *Am J Epidemiol* 1996; **144**: 570–581

Platt MJ, Ashton J. Health and deprivation in Mersey region. Liverpool: Observatory Report Series, Department of Public Health, University of Liverpool, 1991; **2**:1–16

Pothikamjorn SL, Ruxrungtham K, Thampanitchawong P, Fuangthong R, Srasuebkul P, Sangahsapaviriyah A, et al. Impact of particulate air pollutants on allergic diseases, allergic skin reactivity and lung function. *Asian Pac J Allergy Immunol* 2002; **20**:77–83

Pope III CA, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 2002; **287**:1132–41

Power CJ. Fetal environment and subsequent obesity a study of maternal smoking. *Int J Epidemiol* 2002; **31**:413-19

Power MC, Weisskopf MG, Alexeeff SE et al. Traffic-related air pollution and cognitive function in a cohort of older men. *Environ Health Perspect* 2011; **119**: 682–687

Public Health England (2014). Estimating local mortality burdens associated with particulate air pollution. Public health England 2014 Ref: ISBN 978-0-85951-753-9, PHE publications gateway number: 2014016 PHE-CRCE-010

Qian ZHL, Chinchilli VM, Lehman EB, Stewart WF, Shah N, Duan Y et al. Associations between air pollution and peak expiratory flow among patients with persistent asthma. *J Toxicol Environ Health* 2009; **72**: 39–46

Radio Free Asia, China: Beijing issues 'red alert' smog warning amid growing public anger, 8 December 2015, available at: http://www. refworld.org/docid /567403fa6.html [accessed 12 March 2016]

Ramadour M, Burrel C, Lanteaume A, Vervloet D, Charpin D, Brisse F, et al. Prevalence of asthma and rhinitis in relation to long-term exposure to gaseous air pollutants. *Allergy* 2000; **55**:1163–1169

Ray S, Kumar N. Weather Conditions and Air Pollution Impact of Odd-even Policy in Delhi. Economic and Political Weekly 2016:51 available athttp://www.epw.in/journal/2016/10/web-exclusives/impact-weather-conditions-airpollution.html ?0=ip_login_no_cache %3D 21fde79d8a80c0d347aeb56f 4 23a 857d#sthash.cHT3HExw.dpuf accessed on 12th March 2016

RCP and RCPCH Air Pollution Report (2016). Every breath we take: the lifelong impact of air pollution Executive summary available at www.rcplondon.ac.uk/projects/outputs/every-breath-we-take-lifelong-impact-airpollution accessed on 26th February 2016

Repace JL 2007 Exposure to secondhand smoke. Chapter 9 in Ott, Steinemann, and Wallace, eds., Exposure Analysis, CRC-Press, Taylor & Francis Group, Boca Rotan, FL

Riedel C, Schönberger K, Yang S, Koshy G, Chen YC, Gopinath B, Ziebarth S, von Kries R. Parental smoking and childhood obesity: higher effect estimates for maternal smoking in pregnancy compared with paternal smoking--a meta-analysis. *Int J Epid* 2014; **43**:1593-606

Rizwan S, Manning JT, Brabin BJ. Maternal smoking during pregnancy and possible effects of in utero testosterone: evidence from the 2D:4D finger length ratio. *Early human development* 2007; **83**:87-90

Rizwan S, Reid J, Kelly Y, Bundred PE, Pearson M, Brabin BJ. Trends in childhood and parental asthma prevalence in Merseyside, 1991-1998. *J Pub health* 2004; **26**:337-42

Roberts JD Jr, Fineman JR, Morin FC, Shaul PW, Rimar S, Schreiber MD, et al. Inhaled nitric oxide and persistent pulmonary hypertension of the newborn. The Inhaled Nitric Oxide Study Group. *N Engl J Med* 1997; **336**:605–10

Roberts G, Hurley C, Bush A, Lack G. Longitudinal study of grass pollen exposure, symptoms, and exhaled nitric oxide in childhood seasonal allergic asthma. *Thorax* 2004; **59**:752–756

Rodrigues LC, Newcombe_PJ, Cunhaw SS, Alcantara-Nevesz MB, Cruz AA et al. Early infection with Trichuris trichiura and allergen skin test reactivity in later childhood. *Clin Exper Allergy* 2008; **38**:1769–77

Roemer W, Hoek G, Brunekreef B. Effect of ambient winter air pollution on respiratory health of children with chronic respiratory symptoms. *Am Rev Respir Dis* 1993; **147**:118–124

Rolstad S, Adler J, Rydén A. Response burden and questionnaire length: is shorter better? A review and meta-analysis. *Value Health* 2011; **14**:1101-8

Romão R, Pereira LA, Saldiva PH, Pinheiro PM, Braga AL, Martins LC. The relationship between low birth weight and exposure to inhalable particulate matter. *Cad Saude Publica* 2013; **29**:1101-8

Romieu I, Tellez-Rojo MM, Lazo M, Manzano-Patino A, Cortez-Lugo M, Julien P, et al. Omega-3 fatty acid prevents heart rate variability reductions associated with particulate matter. *Am J Respir Crit Care Med* 2005; **172**:1534–1540

Rosen L, Zucker D, Hovell M, Brown N, Ram A, Myers V. Feasibility of Measuring tobacco smoke air pollution in homes: Report from a Pilot Study. Tchounwou PB, ed. *Int J Environ Res Publ Health* 2015; 1**2**:15129-15142

Rothman KJ, Greenland S, Lash TL. Modern Epidemiology. Philadelphia, PA: Lippincott Williams and Wilkins; 2008: **97**

Rotko T, Koistinen K, Hanninen O, Jantunen M. Sociodemographic descriptors of personal exposure to fine particles (PM2.5) in EXPOLIS Helsinki. *J Expo Anal Environ Epidemiol* 2000; **10**:385–393

Rotko T, Kousa A, Alm S, Jantunen M. Exposures to nitrogen dioxide in EXPOLIS-Helsinki: microenvironment, behavioral and sociodemographic factors. *J Expo Anal Environ Epidemiol* 2001; **11**:216–223

Rudolf M, Leigh-Hunt N. A review of local practice regarding investigations in children attending obesity clinics and a comparison of the results with other studies. *Child Care Health Develop* 2008; **34**: 55-8

Rutishauser M, Ackermann U, Braun C, Gnehm HP, Wanner U. Correlation of respiratory tract symptoms in young children and NO_2 concentrations of the outside air. *Pneumologie* 1990; **44**: 245-6

Sacks JD, Stanek LW, Luben TJ, Johns DO, Buckley BJ, Brown JS et al. Particulate matter-induced health effects: who is susceptible? *Environ Health Perspect* 2011; **119**:446–454

Salam MT, Lin PC, Avol EL, Gauderman WJ, Gilliland FD. Microsomal epoxide hydrolase, glutathione S-transferase P1, traffic and childhood asthma. *Thorax* 2007b; **62**: 1050–1057

Salam MT, Millstein J, Li YF, Lurmann FW, Margolis HG, Gilliland FD. Birth outcomes and prenatal exposure to ozone, carbon monoxide, and particulate matter: results from the Children's Health Study. *Environ Health Perspect* 2005; **113**: 1638–1644

Salam MT, Gauderman WJ, McConnell R, Lin PC, Gilliland FD. Transforming growth factor-β1 C-509T polymorphism, oxidant stress, and early-onset childhood asthma. *Am J Respir Crit Care Med* 2007a; **176**:1192–1199

Salathe M, Lipson EJ, Ivonnet PI, Bookman RJ. Muscarinic signaling in ciliated tracheal epithelial cells: dual effects on Ca2+ and ciliary beating. *Am J Physiol* 1997; **272**: L301-10

Samet JM, Dominici F, Curriero FC, Cousac J, Zeger SL. Fine particulate air pollution and mortality in 20 US cities 1987 - 1994. *N Engl J Med* 2000; **343**:1242-9

Sarnat JA, Holguin F. Asthma and air quality. *Curr Opin Pulm Med* 2007; **13**: 63–66

Schwartz J. Air pollution and children's health. *Pediatrics* 2004; **113**:1037–1043

Schifano P, Lallo A, Asta F, De Sario M, Davoli M, Michelozzi P. Effect of ambient temperature and air pollutants on the risk of preterm birth, Rome 2001-2010. *Environ Int* 2013; **61**:77-87

Schikowski T, Adam M, Marcon A, Cai Y, Vierkötter A, Carsin AE et al. Association of ambient air pollution with the prevalence and incidence of COPD. *Eur Respir J* 2014; **44**:614-26

Schildcrout JS, Sheppard L, Lumley T, Slaughter JC, Koenig JQ, Shapiro GG. Ambient air pollution and asthma exacerbations in children: an eight-city analysis. *Am J Epidemiol* 2006; **164**: 505–517

Schumaker, Randall E. A beginner's guide to structural equation modelling 2nd edition 2004

Seaton A, Dennekamp M. Hypothesis: ill health associated with low concentrations of nitrogen dioxide—an effect of ultrafine particles? *Thorax* 2003; **58**:1012–1015

Seaton A, Godden DJ, Brown K. Increase in asthma: A more toxic environment, or a more susceptible population? *Thorax* 1994; **49**:171–174

Segala C, Fauroux B, Just J, Pascual L, Grimfeld A, Neukirch F. Short-term effect of winter air pollution on respiratory health of asthmatic children in Paris. *Eur Respir J* 1998; **11**:677–685

Semple S, Apsley A, Galea KS, MacCalman L, Friel B, Snelgrove V. Secondhand smoke in cars: Assessing children's potential exposure during typical journey conditions. *Tob Control* 2012; 21**:578**-583

Sexton K, Gong H, Jr, Bailar JC, III, Ford JG, Gold DR, Lambert WE, et al. Air pollution health risks: do class and race matter? *Toxicol Ind Health* 1993; **9**:843–878

Shah ASV, Langrish JP, Nair H, McAllister DA, Hunter LA. Global association of air pollution and heart failure: a systematic review and meta-analysis. *The Lancet* 2013; **382**: 1039 -1048

Sharp GC, Lawlor DA, Richmond RC, Fraser A, Simpkin A, Suderman M. Maternal pre-pregnancy BMI and gestational weight gain, offspring DNA methylation and later offspring adiposity: findings from the Avon Longitudinal Study of Parents and Children. *Int J Epidemiol* 2015; **44**: 1288-304

Shima M, Adachi M. Effect of outdoor and indoor nitrogen dioxide on respiratory symptoms in school children. *Int J Epidemiol* 2000; **29**: 862–870

Simoni M, Jaakkola MS, Carrozzi L, Baldacci S, Di Pede F, Viegi G. Indoor air pollution and respiratory health in the elderly. *Eur Respir J* 2003; **40**: 15–20

Simpson A, Custovic A, Pipis S, Adisesh A, Faragher B, Woodcock A. Exhaled nitric oxide, sensitization, and exposure to allergens in patients with asthma who are not taking inhaled steroids. *Am J Respir Crit Care Med* 1999; **160**:45–49

Simeoni U, Zetterström R. Long-term circulatory and renal consequences of intrauterine growth restriction. *Acta Pædiatrica* 2007; **94**: 819-24

Simoes MJ. Study of frequency of smoking during pregnancy, Ribeirao Preto. *Sao Paulo Rev Clinc Biomed* 1985; **6**: 61-9.

Skene KJ, Gent JF, McKay LA, Belanger K, Leaderer BP, Holford TR. Modeling effects of traffic and landscape characteristics on ambient nitrogen dioxide levels in Connecticut. *Atmos Environ* 2010; **44**: 5156–5164

Smith KR. Fuel combustion, air pollution exposure, and health: The situation in developing countries. *Annu Rev Energy Envir* 1993; **18**: 529–566

Smith GD, Hart C, Blane D, Hole D. Adverse socio economic conditions in childhood and cause specific adult mortality: prospective observational study. *BMJ* 1998; **316**:1631-35

Smith BJ, Nitschke M, Pilotto LS, Ruffin RE, Pisaniello DL, Willson KJ. Health effects of daily indoor nitrogen dioxide exposure in people with asthma. *Eur Respir J* 2000; **16**: 879–885

Sobral HR Air pollution and respiratory diseases in children in Sao Paulo Brazil. *Soc Sci Med* 1989; **29**: 959-964

Sorensen M, Autrup H, Hertel O, Wallin H, Knudsen LE, Loft S. Personal exposure to PM2.5 and biomarkers of DNA damage. *Cancer Epidemiol Biomarkers Prev* 2003 **12**, 191–196

Soriano JB, Davis KJ, Coleman B, Visick G, Mannino D, Pride NB. The proportional Venn diagram of obstructive lung disease: two approximations from the United States and the United Kingdom. *Chest* 2003; **124**:474–81.

Spanier AJ, Kahn RS, Hornung RW, Wang N, Sun G, Lierl MB. Environmental Exposures, Nitric Oxide Synthase Genes, and Exhaled Nitric Oxide in Asthmatic Children. *Pediatr Pulmonol* 2009; **44**: 812–819

Stebbings JH, Jr, Fogleman DG. Identifying a susceptible subgroup: effects of the Pittsburgh air pollution episode upon schoolchildren. *Am J Epidemiol* 1979; **110**:27–40

Steiniger S, Hunter AJS. Geospatial free and open source software in the 21st century: *Proceedings of the first open source geospatial research symposium* 2012; **23**: 247–261

Stewart WF, Schwartz BS, Davatzikos C, Shen D, Liu D et al. Past adult lead exposure is linked to neuro-degeneration measured by brain MRI. *Neurology* 2006; **66**: 1476–1484

Stewart HC, Stevenson TN, Bruce JS, Greenberg B, Chamberlain LJ. Attitudes toward smoking cessation among sheltered homeless parents. *J Comm Health* 2015; **23**:1-9

Sughis M, Nawrot TS, Haque SI, Amjad A, Nemery B Blood pressure and particulate air pollution in schoolchildren of Lahore, Pakistan. *BMC Public Health* 2012; **12**: 378

Sunyer J, Spix C, Quénel P, Ponce-de-Deón A, Pönka A, Barumandzadeh T, et al. Urban air pollution and emergency admissions for asthma in four European cities: the APHEA Project. *Thorax* 1997; **52**:760–765

Sunyer J, Puig C, Torrent M, Garcia-Algar O, Calico I, Munoz-Ortiz L, et al. Nitrogen dioxide is not associated with respiratory infection during the first year of life. *Int J Epidemiol* 2004; **33**:116–120

Tam AYC, Wong CM, Lam TH, et al. Bronchial responsiveness in children exposed to atmospheric pollution in Hong Kong. *Chest* 1994; **106**:1056–60

Taylor-Robinson D, Agarwal U, Diggle PJ, Platt MJ, Yoxall B, et al. Quantifying the Impact of Deprivation on Preterm Births: A retrospective cohort study. *PLoS One* 2011; **6**: e23163

Tecer LH, Alagha O, Karaca F, Tuncel G, Eldes N. Particulate matter (PM2.5, PM_{10}–2.5, and PM_{10}) and children's hospital admissions for asthma and respiratory diseases: a bidirectional case-crossover study. *J Toxicol Environ Health* 2008; **71**: 512–520

Thayer KA, Heindel JJ, Bucher JR, Gallo MA. Role of environmental chemicals in diabetes and obesity: a National Toxicology Program workshop review. *Environ Health Perspect* 2012; **120**:779-89

The Air Quality Strategy for England, Scotland, Wales and Northern Ireland. DEFRA (2007).

The International Study of Asthma and Allergies in Childhood (ISAAC). Worldwide variation in prevalence of symptoms of asthma, allergic rhino-conjunctivitis, and atopic eczema: ISAAC. The International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. *Lancet* 1998; **351**: 1225–1232

Thurston GD, Bates DV. Air pollution as an underappreciated cause of asthma symptoms. *J Am Med Assoc* 2003; **290**:1915–1917

Toledo-Corral CM, Alderete TL, Habre R et al. Effects of air pollution exposure on glucose metabolism in Los Angeles minority children. *Pediatr Obes* 2016 doi: 10.1111/ijpo.12188.

Toschke AM, Koletzko O, Slikker Jr, Hermann M, Von kries R. Childhood obesity in association with maternal smoking in pregnancy. *Eur J Paed* 2003; **161**: 445-448

Townsend P, Philipmore P, Beattie A. Health and deprivation: Inequality and the North London: Room Helm, 1988

Tunnicliffe WS, Burge PS, Ayres JG. Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. *Lancet* 1994; **344**: 1733–1736

Turpin BJ, Weisel CP, Morandi M, Colome S, Stock T, Eisenreich S, Buckley B. Relationships of Indoor, Outdoor, and Personal Air (RIOPA): part II. Analyses of concentrations of particulate matter species. *Res Rep Health Eff Inst* 2007; **130**: 1–77

Tzoulaki I, Jarvelin MR, Hartikainen AL, Leinonen M, Pouta A, Paldanius M. Size at birth, weight gain over the life course, and low-grade inflammation in young adulthood: northern Finland 1966 Birth Cohort study. *Eur Heart J* 2008; **8**:1049-56

Wilson I, Semple S, Mills LM, Ritchie D, Shaw A, O'Donnell R et al. REFRESH—Reducing families' exposure to secondhand smoke in the home: A feasibility study. *Tob Control* 2013; **22** doi: 10.1136/tobaccocontrol-2011-050212

World Bank and Institute for Health Metrics and Evaluation. 2016. The Cost of Air Pollution: Strengthening the Economic Case for Action. World Bank, Washington, DC. © World Bank. https://openknowledge.worldbank.org/handle/10986/25013 License: CC BY 3.0 IGO

U.S. Department of Health and Human Services. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2014

Valente P, Forastiere F, Bacosi A, Cattani G, di Carlo S, Ferri M et al. Exposure to fine and ultrafine particles from secondhand smoke in public places before and after the smoking ban, Italy 2005 *Tob Control* 2007;**16**:312–317

Van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res* 1997; **74**:122–132

Vedal S, Schenker MB, Munoz A, Samet JM, Batterman S, Speizer FE. Daily air pollution effects on children's respiratory symptoms and peak expiratory flow. *Am J Public Health* 1987; **77**: 694-8

Venn AJ, Lewis SA, Cooper M, Hubbard R, Britton J. Living near a main road and the risk of wheezing illness in children. *Am J Respir Crit Care Med* 2001; **164**: 2177–2180

Venn J. On the diagrammatic and mechanical representation of propositions and reasonings. *The London, Edinburgh, and Dublin Philosophical Magazine and Journal of Science* 1880; **1**:18

Verleden GM, Dupont LJ, Verpeut AC, Demedts MG. The effect of cigarette smoking on exhaled nitric oxide in mild steroid-naive asthmatics. *Chest* 1999; **116**:59–64

Viegi G, Simoni M, Scognamiglio A, et al. Indoor air pollution and airway disease. *Int J Tuberc Lung Dis* 2004; **8**:1401–1415

Villeneuve PJ, Chen L, Rowe BH, Coates F. Outdoor air pollution and emergency department visits for asthma among children and adults: a case-crossover study in northern Alberta, Canada. *Environ Health* 2007; **6**:40

Viloria T, Rubio MC, Rodrigo L, Calderon G, Mercader A, Mateu E et al. Smoking habits of parents and male : female ratio in spermatozoa and pre-implantation embryos. *Hum Reprod* 2005; **20**: 2517-22

Vineis P, Pursiainen KH. Air pollution and cancer: biomarker studies in human populations. *Carcinogenesis* 2005; **26**: 1846-1855

Vladimirov D, Niemela S, Auvinen J et al. Changes in alcohol use in relation to sociodemographic factors in early midlife. *Scandinavian Journal of Public health* 2015; **44**, 249-257

Von Klot S, Wolke G, Tuch T, Heinrich J, Dockery DW, Schwartz J et al. Increased asthma medication use in association with ambient fine and ultrafine particles. *Eur Respir J* 2002; **20**: 691–702

Von Kries R, André MT, Berthold K, William S. Maternal smoking during pregnancy and childhood obesity. *Am J Epidemiol* 2002; **156**: 954–61

Von Mutius E, Weiland SK, Fritzsch C, Duhme H, Keil U. Increasing prevalence of hay fever and atopy among children in Leipzig, East Germany. *Lancet* 1998; **351**: 862–866

Vondra V, Branis M, Reisova M, Maly M, Hoek G. Air pollution and respiratory health of children: the PEACE panel study in Prague, Czech Republic. *Eur Respir Rev* 1998; **8**: 78–85

Wallace L. Indoor particle: a review. *J Air Waste Manag Assoc* 1996; **46**:98–126

Walton H, Dajnak D, Beevers S, Williams M, Watkiss P, Hunt A. Understanding the Health Impacts of Air Pollution in London for Transport for London and the Greater London Authority; *Environmental Research Group*, School of Biomedical Sciences, King's College London, 2015;**33**-34

Wang S, Zhang J, Zeng X, et al. Association of traffic-related air pollution with children's neurobehavioral functions in Quanzhou, China. *Environ Health Perspect* 2009; **117**:1612–1618

Wang X, Zuckermann B, Pearson C et al. Maternal cigarette smoking, metabolic gene polymorphism and infancy birth weight. *JAMA* 2002; **287**: 195-7

Ward DJ, Ayres JG. Particulate air pollution and panel studies in children: a systematic review. *Occup Environ Med* 2004; **61**: e13

Ware JH, Ferris BG, Dockery DW, Sprengler JD, Stram DO Speizer FE. Effects of ambient sulphur oxide and suspended particles on respiratory health of pre- adolescent children. *Am Rev Resp Dis* 1986; **133**: 834-842

Weidman JR, Dolinoy DC, Murphy SK, Jirtle RL. Cancer susceptibility: epigenetic manifestation of environmental exposures. *Cancer J* 2007; **13**: 9–16

Weiland SK, Mundt KA, Ruckmann A, Keil U. Self-reported wheezing and allergic rhinitis in children and traffic density on street of residence. *Ann Epidemiol* 1994; **4**:243–247

Weinmayr G, Romeo E, De Sario M, Weiland SK, Forastiere F. Short-term effects of PM_{10} and NO_2 on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. *Environ Health Perspect* 2010; **118**:449–457

Weitzman, M et al. Tobacco smoke exposure is associated with the metabolic syndrome in adolescents. *Circulation* 2005; **112**: 862–869

West SK, Bates MN, Lee JS, Schaumberg DA, Lee DJ, Adair-Rohani H et al. Is Household Air Pollution a Risk Factor for Eye Disease? *Int J Environ Res Public Health* 2013; **10**: 5378-5398

Wilhelm M, Ritz B. Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, California, USA. *Environ Health Perspect* 2005; **113**:1212–1221

Wirral Economic Profile (2014) available at http://info. wirral.nhs.uk/document_ uploads/Wirral%20Economic%20Profiles/Wirral_Economic_Profile_January_2014.pdf accessed on 19th May 2014

Wolkoff, P. Indoor air pollutants in office environments: Assessment of comfort, health, and performance. *Int J Hyg Environ Health* 2013; **216**: 371–394

Wong CM, Ma S, Hedley AJ, Lam TH. Effect of air pollution on daily mortality in Hong Kong. *Environ Health Perspect* 2001; **109**:335–340

World Bank and Institute for Health Metrics and Evaluation. 2016. The Cost of Air Pollution: Strengthening the Economic Case for Action. World Bank, Washington, DC. © World Bank. https://openknowledge.worldbank.org/handle/10986/25013 License: CC BY 3.0 IGO

WHO (1987). Air quality guidelines for Europe. Copenhagen, World Health Organization Regional Office for Europe, 1987 (WHO Regional Publications, European Series No. 23).
WHO (2000). Air quality guidelines for Europe, 2nd ed. Copenhagen, World Health Organization Regional Office for Europe, 2000 (WHO Regional Publications, European Series No. 91).

WHO. Children's health and the environment. World Health Organization, Geneva; 2003.

WHO (2014). Air pollution news release available at http://www.who.int/mediacentre/news /releases/2014/air-pollution/en/ accessed on 6th March 2016

World Health Organization (WHO) and European Commission Joint Research Centre (EC RJC) Guidelines for Concentration and Exposure-Response Measurement of Fine and Ultra Fine Particulate Matter for Use in Epidemiological Studies (Schwela D, Morawska L, Kotzias D, eds) (2002) available at http:// whqlibdoc.who .int/hq/2002/a76621.pdf accessed on 12th October 2015

World Health Organization. WHO methods and data sources for global causes of death 2000–2012. Global Health Estimates Technical Paper WHO/HIS/HSI/GHE/2014.7. 2014. available at http:// www.who.int/healthinfo/global_burden_disease /GlobalCOD_ method _2000 _2012.pdf accessed on 10-09-2016

World Health Organization. Burden of disease from the joint effects of Household and Ambient Air Pollution for 2012. WHO Technical Report. 2012 available: http://www.who.int/phe/health_topics/outdoorair/databases/AP_jointeffect_BoD_results_March2014.pdf accessed on 10-09-2016

Wyler C, Braun-Fahrlander C, Kunzli N, Schindler C, Ackermann-Liebrich U, Perruchoud AP et al. Exposure to motor vehicle traffic and allergic sensitization. The Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. *Epidemiology* 2000; **11**:450–456

Xie B, Palmer PH, Pang Z, Sun P, Duan H, Johnson CA et al. Environmental tobacco use and indicators of metabolic syndrome in Chinese adults. *Nicot Tob Res 2010*; **12**:198–206

Yamazaki S, Nitta H, Ono M, Green J, Fukuhara S. Intracerebral haemorrhage associated with hourly concentration of ambient particulate matter: Case-crossover analysis. *Occup Environ Med* 2007; **64**:17-24

Yang G, Wang Y, Zeng Y, Gao GF, Liang X, et al. Rapid health transition in China, 1990–2010: findings from the Global Burden of Disease Study 2010. *Lancet* 2013; **381**: 1987–2015

Yates DH, Breen H, Thomas PS. Passive smoke inhalation decreases exhaled nitric oxide in normal subjects. *Am J Respir Crit Care Med* 2001; **164**:1043–1046

Yu JH, Lue KH, Lu KH, Sun HL, Lin YH, Chou MC. The relationship of air pollution to the prevalence of allergic diseases in Taichung and Chu-Shan in 2002. *J Microbiol Immunol Infect* 2005; **38**:123–126

Zanobetti A, Schwartz J. Particulate air pollution, progression, and survival after myocardial infarction. *Environ Health Perspect* 2007; **115**:769–775

Zeka A, Zanobetti A, Schwartz J. Individual-level modifiers of the effects of particulate matter on daily mortality. *Am J Epidemiol* 2006; **163**:849–859
Zemek R, Mieczysław Szyszkowicz, Brian H. Rowe. Air Pollution and Emergency Department Visits for Otitis Media: A case-crossover study in Edmonton, Canada. *Environ Health Perspect* 2010; **118**: 1631–1636

Zipfel PF, Lauer N, Skerka C. The role of complement in AMD. *Adv Exp Med Biol* 2010; **703**: 9–24

Zou B, Peng F, Wan N, Mamady K, Wilson GJ. Spatial cluster detection of air pollution exposure inequities across the United States. *PLoS One* 2014; **19**:e91917

APPENDICES

Appendix A

Search Strategy

Search strategy

Search terms used (for air pollution and birth and child health outcomes)

1 air

2 pollution

3 pollutant

4 2 or 3

5 1 and 4

6 birth

7 child

8 childhood

9 children

10 7 or 8 or 9

11 6 or 10

12 health

13 problem

14 problems

15 13 or 14

16 outcome

17 outcomes

18 16 or 17

19 15 or 18

20 5 and 11 and 12 and 19

(((((air) AND ((pollution) OR pollutant))) AND ((birth) OR (((child) OR children) OR childhood))) AND health) AND ((((problem) OR problems)) OR ((outcome) OR outcomes)) Filters: publishedfrom 1990 onwards and in humans

Appendix B

List of relevant questions extracted from the original questionnaire (2006 Community Child Health Survey) used in MPhil project

1 Child's weight in kgs…….

2 Child's height in cms…….

3 School……..

4 Postcode……

5 Child age…….years……..months

6 Sex……..Male…….Female

7 Has your child ever been diagnosed by doctor as having asthma or bronchial asthma? Yes… No…

8 If you child has asthma do you consider it well controlled? Yes… No…

9 Has your child ever been diagnosed by doctor as having bronchitis? Yes… No…

10 Has your child ever been diagnosed by doctor as having Croup? Yes… No…

11 Does your child have any allergies? Yes… No…

12 If yes for 11, does your child have pet allergy? Yes… No…

13 If yes for 11, does your child have hay fever? Yes… No…

14 If yes for 11, does your child have food allergy? Yes… No…

15 If yes for 11, does your child have eczema? Yes… No…

16 Does any person smoke in the household? Yes… No…

17 Did the mother of this child smoke during pregnancy? Yes… No…

18 Did the father of this child smoke during mother's pregnancy? Yes… No…

19 Did the maternal grandmother of this child smoke during the mother's pregnancy? Yes… No…

20 Did the maternal grandfather of this child smoke during the mother's pregnancy? Yes… No…

21 Are you presently in paid employment? For mother……Yes… No…

22 Are you presently in paid employment? For father……. Yes… No…

23 Are you a single parent family? Yes… No…

24 What was the birthweight of your child in kgs……. or in lbs…….

25 Was your child born prematurely? Yes… No…

26 Was the baby breastfed? Yes… No…

27 Does the child's mother or father suffer from asthma? Yes…No…

28 Does your house have damp patches on the wall? Yes…No…

29 Do you feel your child's health has affected his or her school attendance? Yes…No…

30 Does your child have Attention Deficit Hyperactivity Disorder (ADHD) which has been diagnosed by a doctor? Yes…No…

31 Has your child ever had wheezing (by wheezing I mean noisy breathing with a whistling sound coming from the chest not the throat)? Yes…No…

32 Has your child been breathless at any during the last 12 months? Yes…No…

33 Has your child ever seemed to cough more (or to get more coughs compared to other children? Yes…No…

Appendix C

Information document and consent form

LIVERPOOL
SCHOOL OF
TROPICAL
MEDICINE (Affiliated to the University of Liverpool)

Pembroke Place
Liverpool L3 5QA
Telephone: 0151·708 9393
Fax: 0151·705 3370
http://www.liv.ac.uk/lstm/lstm.html

7th September 2006

Merseyside Surveys of Childhood Asthma and Obesity

Parents/Guardians Information Sheet

We are conducting a survey to determine the current respiratory health status of children in primary schools in Merseyside. This survey will help us to identify what factors predispose to asthmatic symptoms in children. We are interested in finding out about changes in the respiratory health of children who have been living in Merseyside over the past several years. The survey is designed also to find out about parental smoking patterns and their possible link to the increasing the risk of childhood overweight and obesity. Previously we have conducted a very similar survey of children at your child's schools in 1991, 1993 and 1998. The present study follows the previous studies and has a similar questionnaire. The information provided in the previous surveys showed that almost 30 % of children of your child's age had doctor diagnosed asthma and that the number of affected children had risen substantially between 1991 and 1998. This survey will help us to understand how and possibly why the number of asthmatic children has changed since the previous surveys.

To do this we need to ask parents/guardians to complete the enclosed questionnaire. Completing of the questionnaire would take 20-30 minutes. Also we wish to measure your child's height and weight at the school with the assistance of the class teacher.

The information on the questionnaire will remain confidential at all time. You do not have to take part in this survey, if you do not want to and can withdraw without giving any reason and this will not influence your child's situation. If you wish to complete the questionnaire and allow the measurement on your child's height and weight at the school, then please indicate this on the attached consent form. If you do not wish to take part, please return the questionnaire and other sheets unanswered. If you have any queries pleased do not hesitate to contact Professor Bernard Brabin (or Dr Ali Delpisheh) at the telephone number above.

Thank you for your co-operation

Bernard J. Brabin

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7th September 2006

Merseyside Surveys of Childhood Asthma and Obesity

Consent Form

I give/do not give permission for my child …… class……to be included in the survey. I have read the information sheet and know that the survey includes a questionnaire which I have/have not completed. I agree/do not agree to the measurement of my child's weight and height which will take place at the school with assistance of the class teacher.

Print name of parent/guardian…….

Signature of parent/guardian…….. Date:……

Please insert this form into the questionnaire and replace in the envelope provided which your child should return it to his/her class teacher.

7th September 2006

Merseyside Surveys of Childhood Asthma and Obesity

LIVERPOOL
SCHOOL OF
TROPICAL
MEDICINE (Affiliated to the University of Liverpool)

Pembroke Place
Liverpool L3 5QA
Telephone: 0151-708 9393
Fax: 0151-705 3370
http://www.liv.ac.uk/lstm/lstm.html

Letter of invitation

Dear Parent/Guardian

We would like to invite you and your child to take part in the Merseyside surveys of childhood asthma and obesity. This survey is part of a series surveys done in your child's primary schools between 1991, 1995 and 1998. We are repeating the survey in 2006 in order to assess community trends in asthma and how these relate to nutrition and exposure to cigarette smoke. Attached is an information sheet which gives more details. Also enclosed are the consent form and the study questionnaire.

We will be grateful if you would return the questionnaire and consent form in the envelope provided to your child's class teacher.

Yours truly,

Bernard J. Brabin

Professor of Tropical Child Health

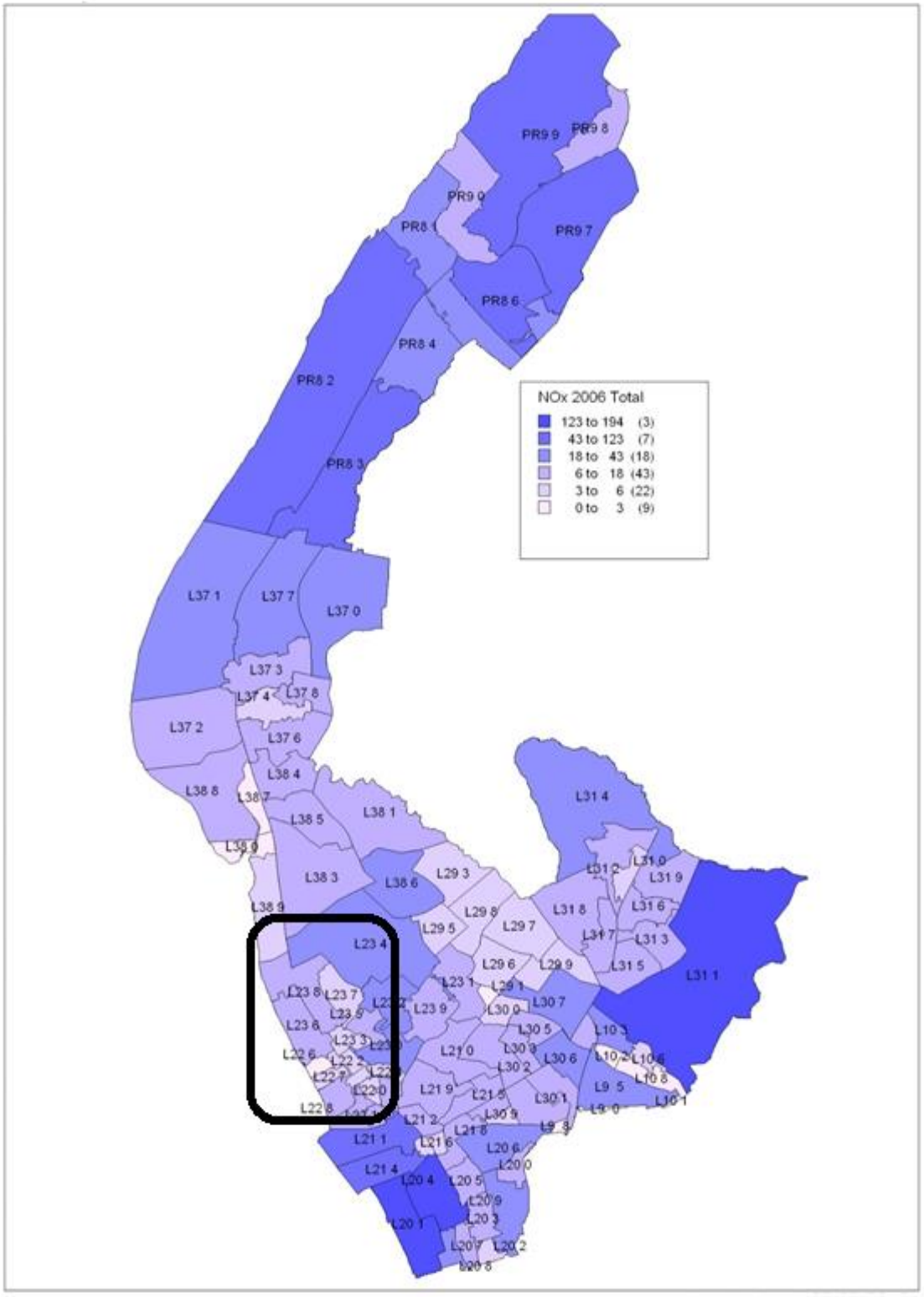
Head Teacher

Appendix D

Sefton postcode sector based

NO emissions (2006)

Figure A NOx emissions map



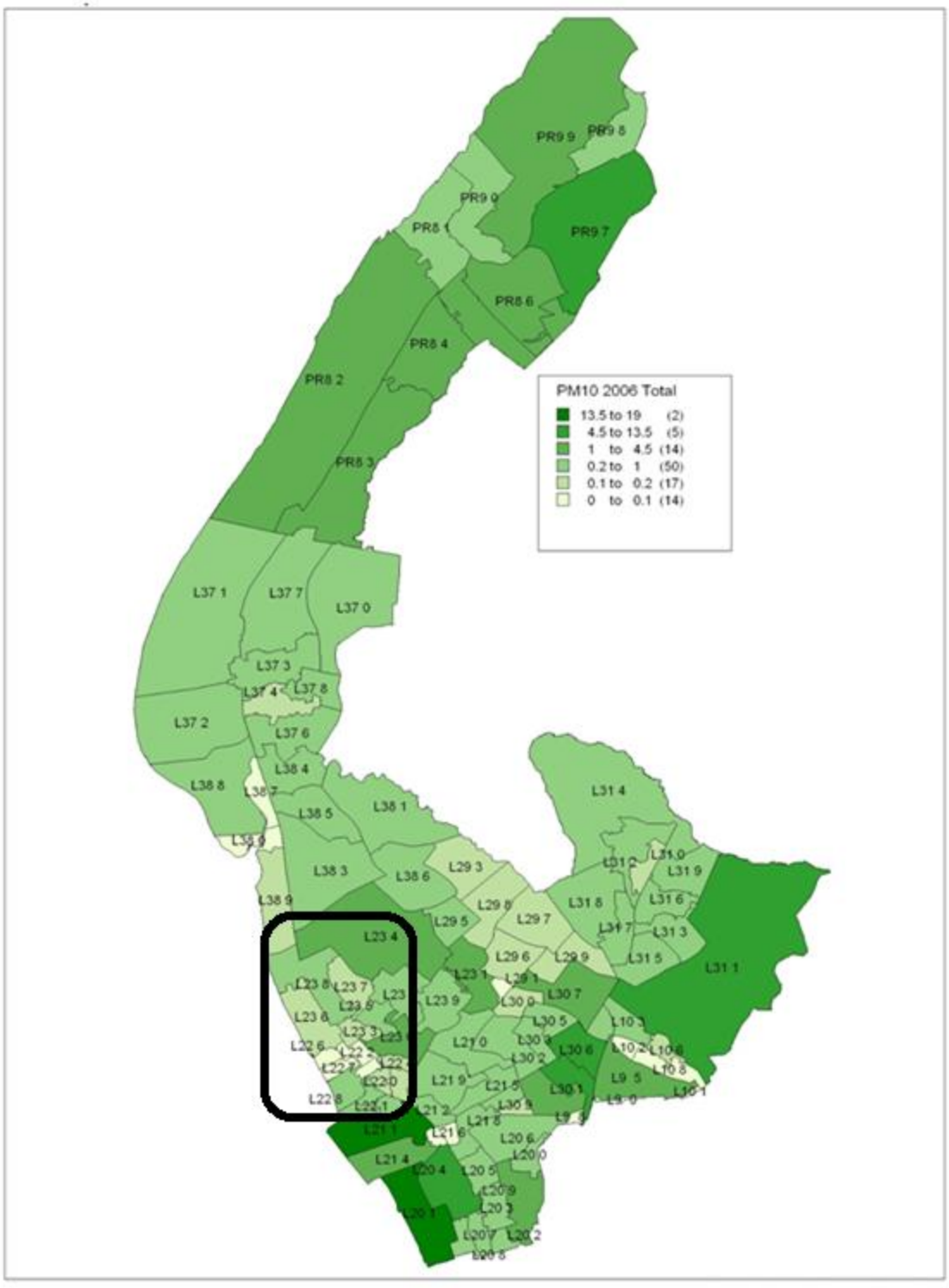
Source: Merseyside Atmospheric Emissions Inventory 2006

Appendix E

Sefton postcode sector based

PM_{10} emissions (2006)

Figure B PM_{10} emissions map



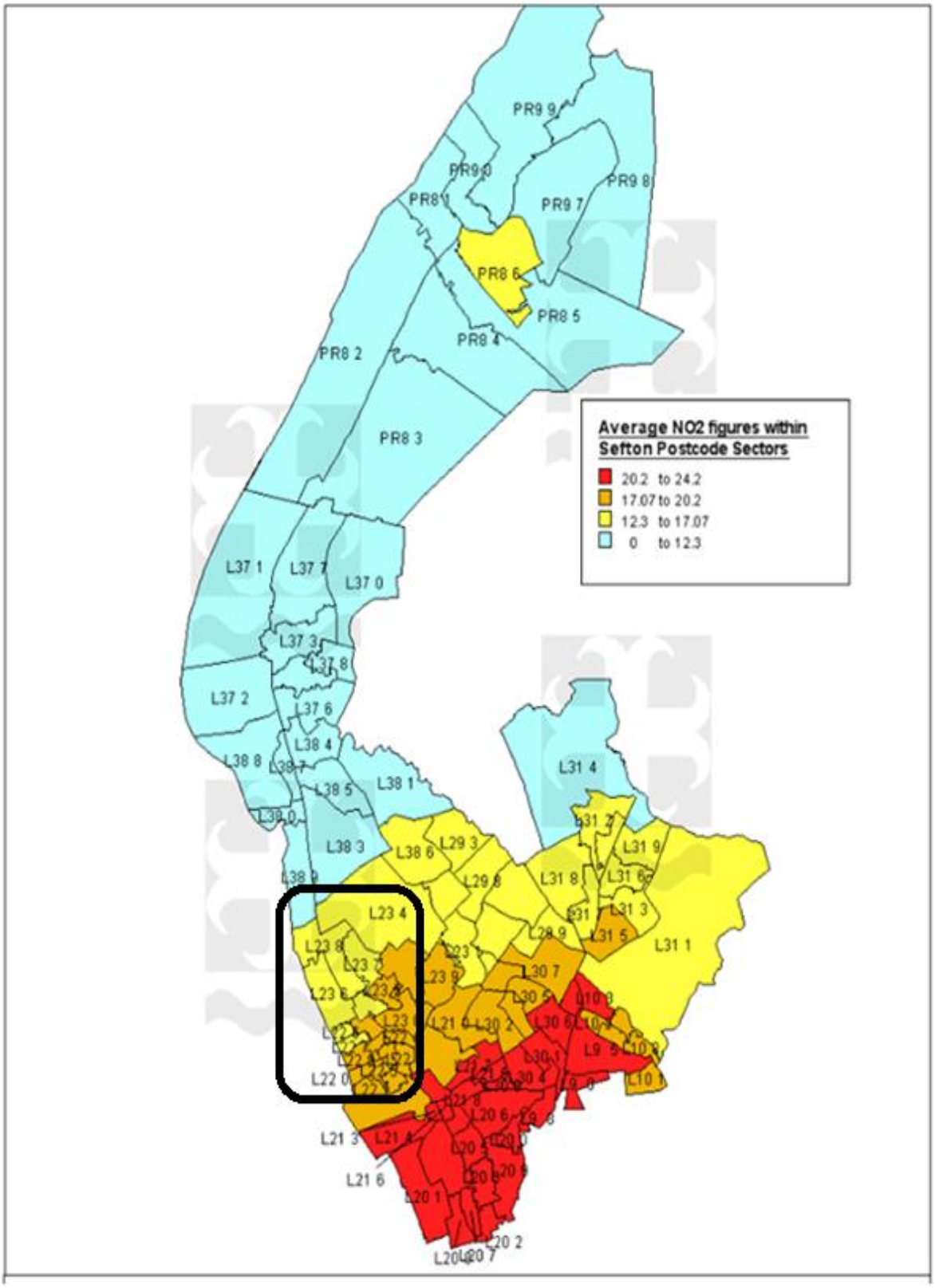
Source: Merseyside Atmospheric Emissions Inventory 2006

Appendix F

Sefton postcode sector based

NO_2 concentrations (2006)

Figure C NO_2 concentrations map



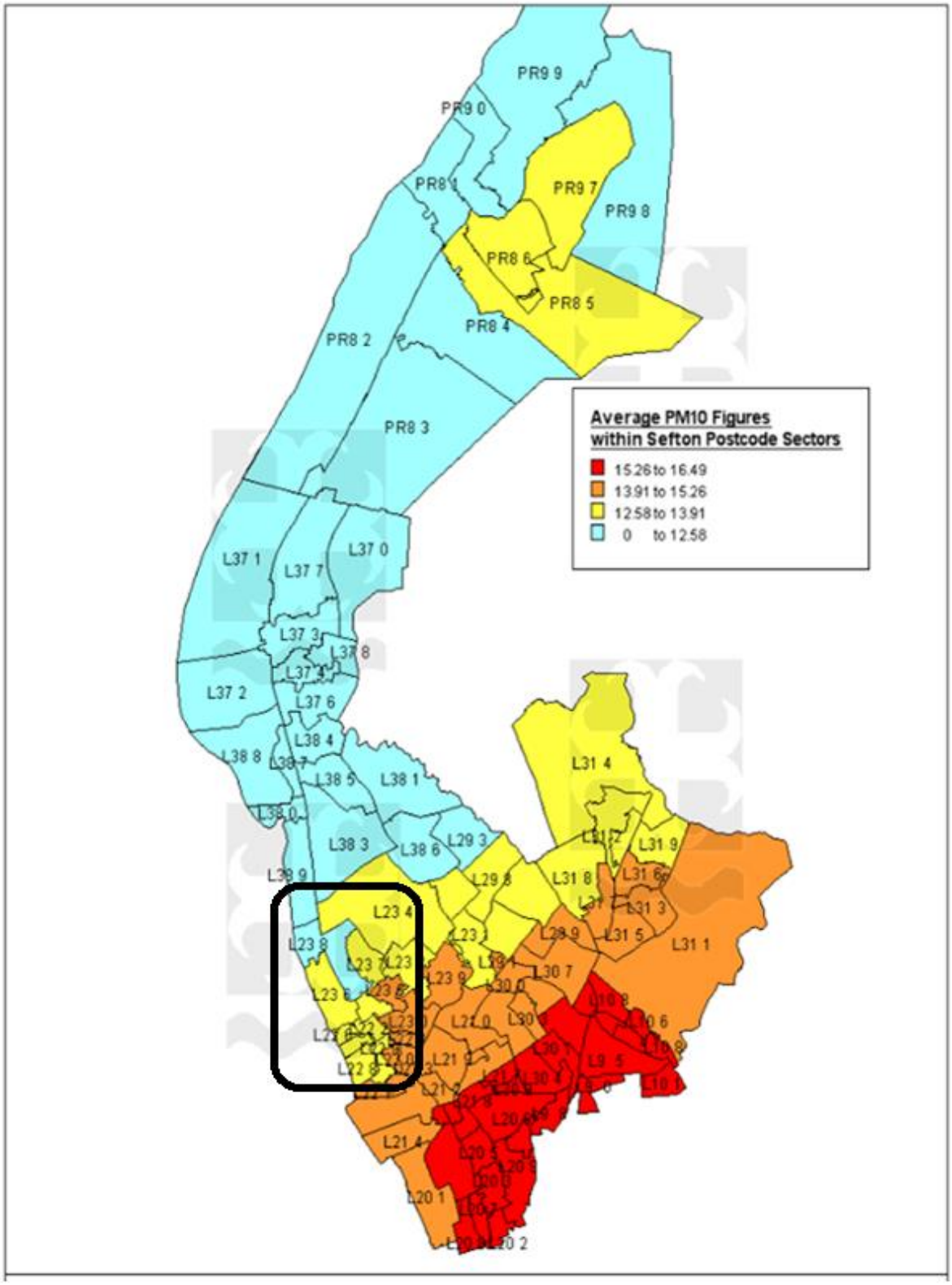
Source: Merseyside Atmospheric Emissions Inventory 2006

Appendix G

Sefton postcode sector based

PM_{10} concentrations (2006)

Figure D PM_{10} concentrations map



Source: Merseyside Atmospheric Emissions Inventory 2006

Appendix H

Sefton postcode sectors and emissions values (2006)

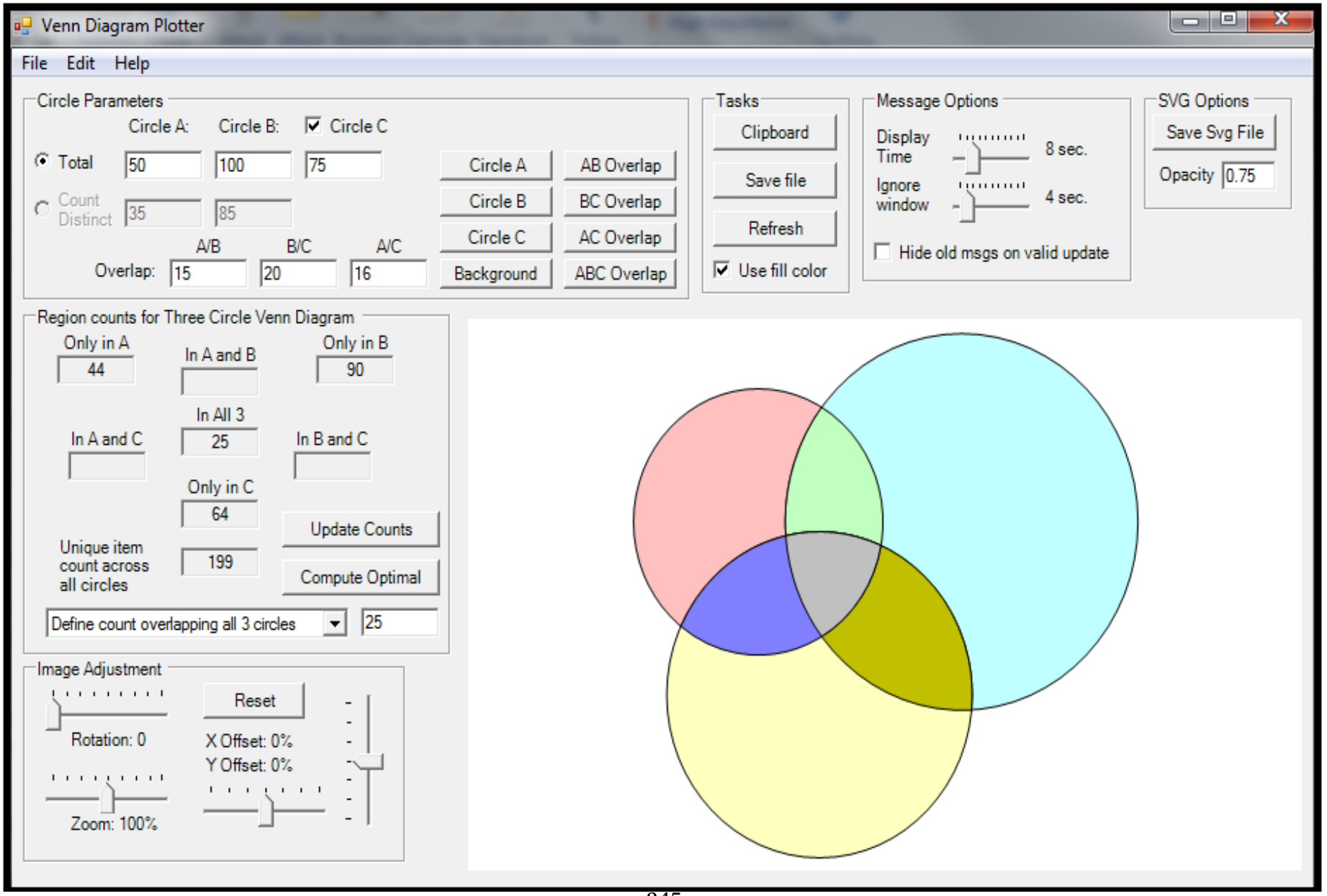
Postcode	Post code sector	NOxTotE	NOxAvgE	PM10TotE	PM10AvgE	NO2AvgConc	PM10AvgConc
L10 1LB	L10 1	0.319226	0.417753	0.00937787	0.0122723	19.69	15.5864
L10 2LD	L10 2	1.553420	0.313793	0.05179970	0.0104637	20.14	15.8499
L10 6LZ	L10 6	3.056850	0.458000	0.11860100	0.0177697	19.42	15.5262
L10 8LL	L10 8	2.224240	0.167225	0.06609750	0.0049694	19.71	15.7389
L20 0EE	L20 0	7.280530	0.946225	0.27115000	0.0352405	22.77	16.0321
L20 1BZ	L20 1	193.710000	4.590710	13.66690000	0.3238940	21.10	14.9477
L20 2EX	L20 2	5.939120	1.130380	0.20427400	0.0388791	23.26	16.0778
L20 3QN	L20 3	14.577800	1.022620	0.54469000	0.0382094	22.92	16.2343
L20 4SP	L20 4	129.898000	3.812940	6.35245000	0.1864680	22.42	15.6449
L20 5ES	L20 5	12.503200	0.941291	0.67283800	0.0506540	22.85	16.0207
L20 6HX	L20 6	22.422800	0.671802	0.96691500	0.0289695	22.16	15.8652
L20 7BX	L20 7	10.411500	1.228650	0.33867400	0.0399665	23.71	16.1727
L20 8HE	L20 8	23.460400	2.916680	0.87956000	0.1093500	24.10	15.4782
L20 9NU	L20 9	30.883900	1.076970	1.14412000	0.0398974	23.92	16.4828
L21 0JW	L21 0	14.034400	0.457649	0.51412900	0.0167653	18.76	14.7077
L21 2PB	L21 2	7.171480	0.767830	0.24320700	0.0260395	20.25	15.0771
L21 3TE	L21 3	5.961040	1.793500	0.09265290	0.0278771	21.91	15.3245
L21 3TN	L21 1	64.606400	1.838230	18.90430000	0.5378640	20.13	14.3973
L21 4LU	L21 4	61.636300	2.393390	2.16003000	0.0838757	20.79	14.4818
L21 5JB	L21 5	10.070700	1.386690	0.34754900	0.0478560	21.58	15.6415
L21 6NU	L21 6	3.423380	0.880667	0.09946610	0.0255878	21.34	15.2721
L21 7LN	L21 7	17.757400	1.105060	0.58655800	0.0365021	20.72	15.2406
L21 8HX	L21 8	13.237200	0.938281	0.43648900	0.0309392	21.65	15.4539
L21 9HP	L21 9	14.649100	0.476602	0.48495300	0.0157777	19.32	14.8208
L22 0LG	L22 0	5.565820	1.160650	0.14488900	0.0302138	19.22	14.2390
L22 1AR	L22 1	16.814100	1.430380	0.66512700	0.0565826	18.92	13.9984
L22 2AS	L22 2	4.682420	0.706547	0.08303350	0.0125292	17.22	13.5336
L22 3YB	L22 3	6.570660	0.874987	0.16819100	0.0223973	19.02	14.3458
L22 4RA	L22 4	5.291170	0.985057	0.12311200	0.0229198	19.10	14.3247
L22 5PY	L22 5	7.377040	1.314960	0.26844600	0.0478506	18.86	13.8225
L22 6LH	L22 6	1.490060	0.347021	0.02515520	0.0058584	15.88	13.2418

L22 7RH	L22 7	3.280330	0.674301	0.05537840	0.0113835	16.48	13.4256
L22 8QP	L22 8	15.532400	1.309220	0.50398700	0.0424812	17.45	13.6058
L22 9QQ	L22 9	3.581300	0.966562	0.07539470	0.0203484	18.07	13.7244
L23 0TH	L23 0	30.883400	1.414600	1.14451000	0.0524235	19.90	14.3108
L23 1	L23 1	10.160600	0.372013	1.34074000	0.0490891	16.09	13.8025
L23 2RW	L23 2	20.569900	0.821144	0.82276500	0.0328445	17.52	13.7586
L23 3BW	L23 3	3.540740	0.408011	0.11801700	0.0135995	16.71	13.5456
L23 4TG	L23 4	27.465500	0.251091	1.13613000	0.0103866	13.73	12.7662
L23 4UJ	L38 6	18.352300	0.396443	0.84039600	0.0181541	13.07	12.4922
L23 5TP	L23 5	15.932400	1.629350	0.62748400	0.0641709	19.28	14.0217
L23 6UJ	L23 6	6.364260	0.227529	0.17926600	0.0064090	14.20	12.7352
L23 7YA	L23 7	4.126270	0.232733	0.11776900	0.0066425	15.09	13.0541
L23 8UQ	L23 8	7.650940	0.208845	0.21855700	0.0059659	13.68	12.5491
L23 9UR	L23 9	9.574930	0.354145	0.40856900	0.0151116	17.29	14.0710
L29 1YB	L29 1	1.183400	0.363621	0.05024310	0.0154381	16.21	13.9141
L29 3EA	L29 3	3.180020	0.100016	0.14985000	0.0047130	12.41	12.4251
L29 5XB	L29 5	5.228250	0.280265	0.23415800	0.0125523	14.21	13.0287
L29 7WA	L29 6	4.163140	0.171553	0.19409000	0.0079980	15.23	13.6041
L29 7WA	L29 7	4.769730	0.133270	0.19547500	0.0054617	14.16	13.1876
L29 8YA	L29 8	3.878380	0.120108	0.19087400	0.0059111	13.41	12.8177
L29 9AF	L29 9	3.745950	0.152091	0.16079600	0.0065286	16.09	14.0521
L30 0QH	L30 0	4.118420	0.372201	0.16435400	0.0148535	17.49	14.3530
L30 1PE	L30 1	15.377000	0.909354	8.62973000	0.5103460	21.09	16.2357
L30 2RL	L30 2	17.185200	0.636162	0.59468000	0.0220138	19.11	14.9791
L30 3SU	L30 3	8.356070	0.755894	0.25100200	0.0227057	19.73	15.1782
L30 4UD	L30 4	17.654500	0.774680	3.53657000	0.1551880	21.56	15.9567
L30 5RL	L30 5	7.707690	0.585309	0.23268900	0.0176700	19.49	15.0840
L30 6UQ	L30 6	22.534400	1.078530	10.17430000	0.4869590	20.69	16.3347
L30 7PX	L30 7	27.332600	0.698694	1.04503000	0.0267138	18.24	14.6589
L30 9SL	L30 9	3.094770	0.718844	0.11497200	0.0267054	21.59	15.7849
L31 0BW	L31 0	5.023500	0.467370	0.17513000	0.0162935	14.55	13.5693
L31 1	L31 1	153.578000	0.593802	5.78927000	0.0223840	16.93	14.3577
L31 2ND	L31 2	9.975140	0.344692	0.40846400	0.0141146	13.89	13.3921
L31 3ET	L31 3	10.823200	0.489339	0.35950200	0.0162538	16.34	14.2064
L31 4HS	L31 4	25.195800	0.188711	0.99957300	0.0074866	12.25	12.6251

L31 5JQ	L31 5	17.919100	0.714645	0.62978500	0.0251169	17.40	14.4486
L31 6EA	L31 6	8.303130	0.428650	0.24316300	0.0125533	15.47	13.9121
L31 7DJ	L31 7	7.698030	0.399974	0.22350200	0.0116127	15.94	14.0189
L31 8ED	L31 8	7.735520	0.163038	0.27553600	0.0058074	14.30	13.4668
L31 9AP	L31 9	9.433800	0.348842	0.27243300	0.0100740	15.04	13.7914
L37 0AH	L37 0	18.802500	0.210527	0.79131600	0.0088602	10.03	11.7885
L37 1LB	L37 1	18.926400	0.108459	0.75330100	0.0043169	9.04	11.2930
L37 2EN	L37 2	15.737300	0.184126	0.40695100	0.0047613	9.51	11.5038
L37 3HZ	L37 3	17.724600	0.461388	0.48204300	0.0125480	10.98	12.1284
L37 4EU	L37 4	3.530620	0.178450	0.13162000	0.0066525	10.62	12.1112
L37 6DQ	L37 6	13.544100	0.357398	0.63017100	0.0166288	10.74	12.0606
L37 7HN	L37 7	20.518300	0.246271	0.89737300	0.0107707	9.74	11.7073
L37 8DP	L37 8	10.628400	0.594624	0.39040800	0.0218419	11.57	12.2022
L38 0BB	L38 0	1.195260	0.091184	0.05091030	0.0038839	10.33	11.6212
L38 1QF	L38 1	17.643200	0.247832	0.81489200	0.0114467	11.68	12.1469
L38 3RB	L38 3	13.334400	0.170196	0.58532400	0.0074709	11.60	12.0664
L38 4JB	L38 4	10.008000	0.353828	0.45882400	0.0162215	10.65	11.9223
L38 5DA	L38 5	9.996530	0.302758	0.45831700	0.0138807	10.99	11.9593
L38 7JE	L38 7	1.151040	0.091337	0.05327280	0.0042273	10.15	11.7084
L38 8AE	L38 8	8.462360	0.127029	0.28734300	0.0043134	9.81	11.5296
L38 9EP	L38 9	5.526810	0.159792	0.19203300	0.0055521	11.16	11.7959
L9 0NB	L9 0	0.598682	1.018840	0.01442110	0.0245418	22.90	16.1961
L9 5AB	L10 3	19.671600	1.508370	0.77486300	0.0594146	20.21	15.2972
L9 5AB	L30 8	11.852800	1.617580	0.42283200	0.0577050	20.37	15.3825
L9 5AB	L9 5	19.294000	0.507509	1.09714000	0.0288594	20.66	16.2239
L9 8DQ	L9 8	0.717540	0.325694	0.02628580	0.0119312	22.33	15.9905
PR8 1HN	PR8 1	18.886700	0.337523	0.58736800	0.0104968	10.79	12.0987
PR8 2BA	PR8 2	88.794500	0.315801	3.48994000	0.0124121	9.56	11.5764
PR8 3DJ	PR8 3	46.218700	0.428944	1.55394000	0.0144217	9.85	11.7056
PR8 4JA	PR8 4	30.279300	0.517311	1.13570000	0.0194031	11.39	12.5691
PR8 5HS	PR8 5	25.534900	0.554247	1.07996000	0.0234411	12.10	12.9998
PR8 6AE	PR9 0	14.755200	0.272986	0.53578400	0.0099126	11.44	12.2967
PR8 6LH	PR8 6	47.201700	0.652494	1.53526000	0.0212229	12.72	13.0346
PR9 7QS	PR9 7	56.114200	0.459496	6.56434000	0.0537535	12.27	12.6630
PR9 8AE	PR9 8	13.025100	0.388822	0.46328600	0.0138299	10.83	12.2379

Appendix I

Venn diagram software


Venn Diagram Plotter
File Edit Help
Circle Parameters
Circle A: Circle B: Circle C
Total 50 100 75
Count Distinct 35 85
Overlap: A/B 15 B/C 20 A/C 16
Circle A
Circle B
Circle C
Background
AB Overlap
BC Overlap
AC Overlap
ABC Overlap
Tasks
Clipboard
Save file
Refresh
Use fill color
Message Options
Display Time 8 sec.
Ignore window 4 sec.
Hide old msgs on valid update
SVG Options
Save Svg File
Opacity 0.75
Region counts for Three Circle Venn Diagram
Only in A 44
In A and B
Only in B 90
In All 3 25
In A and C
In B and C
Only in C 64
Unique item count across all circles 199
Update Counts
Compute Optimal
Define count overlapping all 3 circles 25
Image Adjustment
Reset
Rotation: 0
X Offset: 0%
Y Offset: 0%
Zoom: 100%

Appendix J

Ethical Committee Approval letter

Liverpool Paediatric Research Ethics Committee
Ground Floor
1 Arthouse Square
61-69 Seel Street
Liverpool
L1 4AZ

Telephone: 0151 293 7541
Facsimile: 0151 296 7536

20 June 2006

Professor Bernard Brabin
Head of Child and Reproductive Health Department
Liverpool School of Tropical Medicine
Pembroke Place
Liverpool
L3 5QA

Dear Professor Brabin

Full title of study: **Childhood Asthma,Obesity Prevalence and Parental Smoking in Merseyside**
REC reference number: **06/Q1502/41**

Thank you for your letter of 15 June 2006, responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair who has requested, that you provide a copy of the finalised questionnaire when completed

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised.

Ethical review of research sites

The Committee agreed that all sites in this study should be exempt from site-specific assessment (SSA). There is no need to complete Part C of the application form or to inform Local Research Ethics Committees (LRECs) about the research. The favourable opinion for the study applies to all sites involved in the research

Conditions of approval

The favourable opinion is given provided that you comply with the conditions set out in the attached document. You are advised to study the conditions carefully.

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	*Version*	*Date*
Application		31 March 2006
Investigator CV		10 April 2006

Protocol		10 April 2006
Covering Letter		05 April 2006
Letter from Sponsor		10 April 2006
Statistician Comments		
Questionnaire: v1		10 April 2006
Letter of invitation to participant		10 April 2006
GP/Consultant Information Sheets		10 April 2006
Participant Information Sheet: v1		10 April 2006
Participant Consent Form: v1		10 April 2006
Response to Request for Further Information		
Statement of indemnity		10 April 2006

Research governance approval

The study should not commence at any NHS site until the local Principal Investigator has obtained final research governance approval from the R&D Department for the relevant NHS care organisation.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

06/Q1502/41	**Please quote this number on all correspondence**

With the Committee's best wishes for the success of this project

Yours sincerely

Chair

Email: Ronald.Wall@centralliverpoolpct.nhs.uk

Enclosures: *Standard approval conditions*

Appendix K

Sefton Council Project Approval letter

Department of Built Environment
1st floor, Magdalen House
30 Trinity Road, Bootle
Merseyside, L20 3NJ

Dr Gibby Koshy
Liverpool School of Tropical Medicine

Date: 25/04/2014
Our Ref: DoBE/EP/DRP
Your Ref: LSTM

Please contact: David Packard
Contact Number: 0151 934 4016
Fax No: 0151 934 4276
e-mail: david.packard@sefton.gov.uk

Dear Gibby

I confirm the approval of Sefton Council for the use of the following data to support analytical dissertations or similar studies:

- 2006 Emissions Data from the Merseyside Atmospheric Emissions Inventory.
- 2006 Concentration data modelled using the Airviro Dispersion Model.
- 2006 Monitoring data from the monitoring station operated by Sefton Council.

Yours sincerely

David Packard
Head of Environment Services

Appendix L

Differences between MPhil and PhD Thesis

Table A Differences between MPhil and PhD thesis

Contents	MPhil	PhD
Air pollution	Yes	No
University	University of Liverpool	University of Amsterdam
Mode	Based on thesis	Based on published papers
Thesis purpose	Original research on air pollution and substantial development of previous work to address air pollution issue, when combined with pregnancy smoking exposure for the sake of completeness and with clear identification of previous work	Original research on pregnancy smoking and birth and child health outcomes/parental asthma and birth outcomes, asthma trends, parental compliance
Previous research	No previous research reported on combined air pollution and pregnancy smoking by data linkage method using post code sectors and assessment of multiple birth and child health outcomes at the same time	Previous studies on pregnancy smoking with different publications on individual outcomes
Thesis title	Application of Merseyside Community Child Health Survey data for estimating health risks from air pollution and exposure to cigarette smoke during pregnancy	Pregnancy Smoking, Child health and nutrition
Exposure factor	Air pollution / Combined air pollution and pregnancy smoking exposure	Pregnancy smoking and parental asthma
Main exposure subcategories	NO_X emissions PM_{10} emissions NO_2 concentrations PM_{10} concentrations	MSDP PSDP HSDP Parental asthma
Recreation of new exposure categories	Yes	No
Total exposure categories	36	5
Sample size	972	1074
Number of surveys	1	4
Study location	Liverpool	Liverpool and Wallasey
Setting	Community	Community + hospital
Years of survey	2006	1991,1993,1998 and 2006
Study period	2006	Community data 1991-2006 Hospital data 1998-2003
Post code level	Post code sector	Full post code
Field visits	Air quality monitoring stations Merseyside Atmospheric Emissions Inventory	15 primary schools

	Sefton Council	
Data matching	Yes	No
Schools	10	15
Hospital data	No	Yes
Pregnancy smoking	Combined	Individual
Biomarkers details	Combined air pollution and pregnancy smoking	Pregnancy smoking alone
Literature review	Air pollution / combined air pollution and pregnancy smoking exposures and birth and child health outcomes	Pregnancy smoking exposure and birth and child health outcomes
Asthma trends	No	Yes
Data Linkage	Yes	No
Statistical Framework	Major component	Minor component
Advanced Statistical Analysis	More advanced with emphasis on Visual Analytical techniques and new analysis software	Not used
Advanced Statistical software used	Arc GIS SPSS Amos Venn diagram Plotter	Not used
Interaction Analysis	Yes	No
Structural Equation Modelling	Yes	No
Spatial Mapping	Yes	No
Venn diagrams	Yes	No
Compare means method	Yes	No