TO STUDY THE EPIDEMIOLOGICAL ASPECTS IN DETERMINING THE PREVALENCE AND EXPRESSION OF ASTHMA PHENOTYPES IN THE URBAN POPULATION OF NORTH CHENNAI

Dissertation submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the requirements for the degree of

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Tuberculosis and Respiratory Diseases

Branch – XVII

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APRIL 2017

BONAFIDE CERTIFICATE

This is to certify that the dissertation "To study the epidemiological aspects in determining the prevalence and expression of asthma phenotypes in the urban population of north Chennai" is the Bonafide work done by **Dr. P. Dhamodharan** during his **MD (Tuberculosis and Respiratory Diseases)** course from June 2014 to May 2017 at Government Kilpauk Medical College, Chennai.

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This is to certify that the dissertation titled "To study the epidemiological aspects in determining the prevalence and expression of asthma phenotypes in the urban population of north Chennai" is the Bonafide work done by **Dr. P. Dhamodharan,** during his MD **(Tuberculosis and Respiratory Diseases)** course in the academic year 2014-2017 at Government Kilpauk Medical College, Chennai, under my guidance

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DECLARATION BY THE SCHOLAR

I**, Dr. P. Dhamodharan,** solemnly declare that the dissertation titled "To study the epidemiological aspects in determining the prevalence and expression of asthma phenotypes in the urban population of north Chennai**"** has been prepared by me. This is submitted to "**The Tamil Nadu Dr. M.G.R. Medical University, Chennai"** in partial fulfilment of the requirement for the award of M.D degree examination branch XVII **Tuberculosis and Respiratory Diseases** from June 2014 to May 2017.

Place: CHENNAI **(**Dr. P. DHAMODHARAN)

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CONTENTS

INTRODUCTION

Asthma is a heterogeneous disease usually characterized by chronic airway inflammation which is defined by the history of respiratory symptoms like wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation. [1]

These symptoms and airflow limitation characteristically vary over time and in intensity which are often triggered by factors such as exercise, allergen, and change in weather conditions or viral respiratory infections. They may resolve spontaneously or in response to medications but sometimes flares up resulting in life threatening exacerbations carrying significant burden to patients and the community. Asthma is usually associated with airway hyper responsiveness to either direct or indirect stimuli and with chronic airway inflammation.

Asthma is a common disease whose prevalence has increased throughout the world for several decades. For many years the major focus of asthma investigations and treatment was on allergic mechanisms. More recently, studies of the epidemiology, natural history and pathogenesis have clearly demonstrated that asthma is a heterogeneous disease with multiple etiologies, contributing cofactors, complex pathobiologic mechanisms, and different molecular phenotypes.

Understanding these differences is critical for developing various phenotypes of asthma that will be effective for better asthma management.

Fig 1: Symptoms of Asthma

BURDEN OF ASTHMA

- Asthma is one of the most common chronic diseases worldwide with an estimated 300 million affected individuals[2]
- Prevalence is increasing in many countries, especially in children
- Asthma is a major cause of school and work absence
- Health care expenditure on asthma is very high [2]
  - Developed economies might expect to spend 1-2 percent of total health care expenditures on asthma
  - Developing economies likely to face increased demand due to increasing prevalence of asthma
  - Poorly controlled asthma is expensive
  - However, investment in preventive medication is likely to yield cost savings in emergency care

PREVALENCE OF ASTHMA

Asthma prevalence has been steadily increasing over time. Although a family history of allergy is the strongest risk factor for asthma, early life infections are

important cofactors. The increasing prevalence of asthma may relate to the success of domestic hygiene in reducing the rate of exposure to bacterial products or change in the commensal microbiome in early childhood, which would otherwise consolidate antibacterial rather than allergic immune responses.

On the other hand, viral respiratory infections in early childhood are thought to increase the risk for wheezing illnesses and asthma over time. A range of other exposures have been identified as risk factors for asthma, including, diet, stress, exposure to farm products in childhood, second hand smoking, obesity, air pollution, antibiotic use, aspirin use, exercise and occupational exposures.

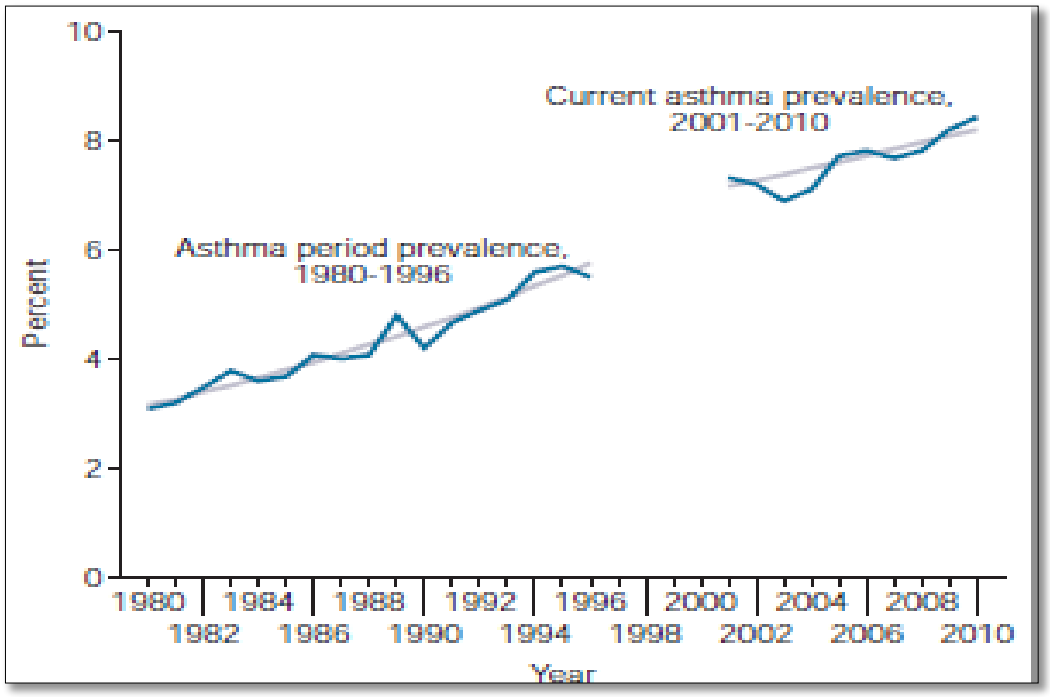

Fig 2: Prevalence of current asthma in United States

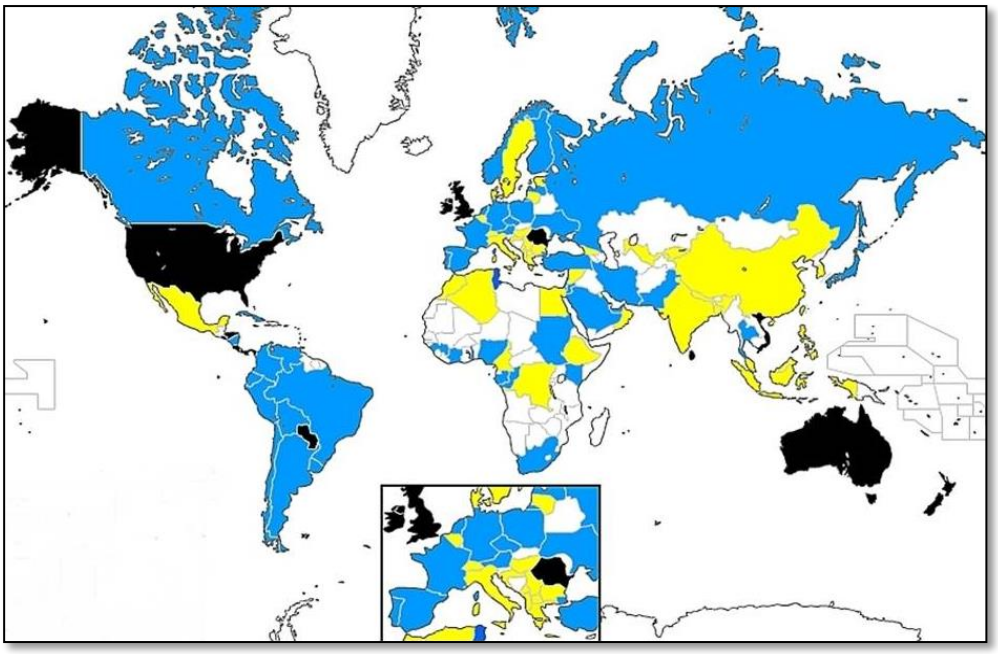

Fig 3: Estimated prevalence of asthma in children (13-14 years)

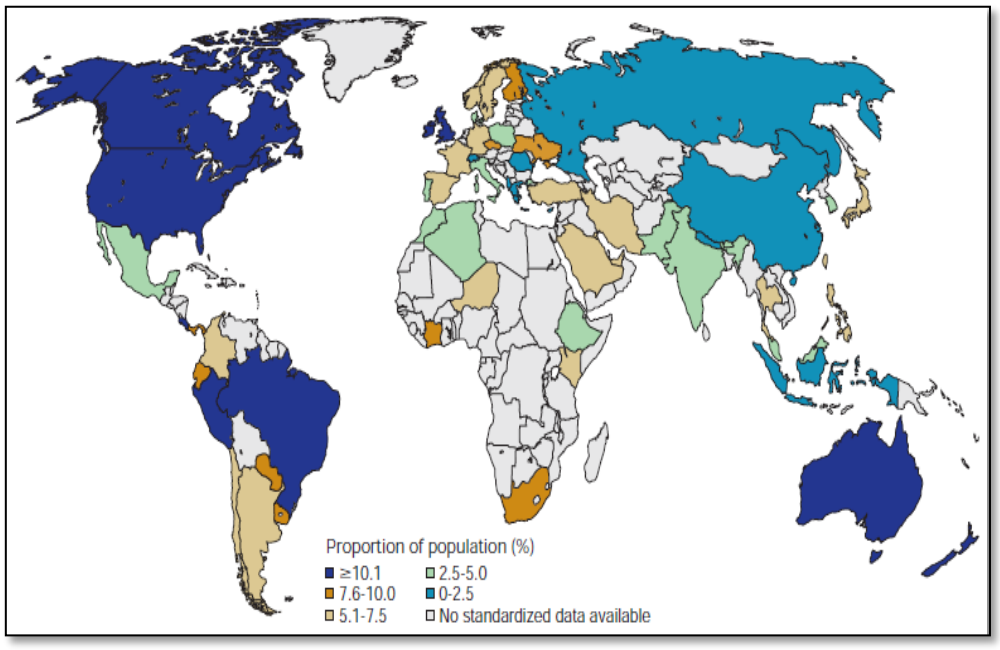

Fig 4: Estimated worldwide prevalence of clinical asthma

In 2012, current asthma prevalence was very high in black non-Hispanics (11.9%), those of Puerto Rican heritage (18.8%), and among those living below the poverty threshold (12.4%). Current asthma prevalence also was higher among children

(9.3%) than adults (8.0%) and among females (9.5%) than males (7.0%). The female-to-male balance changes over development with asthma less common in females than males during childhood (age younger than 18, 8.6% vs. 10%, respectively) but more common in females than males during adulthood (age 18 or older, 9.8% vs. 6%, respectively) [3]

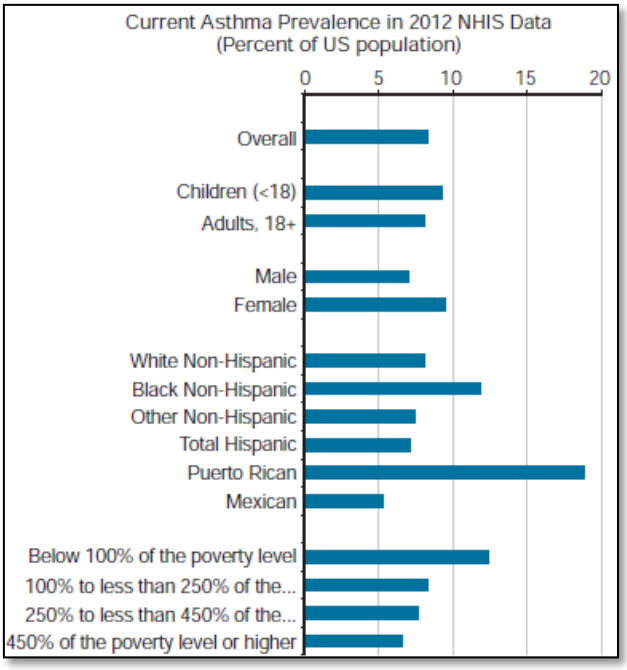

Fig 5: Current asthma prevalence in US by age, gender, ethnic groups and income

RISK FACTORS OF ASTHMA

ALLERGY

The strongest risk factor for asthma is a family history of atopy [4][5]. This increases the risk of developing allergic rhinitis by fivefold and the risk of asthma by threefold to fourfold [6]. In children 3 to 14 years old, both positive skin tests and increases in total serum IgE are strongly associated with asthma [7][8]. Serum IgE also correlates strongly with bronchial hyper responsiveness [9]. In adults, the odds of having asthma increase with the number of positive skin tests to common allergens [10].

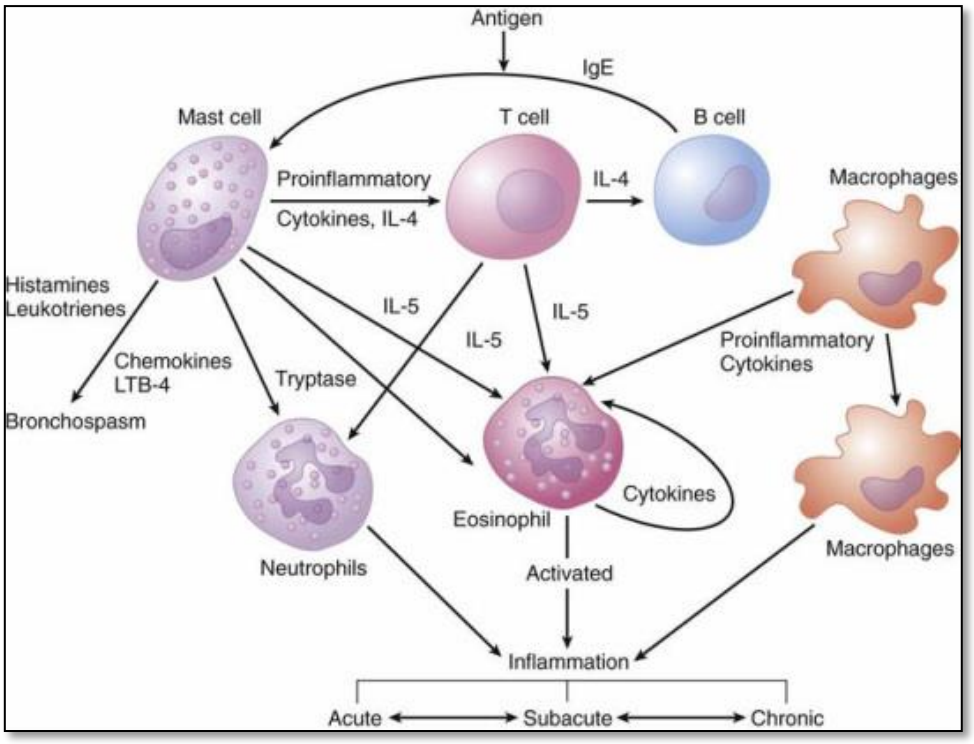

Fig 6: Pathophysiology of atopy in asthma

Allergic asthma is associated with sensitivity to allergens of the indoor environment and these allergens are considered as a primary cause of the rise of asthma in infancy and early childhood. Specific allergens of interest includes house dust mite[11][12], dog and cat dander [13] and cockroach allergens [14]

(a) (b)

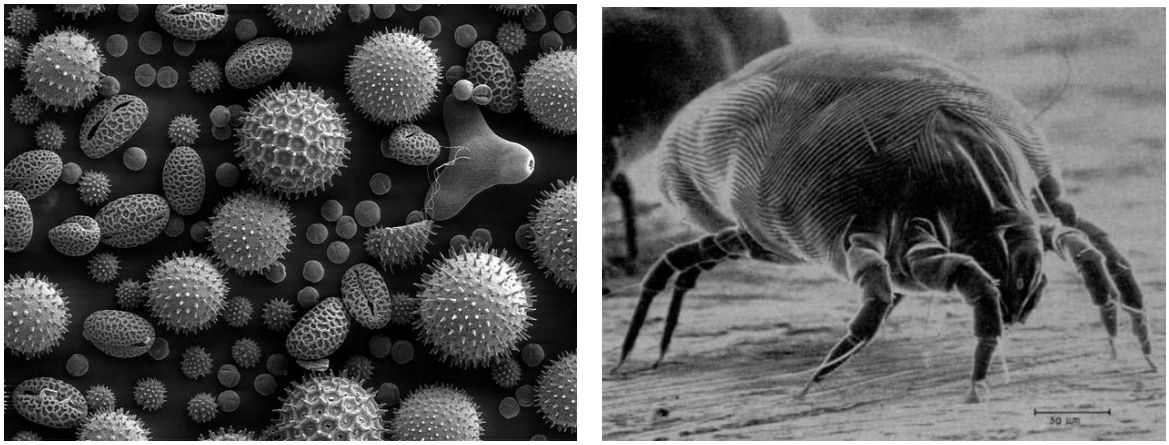

Fig 7: Causative agents in allergic asthma (a) Pollen dust (b) Dust mite

HYGIENE HYPOTHESIS

The cause of increase in asthma and allergies in westernized countries is the "hygiene hypothesis". This holds that the rise in allergies in children is an unintended consequence of the success of domestic hygiene in reducing the rate of infections or exposure to bacterial products in early childhood. This hypothesis was put forward to explain the inverse relationship between hay fever and family size [15].

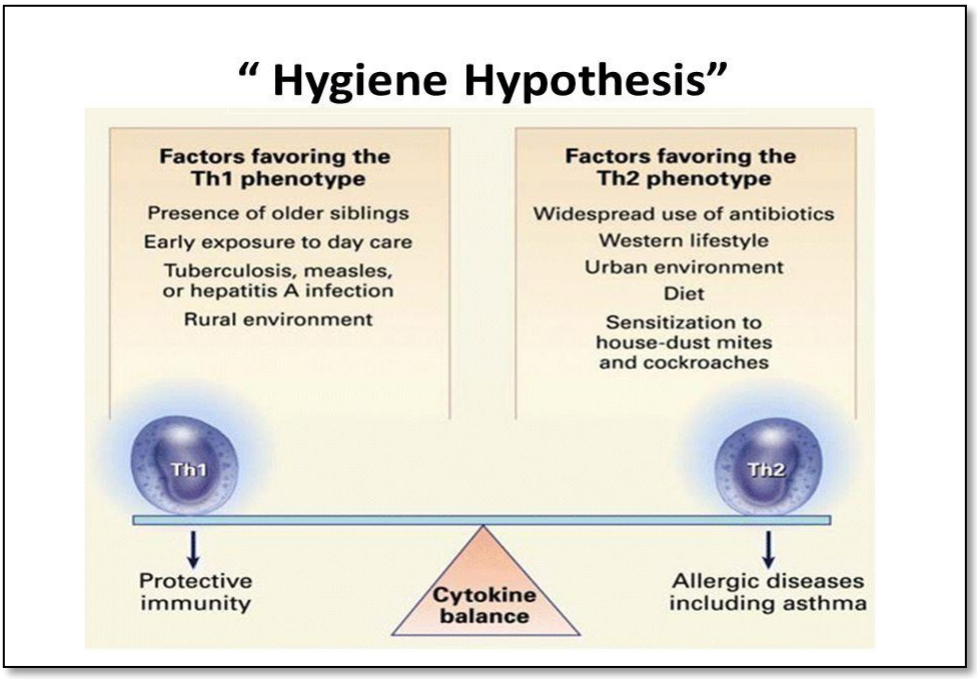

Fig 8: Immunology in hygiene hypothesis

In these studies, children who lived on farms had a lower prevalence of hay fever and asthma than their peers who did not live in an agricultural environment. The reduction in risk was stronger for children whose families were running the farm on a fulltime basis, and stronger yet if the farm included livestock [16][17]. Factors related to environmental influences, such as increased exposure to bacterial compounds in stables, may prevent the development of allergic disorders in children. Continual long-term

exposure to stables until age 5 was associated with very low rates of asthma (0.8%), hay fever (0.8%), and atopic sensitization (8.2%) [18].

HUMAN MICROBIOME

One potential link between changes in hygiene and allergic disease is the effect that "improved" hygiene may have on our indigenous microbiota and the role this microbiota may play in shaping our immune system [19-23]. The biologic model most commonly cited to explain this association is that early-life exposure to factors that promote Th1 immunity are necessary to blunt exuberant *type 2 T helper* (Th2) immunity[24-32].

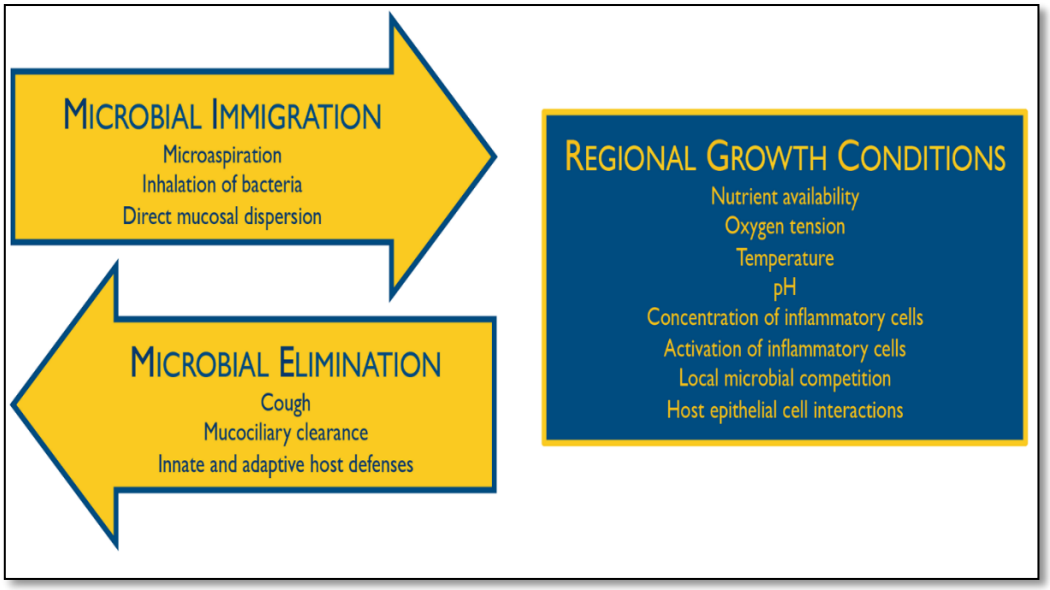

Fig 9: Role of human microbiome in asthma

RESPIRATORY VIRAL INFECTIONS

Viral respiratory tract infections play in the development of asthma [33]. Children who have *lower respiratory tract infections* (LRIs) caused by *respiratory syncytial virus* (RSV) are at a threefold to fourfold risk of subsequent wheezing during the early school years [34-37]. The association between viral LRIs and subsequent asthma depends on

concurrent atopic disease, suggesting that an interaction between atopic predisposition and LRI at an early developmental stage may be critically important [38].

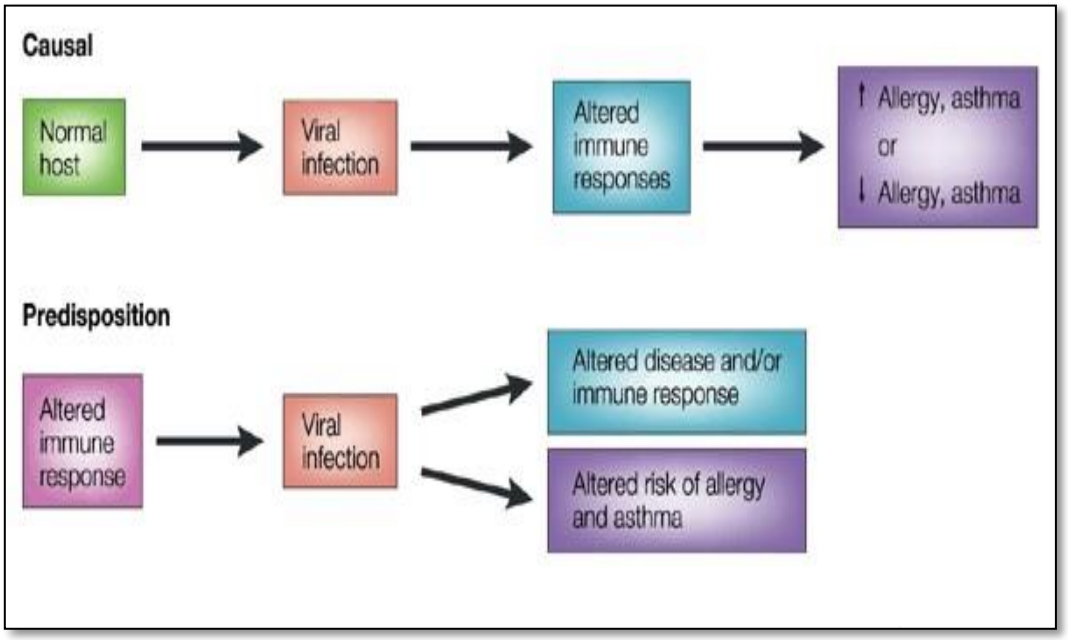

Fig 10: Role of respiratory viral infections in asthma

ATYPICAL BACTERIAL INFECTIONS

Two bacterial causes of "atypical" pneumonia have been implicated in the development of chronic wheezing illnesses, *Chlamydia pneumoniae* and *Mycoplasma pneumoniae*. They are associated with an increase in the tissue mast cells and these atypical infections are associated with asthma exacerbations [39][40]. Both organisms are sensitive to macrolide antibiotics, and several studies have evaluated the utility of macrolides in patients with chronic asthma with variable results.

AIR POLLUTION

The role of air pollution in contribution to the development of asthma is still uncertain. It was widely accepted that air pollution can exacerbate pre-existing asthma[41][42]. It has been postulated that exposure of the lung to air pollution could

increase local oxidative stress, induce or modify local inflammation, enhance sensitization to allergens, impair lung development, or injure small airways. Several recent studies focused specifically on asthma incidence and prevalence by proximity to heavy automobile traffic and suggested that exposure to respirable particulate matter and NO2 in this setting are both associated with the future development of asthma[43-48].

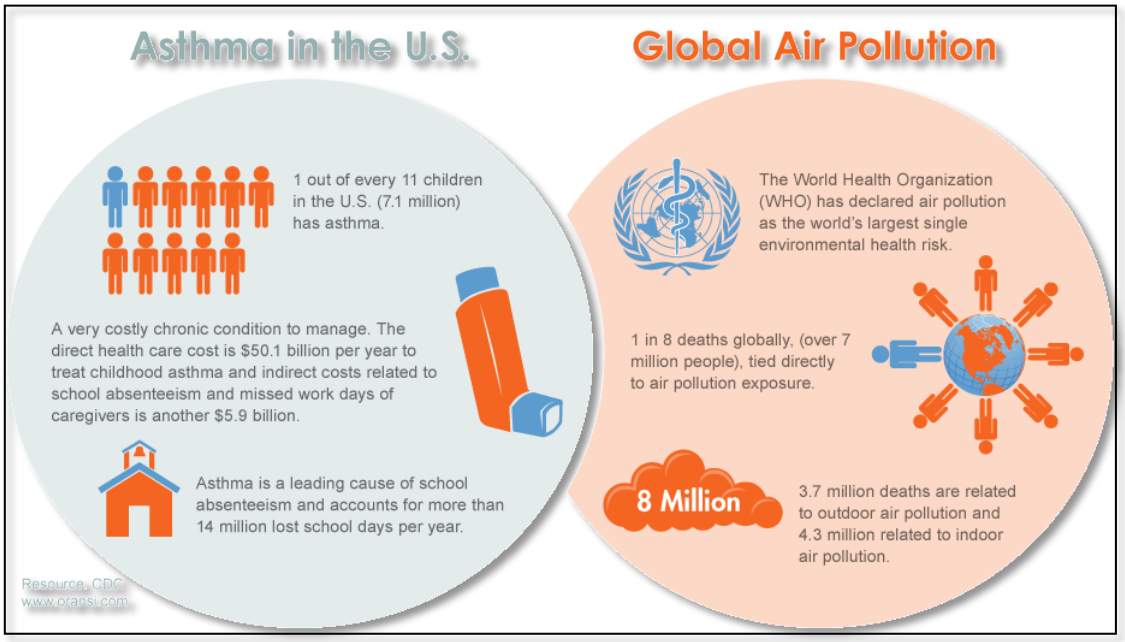

Fig 11: Role of air pollution in asthma

OCCUPATIONAL EXPOSURES

Occupational exposures constitute an important risk factor for a specific subset of patients. Asthma induced by occupational exposures accounts for up to 17% of all adult-onset asthma [49]. Occupational asthma can either result from immunologically mediated sensitization to occupational agents (i.e., sensitizer-induced occupational asthma) or from exposure to high concentrations of irritant compounds (i.e., irritant-induced occupational asthma)

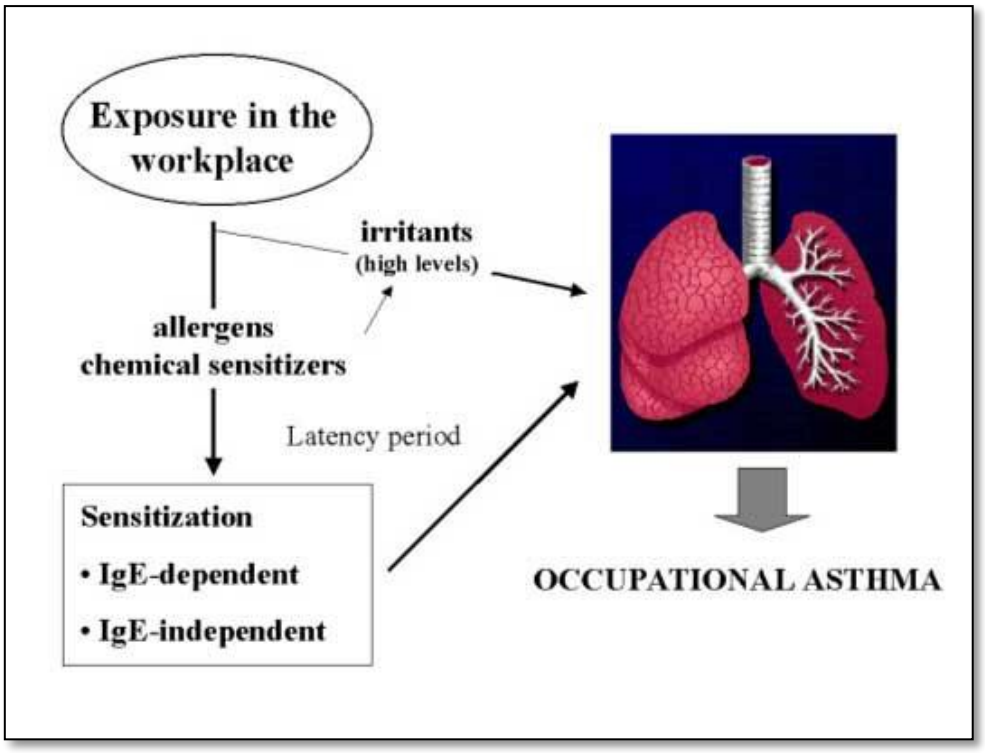

Fig 12: Role of occupation in asthma

PATHOGENESIS OF ASTHMA

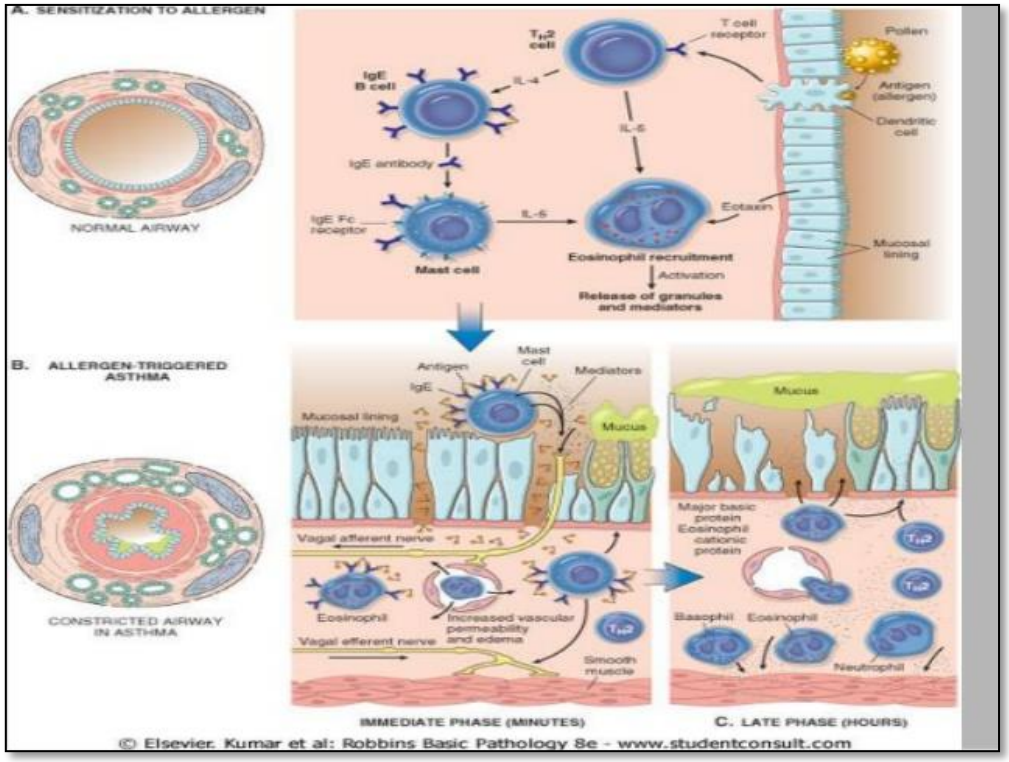

Fig 13: Pathophysiology of asthma

PHENOTYPING

ASTHMA HETEROGENEITY

Patients with asthma can have a great deal of heterogeneity with respect to severity of airflow limitation, symptoms, degree of reversibility, and therapeutic response. Up to 30% to 45% of asthmatics do not respond to high doses of *inhaled corticosteroids* (ICSs) with improvements in lung function [50][51].

There is significant heterogeneity in asthma triggers, the frequency and severity of exacerbations, and long-term outcomes such as irreversible loss of lung function due to airway remodelling.

Several approaches have been taken to assign asthmatics to distinct sub phenotypes. A better appreciation of disease heterogeneity at a molecular and cellular level will be important in treating severe asthma and in the clinical application of emerging asthma therapies.

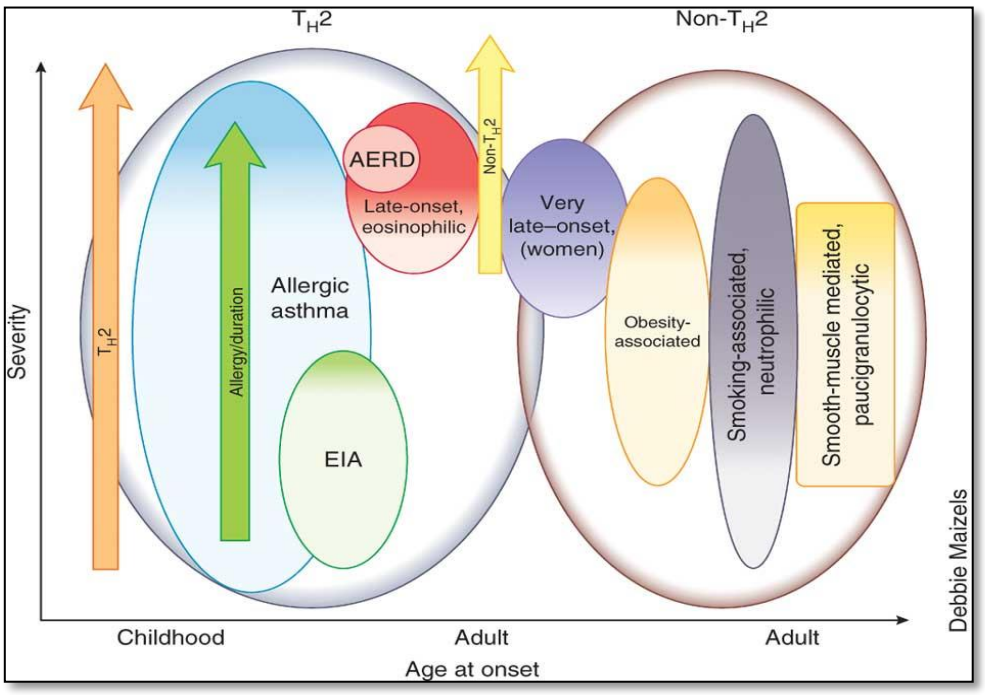

Fig 14: Schematic representation of various asthma phenotypes

CELLULAR PHENOTYPES

Analysis of sputum, bronchoalveolar lavage and endobronchial biopsy specimens from asthmatic patients had found that majority of asthmatics had elevated eosinophils [52]. Non-eosinophilia is seen in 25% of asthmatics [53]. In severe asthma, noneosinophilic type is seen and it is mainly associated with a lower FEV1, fewer mast cells and less sub epithelial fibrosis [54]. There are four categories of cellular classification of asthma based on induced sputum cytological analysis (1) eosinophilic, (2) neutrophilic, (3) mixed eosinophilic and neutrophilic, and (4) paucigranulocytic asthma, where there is no observable presence of inflammatory cells [55][56].

CLINICAL PHENOTYPES

Cluster-based multivariate approaches, designed to overcome the limitations of using only one variable, such as severity of airflow obstruction or type of cellular inflammation had identified three distinct clusters in mild to moderate asthmatics: one with early-onset atopic asthma and eosinophilia; another with a preponderance of obesity, females, and lack of eosinophilia; and a third with mild disease and lack of airway eosinophilia [57].

MOLECULAR PHENOTYPES (ENDOTYPES)

An alternative approach to clustering subjects with asthma is to group them on the basis of molecular pathways found to be active in individual patients. Creating subgroups based on the activity of specific cytokine pathways has the added advantage that it points to specific pharmaceutic targets and biomarkers for clinical trials. Subgroups of patients who share an underlying disease biology have been named "endotypes."[58]

AIMS AND OBJECTIVES

1. To study the epidemiology of bronchial asthma phenotypes in urban population of North Chennai.

2. To assess the influence of environmental exposure on the prevalence and expressions of various phenotypes of bronchial asthma.

REVIEW OF LITERATURE

Asthma is a complex disease which includes distinct phenotypes with different etiologies, natural histories and treatment responses. Asthma impacts significantly on the rising burden of chronic disease in the developing countries. Approximately 5 to 10% of patients have refractory asthma which was poorly controlled despite maximal inhaled therapy.

Many distinct phenotypes had been identified based on a limited number of characteristics. Most common phenotypes includes the allergic and non-allergic asthma. Other phenotypes defined by clinical and physiological categories like severity, age at onset and chronic airflow obstruction, asthma triggers like exercise, allergens, occupational allergens or irritants or their pathobiology like eosinophilic or neutrophilic asthma had been proposed.

The heterogeneity in the physiologic, pathologic and molecular abnormalities makes the effective clinical care more complicated. Thus identification of more distinct phenotypes of asthma would be possible by comprehensive examination protocol of asthma patients incorporating several domains of the disease. This kind of characterization of asthma would allow a better understanding of the aetiology of asthma and by detecting the environmental and genetic risk factors. Poor coherence and individual subjectivity limits the current description of asthma phenotypes.

Incorporation of multidimensionality of asthma in identifying the subgroups with consistent pattern of the disease provides a framework for identifying distinct

phenotypes with specific abnormalities that predict response to particular therapies focussing the current genetic and molecular studies.

The aim of the present study is to identify distinct asthma phenotypes for use in aetiological studies and towards a personalised treatment of asthma.

ASTHMA WITH OBESITY

The prevalence of asthma and obesity had increased substantially in recent decades in many countries which had led to a state that obese persons might be at increased risk of asthma development. In adults many studies had been done which were consistent with the role of obesity in the pathogenesis of asthma. The incidence of obesity has also been positively associated with obesity. Asthma comprises diverse phenotypes reflecting heterogeneity in a number of characteristics associated with obesity. Obesity is associated with increased prevalence of asthma especially in women and appears to be more severe in the obese.

In a study conducted by Andrea Lessard et al, 44 consecutive obese subjects (BMI≥30 kg/m^2) and 44 consecutive non-obese subjects (BMI<25 kg/m^2) al with asthma were included in study. The asthma control was poorer in obese subjects than in non-obese subjects (P=0.005). They concluded that obese people with asthma had poorer asthma control than the non-obese asthmatics despite similar symptoms perception. Such bronchial and systemic characteristics and the specific pattern of pulmonary function changes suggested a different phenotype of asthma in these obese subjects [59].

Another study by Beuther et al, A Meta-analysis of Prospective Epidemiological Studies, relationship between BMI and incident of asthma was studied and the impact

of sex was evaluated. Data was analysed by inverse-variance weighted, random effects meta-analysis. Stratified analysis between BMI categories and sex was performed. The results were of that when compared with normal weight, overweight and obesity showed increased odds of incident asthma with odds ratio of 1.51. They concluded that overweight and obesity were associated with a dose dependent increase in the odds of incident asthma in men and women and suggested that asthma incidence could be reduced by intervention targeting overweight and obesity [60].

In a study done by Holguin et al, they compared the associations between BMI and clinical parameters across age of onset phenotypes and to compare the rate of BMI change in relation to asthma duration by age of onset phenotypes. Multivariate logistic regression analysis was done to evaluate the association. In a study population consisting of 1049 subjects, the median age of onset was 10 years(interquartile range of 4-25 years); 48% had late onset asthma (≥12 years) and 52% had early onset asthma (<12 years). Compared to obese subjects with late onset asthma, obese subjects with early onset asthma had more airway obstruction and recurrent admissions. They concluded that the asthma subjects were affected differently by obesity and the results highlighted the need to understand obesity as a co-morbidity that affects specific clinical phenotypes and not all asthma subjects are alike [61].

In a study done by Camargo et al, they performed a prospective cohort study of female nurses in the Nurses' Health Study II, the main outcome measure was self-report of physician-diagnosed asthma with recent use of an asthma medication. They found 1596 incident cases of asthma. In a multivariate model controlling for 9 potential confounding factors (including age, race, smoking, physical activity, and energy intake), the relative risks of asthma for 6 increasing categories of BMI in 1991 were 0.9,

1.0 (reference), 1.1, 1.6, 1.7, and 2.7 (*P* for trend <.001). Stronger associations were found using stricter definitions for asthma, and the finding was present in a variety of subgroups. In analyses controlling for the same variables, as well as BMI at age 18, women who gained weight after age 18 were at significantly increased risk of developing asthma during the 4-year follow-up period (*P* for trend <.001). They concluded that BMI has a strong, independent, and positive association with risk of adult-onset asthma. The increasing prevalence of obesity in developed nations may help explain concomitant increases in asthma prevalence [62].

AGE OF ONSET IN DETERMINING ASTHMA PHENOTYPE

Age of asthma onset is often used to distinguish different adult asthma phenotypes but however similarities and differences between early and late onset adult asthma have not been summarized till date.

In a study done by Tan et al, they found 12 studies comparing early and late onset asthma and age 12 was most commonly used to delineate the two age of onset phenotypes. Atopy was more likely associated with adults with early onset asthma and also higher frequency of asthma attacks. And adults with late onset asthma were mostly female, smokers and increased levels of spirometrically defined fixed airflow obstruction. They concluded that distinct phenotypic differences were found in relation to the age of asthma onset. Although early onset asthma was more likely attributed to atopy and potential genetic factors, late onset adult asthma appears to be more related to environmental risk factors and better targeted by preventive strategies [63].

Although asthma is usually considered to originate in childhood, adult-onset disease is being increasingly reported in recent years. In a cohort study done by Akshay Sood et al, titled Adult-onset asthma becomes the dominant phenotype among women

by age 40, they studied the adult and paediatric onset asthma phenotypes using a three-way analysis of covariance model and found out that the asthma of adult onset became the dominant (>50%) phenotype in women by 40 years and it was further lowered for obese, non-atopic group. They concluded that the studies of the differences between paediatric and adult onset asthma might provide greater insight into the phenotypic heterogeneity of asthma [64].

In a study done by Christina Miranda et al, they did a cross sectional analysis of integrated clinical, physiologic and pathologic data collected from 80 subjects with severe asthma. The subjects were divide into 2 groups, one with asthma onset before age 12 years (n=50) and second group with onset after age 12(n=30) and with the presence or absence of lung eosinophils. The results came as those subjects with early onset, severe asthma had significantly more allergen sensitivity (skin test positivity, 98% vs 76%, $P<0.007$) and more allergic symptoms (P values ≤ 0.02) than subjects with late onset asthma. In contrast, subjects with late onset asthma had lower lung function than early onset despite a shorter duration of illness. Both groups had high degree of asthma symptoms and those with high eosinophils had lower lung function and only the early onset asthmatics presented with lymphocytic or mast cell inflammatory process. They concluded that differentiating asthma by age of onset and presence or absence of eosinophils identifies the phenotypes of asthma [65].

In another study done by Valerie Siroux et al, they assessed the relationship of eosinophils, IgE and atopy with asthma according to gender and age of onset. Data was obtained from the Epidemiological study on the Genetics and Environment of Asthma, Bronchial Hyper responsiveness and Atopy. Adults and children with asthma recruited in chest clinics (n = 313) and 1st degree relatives of patients with asthma (n = 214) were

compared with non-asthmatic controls (n = 334) and first-degree relatives without asthma (n = 595). They found that in women, eosinophilia was significantly associated with perimenstrual asthma independent from age, smoking and asthma severity (eosinophil/mm^3 330 vs 194; p=0.01). In non-asthmatic women, IgE was significantly decreased and atopy decreased. Considering both the genders, the increase in the eosinophil count with asthma was significantly greater in women with childhood onset asthma than in women with adulthood onset or in men. No interaction between gender and asthma was observed for eosinophils in children [66].

FAMILY HISTORY OF ASTHMA AND ITS INFLUENCE ON OFFSPRINGS

Family history of asthma and allergies had also played a significant role in the risk of developing asthma in childhood. Although heredity plays a major role in asthma and other allergic diseases, mechanisms underlying the pattern of inheritance of these disorders were poorly understood as well as the relative contribution of maternal and paternal conditions to the risk of the disease. Many studies had been shown that the family history of asthma and allergy increased the risk of asthma in childhood. Based on the prospective birth cohort, Martinez et al had proposed that parental history of asthma and allergy related more strongly to early onset asthma that persists later into childhood.

A cross sectional study was conducted by London et al analysing the relation between family history and the types of asthma and found out that for children with two asthmatic parents, the prevalence ratio for early onset persistent asthma was 12.1 when compared with 7.51 for early onset transient asthma and 5.38 for late onset asthma. They concluded that that the parental history of asthma and allergy was most strongly

associated with early onset persistent asthma. They suggested that in children who are genetically predisposed and who an early environmental exposure and maternal smoking during pregnancy had developed early onset asthma that persists into early childhood [67].

Another study done by Litonjua et al they investigated the maternal and paternal asthma, eczema and hay fever as cross sectional predictors of childhood asthma and allergic disease in 306 children with median age of 3.5 years from families in which at least one parent had a history of either asthma or other allergic conditions. The results were that for asthma in particular, maternal asthma was most strongly associated with asthma in child of all ages in both univariate (OR=3.2) and multivariate (OR=4.1) models. Among children <5 year, the risk of childhood asthma associated with maternal asthma was greater (OR-5.0) than the risk associated with paternal asthma (OR=1.6) where as both maternal asthma and paternal asthma were associated with similar risks among the children ≥5 year of age (OR=4.6 and OR-4.1 respectively). They concluded that the odds of having asthma in child was 3 times greater in families with one asthmatic parent and 6 times greater in families with two asthmatic parents. Also inhalant allergy in one parent had also conferred additional risk in the presence of asthma in other parent [68].

In a study done by Mutius et al, they investigated school children (n=9403), 9-11 years of children were enrolled in a cross sectional survey. The prevalence of asthma and allergic diseases in parents and children were assessed by a parental questionnaire. Atopic sensitization was measured by skin prick tests, and bronchial responsiveness was determined by cold air hyperventilation challenge. The prevalence of asthma alone increased strongly if nearest of kin suffered from asthma alone (4.7 versus 11.7%,

P=0.0001). They concluded that the results strongly suggested a separate genetic factor controlling the development of asthma [69].

Another study done by Lim et al, they had screened the medical literature from 1966 to 2009 and performed a meta-analysis to compare the effect of maternal asthma vs. paternal asthma on susceptibility of asthma in offspring. Consolidating the data from 33 studies, the odds ratio for asthma in children of asthmatic mothers compared with non-asthmatic mothers was significantly increased at 3.04. The corresponding odds ratio for asthma in children of asthmatic fathers was increased at 2.44. When comparing the odds ratios, maternal asthma conferred greater risk of disease than did paternal asthma (3.04 vs. 2.44, p = 0.037). The concluded that in all cases the maternal asthma was a greater risk factor for asthma than paternal asthma [70].

In a study done by Davis et al, they examined the relationship between atopy and wheeze among children and their possible influence on the parental atopy and family size. The prevalence of wheeze was 15.5% in boys, 7.6% in girls and of atopy 19.7% in boys and 8.1% in girls. Of 110 atopic children 70% had no atopic parents whereas 27% had one atopic parent and in about 3% both parents were atopic. The presence of atopy in parents was associated with an increased prevalence of wheeze in boys but not in girls. Prevalence of wheeze among boys was 27.5% if either or both the parents were atopic against 12% with no parental history of atopy ($P<0.05$). They concluded that there was a strong association between parental atopy and wheeze in children [71].

ASTHMA PREVALENCE, FAMILY SIZE AND BIRTH ORDER

The association between the family size and the prevalence of asthma had been a subject of considerable study and remained a matter of controversy. Many studies had found a negative correlation between asthma prevalence and family size. In contrast one

study detected a higher asthma prevalence in larger families while some had found no association between two. Broad implications of the protective mechanism of the sibling were explained in various theories. One of the leading theory, "hygiene hypothesis" had predicated that exposure to bacterial components had protected children from asthma. It is probably through an effect on the relatively immature immune system of the early childhood, thereby preventing the proclivity towards atopy. The theory suggested that the presence of older siblings increased the child's exposure to bacterial burden and as a result, a higher degree of protection was anticipated in younger siblings as they are exposed to more children at home during childhood.

In a study done by Goldberg et al, they examined the relationship of asthma with family size and birth order. Odds ratios for asthma and between birth order and family size, adjusted for each other, were calculated. The prevalence of asthma among males was 8.6% and among girls was 6.9%. The prevalence of asthma was inversely related to the number of children in the family ($P<0.001$). Among subjects who are the only child in the family, the prevalence was 7.3%. The prevalence increased to 8.95% among subjects from families with 3 siblings. Also the prevalence decreased progressively as the number of siblings increased and reached a trough of about 0.58% in families of 15 to 20 siblings. The prevalence of asthma was similar among all birth orders. They concluded that the prevalence of asthma was inversely proportional to number of children in families with four or more children and it is similar to all birth orders. It challenges the hygiene hypothesis as the mechanism of decreased asthma prevalence in large families [72].

In a study done by Bernsen et al, they carried out their study to find out the independent relations of birth order and sibship size with the presence of asthma, allergy

and eczema. 700 families in Netherlands were selected in a retrospective study with index children born during the period from 1988 to 1990. They found out that children with higher birth order had a lower risk of allergy when compared with first-borns. Allergy including eczema also had a significant relation with birth order (P=0.01). For asthma, no clear relationship has been found. A non-significant relationship with sibship size was found for asthma (P=0.06). They had concluded that first born children in small sibship were more at risk than those with larger sibships and hence birth order is inversely related to the risk of allergy independent of the size of sibship [73].

In a study done by Karmaus et al, they reviewed the protective effects of having a higher number of siblings for the risk of atopic eczema, asthma wheezing, hay fever and allergic sensitization by collecting the review of literature from medline since 1965 and identified 53 different studies. Among them 9 of 11 studies had reported an inverse relation with number of siblings for eczema, 21 of 31 studies had reported inverse association for asthma and wheezing and 14 of 16 studies had supported the protective effect of a higher number of siblings for allergic sensitization or IgE reactivity. The study had emphasised a theory that was based exclusively on epidemiological associations. They concluded that the research had not yet answered the question of causal factors explaining the sibling effect and the prevailing 'hygiene hypotheses' had failed to explain the findings adequately [74].

SMOKING RELATED BRONCHIAL ASTHMA

The role of tobacco smoking in the development of bronchial asthma has always remained controversial. Many reviews had been in the view that smoking increases the risk of asthma. But there have been no association found between asthma and smoking.

Some studies had found that there was an increased risk among smoking males but not among females.

Airway inflammation in asthma involves a very complex interaction of cell mediators, cytokines and chemokines. Immune and non-immunologic environmental factors are important triggers of bronchial asthma including cigarette smoking and second-hand smoke. Approximately 25% to 35% of individuals with asthma are current smokers. It has been documented that smoking or exposure to SHS among asthmatics had increased asthma related morbidity and severity of the disease.

In an epidemiological study of bronchial asthma and smoking done by Flodin et al, they compared 79 cases of asthma who were diagnosed between 20 and 65 years of age with 304 randomly drawn population controls of similar age from the same area. The study mainly involved in comparing the questionnaire information on smoking habits, occupational exposures, dwelling conditions, various suspect allergen exposure and atopy. They found that those who had smoked for 3 years or more were at increased risk for bronchial asthma (OR=1.9). The relative risk estimate had not changed even after adjustment by multiple logistic regression for age and gender. They had finally concluded that the exposure to environmental tobacco smoke or passive smoking at work had involve a slightly greater risk in the development of bronchial asthma [75].

In a study done by Siroux et al, they evaluated the role of smoking as a potential risk factor, selection factor and modifying factor of asthma in the Epidemiological study on the Genetics and Environment of Asthma (EGEA). They had analysed 200 adult asthmatics, 265 non-asthmatic controls and 586 relatives of asthmatics and found that less smoking was not associated with asthma in childhood (OR=1.06 in males and 0.98 in females) but smoker asthmatics quit more often than controls (OR=2.20 in males and

1.02 in females). Adult onset asthma was unrelated to ever smoking (OR=1.07 in males and 1.02 in females). In asthmatics, active smoking was associated with asthma severity. No clear pattern regarding the relationship of smoking habits with asthma was observed in first degree relatives. It was concluded that active smoking is not a risk factor for asthma in adulthood, but that smoking increases asthma severity [76].

In a study done by Sapleton et al, they found out that the disease control was poorer in asthmatic smokers than in asthmatic non-smokers. Maternal exposure has been found to have greater impact on asthma and asthmatic children exposed to multiple household smoke were at increased risk. They had concluded that cigarette smoking and second hand smoke in asthmatics had led to detrimental effects in patient outcomes and effectiveness of steroid therapy [77].

Another study done by Verlato et al, they had aimed to study the incidence of asthma as a function of smoking habits in adult population. During their study 145 new cases of asthma were observed with a cumulative incidence of 4.6%. The cumulative incidence of asthma did not significantly differ among never-smokers (4.6%), ex-smokers (5.4%) and current smokers (4.4%) (P=0.641). In a multivariate analysis, the most important risk factor for the onset of asthma was allergic rhinitis (OR=4.0). When compared to never smokers, the risk of asthma onset was slightly increased in ex-smokers (OR=1.28) but not in current smokers (OR=1.01). They concluded that, current smoking was not a risk factor for new onset asthma [78].

Although the occurrence of childhood asthma has been attributed to involuntary exposure to maternal smoking during the *in utero* period and to second hand smoke, few studies had investigated the role of active cigarette smoking on asthma onset during adolescence. In a study done by Gillliand et al, titled regular smoking and asthma

incidence in adolescents, they did a prospective cohort study among 2909 children and followed them annually. They had found that regular smoking was asoociated with increased risk of new onset asthma. Children who had reported smoking 300 or more cigarettes per year had a relative risk of 3.9 for new onset asthma compared with non-smokers. They had concluded that regular smoking had increased the risk for asthma among adolescents especially for non-allergic subjects [79].

ASPIRIN INTOLERENT ASTHMA

Aspirin intolerant asthma, a clinically distinct syndrome, is characterised by precipitation of asthma attacks following the ingestion of aspirin and other non-steroidal anti-inflammatory drugs (NSAIDS). Despite the name relates only aspirin, it has established that the affected persons were cross sensitive to all non-steroidal anti-inflammatory drugs that inhibit cyclo-oxygenase enzymes. Clinical presentation begins in the third or fourth decade and follows a characteristic growth. It is frequently associated with development of chronic nasal congestion, anosmia, rhinorrhoea and nasal polyps. Usually NSAID triggers an acute asthmatic attack which gradually develops into bronchial asthma. Generally these attacks occur within one hour of aspirin ingestion followed by typical presentation of profuse rhinorrhoea, conjunctival irritation and flushing of the head and neck. Only handful of studies have been available worldwide that has provided the estimates of the prevalence of AIA that ranges from 1-2% up to 20%. The exact prevalence of AIA remained uncertain for a long time. Thus a greater understanding of aspirin induced asthma is desirable as there is an increasing trend in over the counter analgesics for minor ailments and their ignorance in relating the asthma with those analgesics.

In a study done by Vally et al, they surveyed three populations to establish the prevalence of AIA among the Australian asthmatics. A total of 1814 asthmatics from hospitals, Asthma Foundation were recruited to the study. They had found that the prevalence of AIA in the hospital and Asthma Foundation cohorts was found to be 10.7% and 10.4% respectively. Univariate analysis in the Asthma Foundation cohort had indicated that AIA was associated with more severe asthma (OR=2.4), nasal polyposis (OR=3.19), atopy (OR=2.96), sulphite sensitivity (OR=3.97) and sensitivity to wine (OR=3.27). They had concluded that prevalence of respiratory symptoms triggered by aspirin/NSAID use was found to be 10-11% in patients with asthma and 2.5% in non-asthmatics [80].

In another study done by Jenkins et al, they reassessed the prevalence of aspirin induced asthma and other issues related to the syndrome. They had restricted the review to respiratory responses to analgesics available without prescription. They had found that the prevalence of aspirin induced asthma was highest when determined by oral provocation testing (adults 21%; children 5%) than by verbal history (adults 3%; children 2%). Patients with aspirin induced asthma also showed cross sensitivity to doses of over the counter non-steroidal anti-inflammatory drugs but the incidence of cross reactivity to paracetamol was found to be only 7%. They concluded that aspirin induced asthma in adults was more prevalent than previously suggested and an oral provocation test should be performed when there is a clinical necessity to use aspirin or a non-steroidal anti-inflammatory drug and there is uncertainty about its safety [81].

MATERIALS AND METHODS

Primary Objectives:

a. To study the epidemiology of bronchial asthma phenotypes in urban population of North Chennai.

b. To assess the influence of environmental exposure on the prevalence and expressions of various phenotypes of bronchial asthma.

Secondary Objectives:

The population of patients with individual asthma phenotypes are expressed in percentage.

The patients with significant environmental exposure expressed as percentage were further correlated with individual phenotypes and its significance is calculated.

Sample Size:

Sample size-250

Allowable alpha error-5%, confidence level of 95% and desired accuracy of 6%

Subject selection:

Patients attending the thoracic medicine outpatient clinic in Government Thiruvotteeswarar Hospital of Thoracic Medicine (GTHTM) and Government Kilpauk Medical College (KMC) with symptoms suggestive of bronchial asthma are selected.

A patient is suspected to have asthma if he or she has any one of the following symptoms as advocated by Global Initiative for Asthma (GINA) guidelines 2015.

- Wheeze, Shortness of breath (dyspnea), Chest tightness or Cough
- Variable expiratory airflow limitation

- Symptoms often worse at night or in the early morning
- Symptoms vary over time and in intensity
- Symptoms are triggered by viral infections, exercise, and allergen exposure, changes in weather or exposure to irritants.

INCLUSION CRITERIA

- Patients aged more than 6 years but less than 60 years
- Patients with Dyspnea, Wheeze, Chest tightness or cough
- Patients with significant post-bronchodilator reversibility
- Patient with no significant lung lesions

EXCLUSION CRITERIA

- Patients with Bronchiectasis, chronic bronchitis and emphysema
- Patients with acute exacerbation of asthma
- Patients who cannot perform spirometry
- Patients with no significant post bronchodilator reversibility

STUDY CENTRES:

The study was conducted at two tertiary care institutes that have outpatient clinics for patients with respiratory illnesses.

- Government Kilpauk Medical College, Chennai.
- Government Thiruvotteeswarar Hospital of Thoracic Medicine, Chennai.

STUDY DESIGN:

- The study was a descriptive cross sectional study
- No specific intervention was carried out
- No controls had been used in the study.

DATA COLLECTION:

The data of each patient was collected on a proforma specially designed for this study.

- Demographic data
- Occupation
- Socio-economic status
- Body Mass Index
- Birth order
- History of Allergy/Atopy
- Presence of disease in family members
- Exposure to farm products in childhood
- Exposure to allergens, chemicals at workplace
- History of sensitivity to Aspirin/NSAIDs
- Exposure to Environmental Tobacco Smoke
- Food habits
- Family size
- History of Recurrent Respiratory Tract infections
- History of Gastro-esophageal reflux disorder
- Changes in climatic conditions
- History of Stress and Emotional conditions
- Knowledge about the disease, diagnosis and mode of treatment
- Sputum cytology
- Absolute Eosinophil Count

DEMOGRAPHIC DATA

Demography (*demos-people; graph-description*) is the statistical study of populations which is very important in analyzing the dynamic living population. It encompasses the study of the size, structure and distribution of these populations and spatial and temporal changes in them.

In my study the demographic details collected include the name, age and gender of the patient and their habitat and level of education.

SOCIO-ECONOMIC STATUS

Socioeconomic status (SES) is an economic and sociological combined total measure of a person's work experience and of an individual's or family's economic and social position in relation to others, based on income, education, and occupation.

Socioeconomic status is typically broken into three categories namely high SES, middle SES, and low SES.

A composite measure that typically incorporates economic, social, and work status.

- Economic status is measured by income.
- Social status is measured by education, and
- Work status is measured by occupation.
- Each status is considered an indicator.

These three indicators are related but do not overlap

Socio-economic Status Scales in India:

- Udai Pareek and G. Trivedi (1964)
- Kuppuswamy scale 1962
- B G Prasad classification proposed in the year 1961

The most widely used scale for urban population was devised by Kuppuswamy in 1976. It is a composite score of education and occupation of the head of the family along with monthly income of the family, which yields a score of 3-29. This scale classifies the study populations into high, middle and low SES.

(A)	Education	Score
1.	Profession or honours	7
2.	Graduate or post graduate	6
3.	Intermediate or post high school diploma	5
4.	High school certificate	4
5.	Middle school certificate	3
6.	Primary school certificate	2
7.	Illiterate	1
(B)	Occupation	Score
1.	Profession	10
2.	Semi-profession	6
3.	Clerical, shop-owner, farmer	5
4.	Skilled worker	4
5.	Semi-skilled worker	3
6.	Unskilled worker	2
7.	Unemployed	1
(C)	Family income per month (in Rs, (1976)	Score
1.	−2000	12
2.	1000-1999	10
3.	750-999	6
4.	500-749	4
5.	300-499	3
6.	101-299	2
7.	−100	1
Total score		**Socioeconomic class**
26-29		Upper(I)
16-25	Middle	Upper middle (II)
11-15		Lower middle (III)
5-10	Lower	Upper lower (IV)
<5		Lower(V)

Fig 15: Modified Kuppuswamy Scale

BODY MASS INDEX

The body mass index (BMI) or Quetelet index is a value derived from the mass (weight) and height of an individual. The BMI is defined as the body mass divided by the square of the body height, and is universally expressed in units of kg/m^2.

The BMI is an attempt to quantify the amount of tissue mass (muscle, fat, and bone) in an individual, and then categorize that person as *underweight*, *normal weight*, *overweight*, or *obese* based on that value.

Patient's body weight is measured to nearest 0.1 kg with subjects in light clothing and patients' height is measured by asking them to stand barefoot with their backs and heels touching a vertical bar to the nearest 0.5 cm and BMI is calculated. Drawback of BMI is it does not assess changes in body composition.

BMI	NUTRITIONAL STATUS
<18.5	Underweight
18.5-24.9	Normal
25-29.9	Overweight
>30	Obese

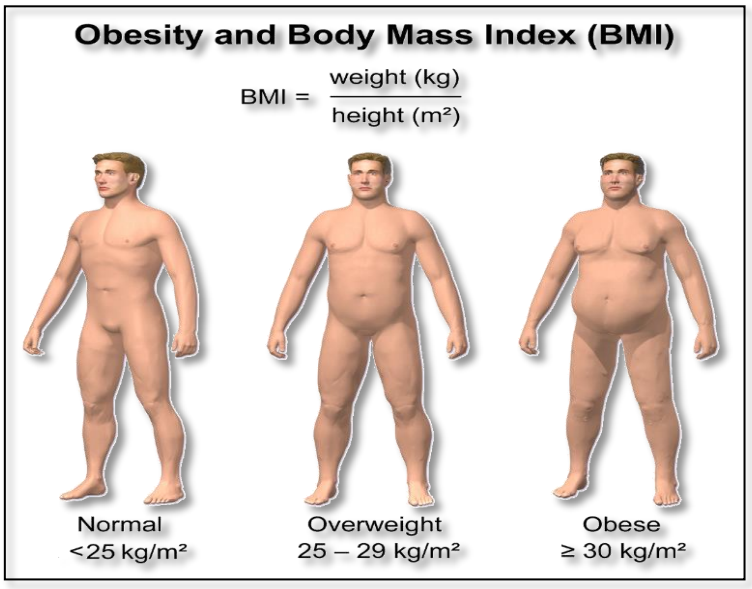

Fig. 16: Pictorial representation of BMI

BIRTH ORDER

Birth order refers to the order a child is born, for example first born, second born etc. Birth order is often believed to have a profound and lasting effect on psychological development.

Asthmatic patients who are enrolled in the study are enquired about their birth order and details are recorded.

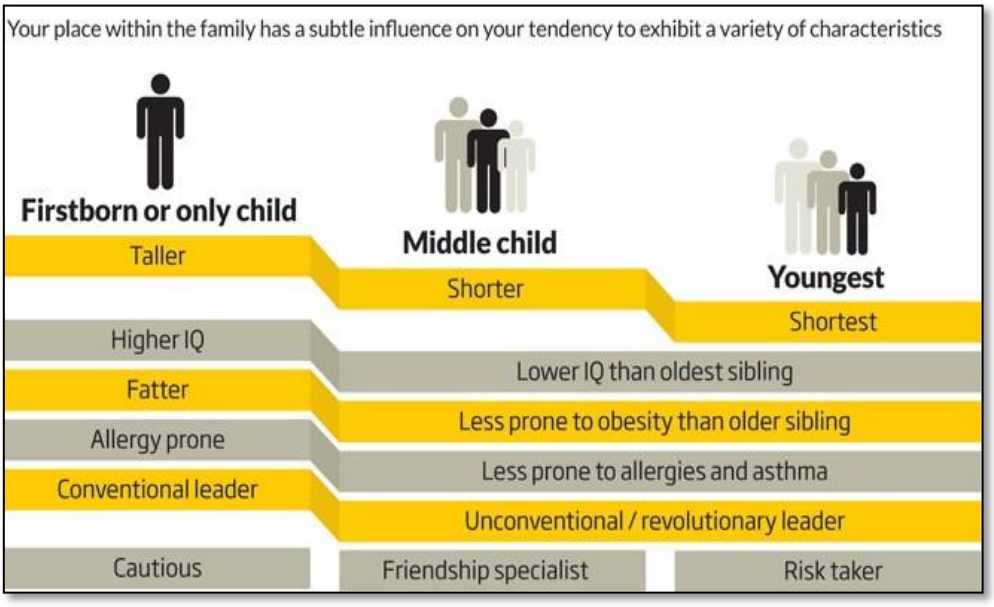

Fig 17. Birth order and susceptibility to atopy

EXPOSURE TO ALLERGENS

An allergen is a type of antigen that produces an abnormally vigorous immune response in which the immune system fights off a perceived threat that would otherwise be harmless to the body. An allergen is an antigen capable of stimulating a type-I hypersensitivity reaction in atopic individuals through Immunoglobulin E (IgE) responses.

Most common extrinsic allergens which is responsible for triggering asthma includes pollen, dust mites, moulds, animal dander and cockroaches.

Subjects are asked about their specific symptom exacerbation after exposing to any of these triggering agents and the specific allergen to which they are sensitized are recorded.

EXPOSURE TO TOBACCO SMOKE AND SECOND HAND SMOKING

Tobacco smoke is one of the most common asthma triggers. Tobacco smoke—including secondhand smoke—is unhealthy for everyone, especially people with asthma. Secondhand smoke is a mixture of gases and fine particles that includes,

- Smoke from a burning cigarette, cigar, or pipe tip
- Smoke that has been exhaled (breathed out) by someone who smokes

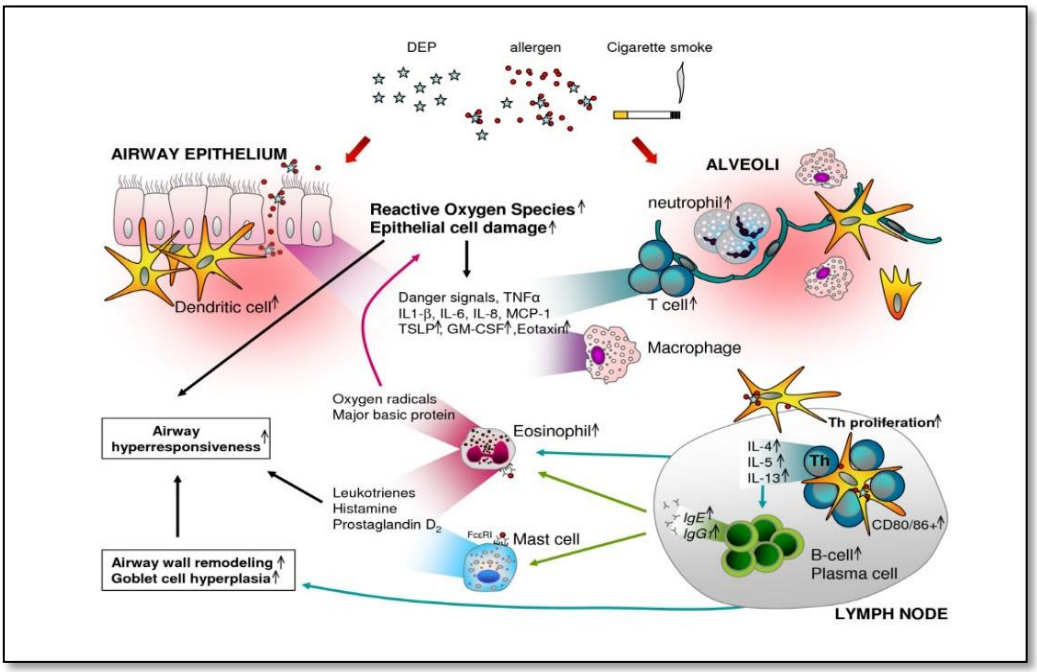

Fig 18. Role of smoking in asthma

Male patients who are asthmatics and included in the study are enquired about their smoking history and female asthmatics and children are enquired about the exposure to passive or second hand smoke in their living room and details were recorded.

STATISTICAL ANALYSIS

Statistical analysis was done using the Microsoft Excel and SPSS software with the help of a statistician. P value is used to assess the significance of correlation between variables.

Pearson correlation is used to assess the strength of correlation between variables

Pearson correlation:

 > 0.5 - Strong correlation

 0.3 to 0.5 - Moderate correlation

 <0.3 - Weak correlation

Chi-square Test:

Chi-square test is performed between two groups and its statistical significance is calculated. The chi-square (χ^2) test of independence is used to test for a statistically significant relationship between two categorical variables. The term "degrees of freedom" is used to refer to the size of the contingency table on which the value of the Chi Square statistic has been computed

$$\chi^2 = \sum \frac{(\text{observed} - \text{expected})^2}{\text{expected}}$$

P value is calculated using Excel CHITEST function:

If P value ≤ 0.05 → statistically significant

If P value > 0.05 → statistically insignificant

RESULTS

AGE DISTRIBUTION OF THE STUDY SUBJECTS:

About 65% of the study subjects with asthma were in the age group of 21 to 40 years. Mean age (± S.D): 32.63 (9.93) years, Minimum: 10 years, Maximum: 56 years.

Table 1: Age distribution of the study subjects (n=250)

Age group	Frequency	Percent
10-20 years	31	12.4
21-30 years	84	33.6
31-40 years	78	31.2
41-50 years	50	20.0
51-60 years	7	2.8
Total	250	100.0

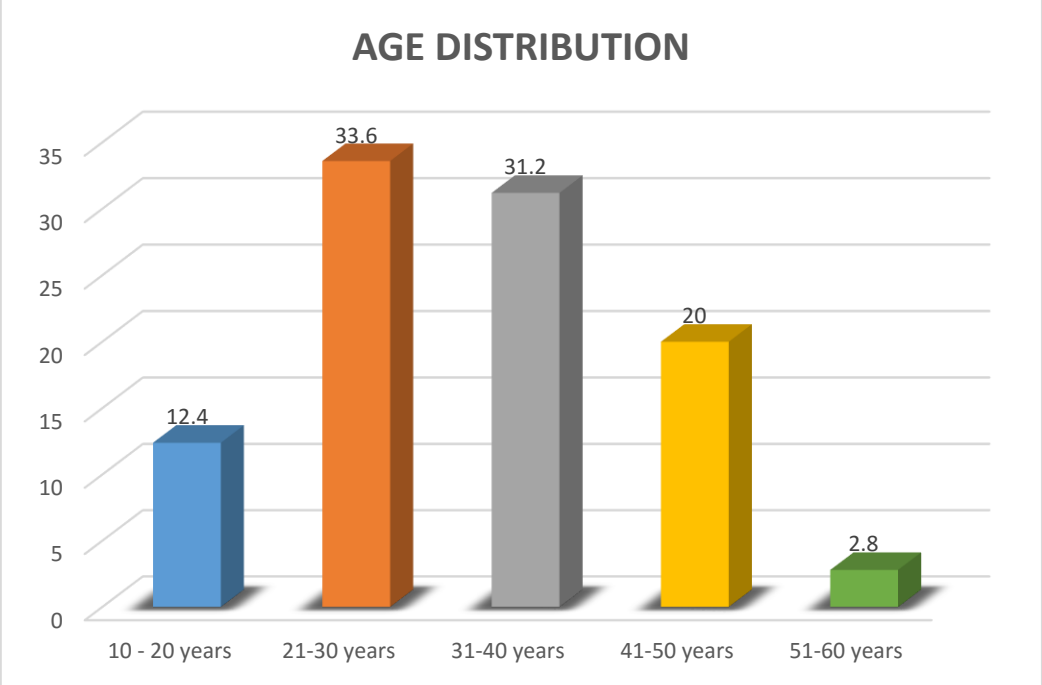

Fig 19: Age distribution of the study subjects (n=250)

GENDER DISTRIBUTION OF THE STUDY SUBJECTS:

About 54% of the study subjects with asthma were females and 46% of the study population were males.

Table 2: Gender distribution of the study subjects (n=250)

Gender	Frequency	Percent
Male	114	45.6
Female	136	54.4
Total	250	100.0

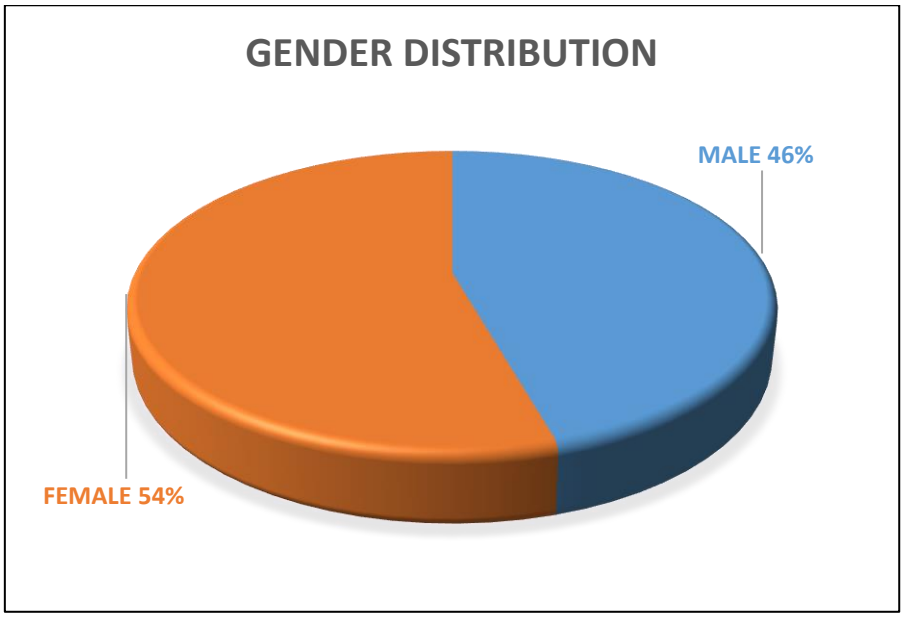

FIG 20: Gender distribution of the study subjects (N=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO LEVEL OF EDUCATION:

About 64.8% of the study subjects were illiterates and 35.2% were literates. The reason of major proportion of the study population being illiterates is due to the poor quality of living of the people in north Chennai from where the majority of patients are coming to our OPD.

Table 3: Distribution of the study subjects according to Level of education (n=250)

Education	Frequency	Percent
Illiterate	162	64.8
Literate	88	35.2
Total	250	100.0

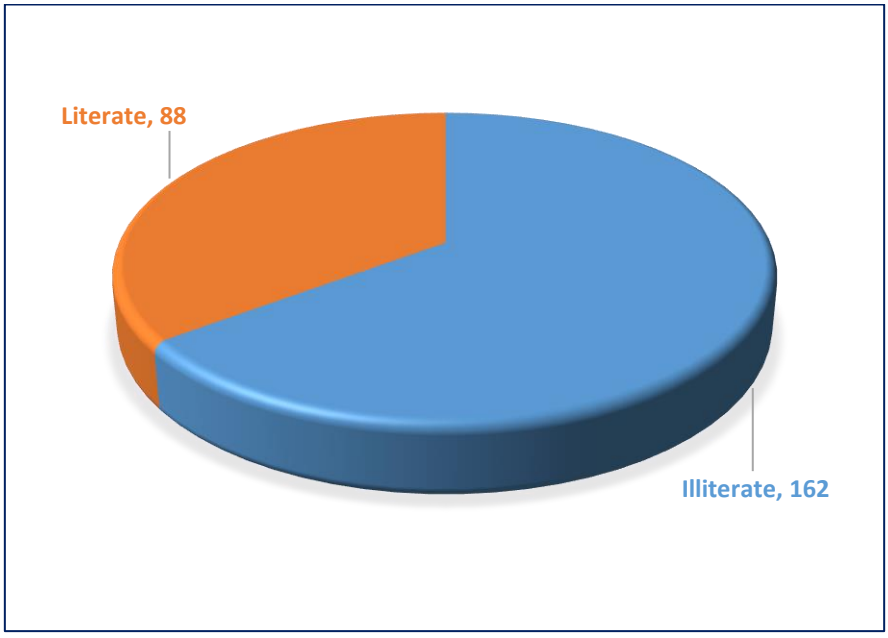

Fig 21: Distribution of the study subjects according to level of education (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SOCIO-ECONOMIC STATUS:

About 64% of the study subjects belonged to low socio-economic status. It also indirectly reflects the poor quality of living of the people of north Chennai who in majority of the proportion were lacking good quality of education and most of them are daily wagers.

Table 4: Distribution of the study subjects according to Socio-economic status (n=250)

Socio-economic status	Frequency	Percent
Low	160	64.0
Middle	90	36.0
Total	250	100.0

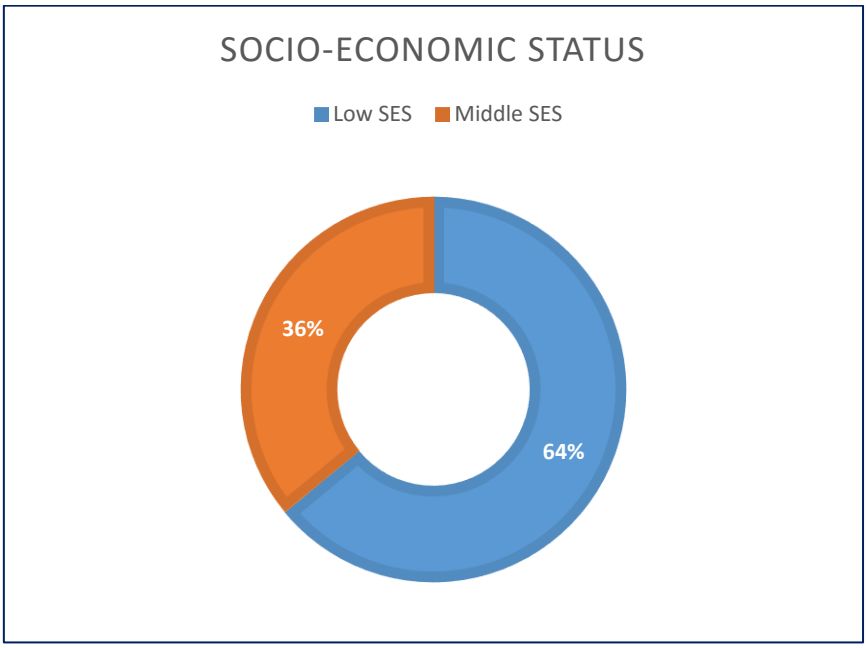

Fig 22: Distribution of the study subjects according to Socio-economic status (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO BODY MASS INDEX:

About 34% of the study subjects were overweight and 7% were obese. Among the obese, majority of the patients were of females and older age group.

Table 5: Distribution of the study subjects according to Body mass index (n=250)

Body mass index	Frequency	Percent
Normal	145	58.0
Overweight	87	34.8
Obese	18	7.2
Total	250	100.0

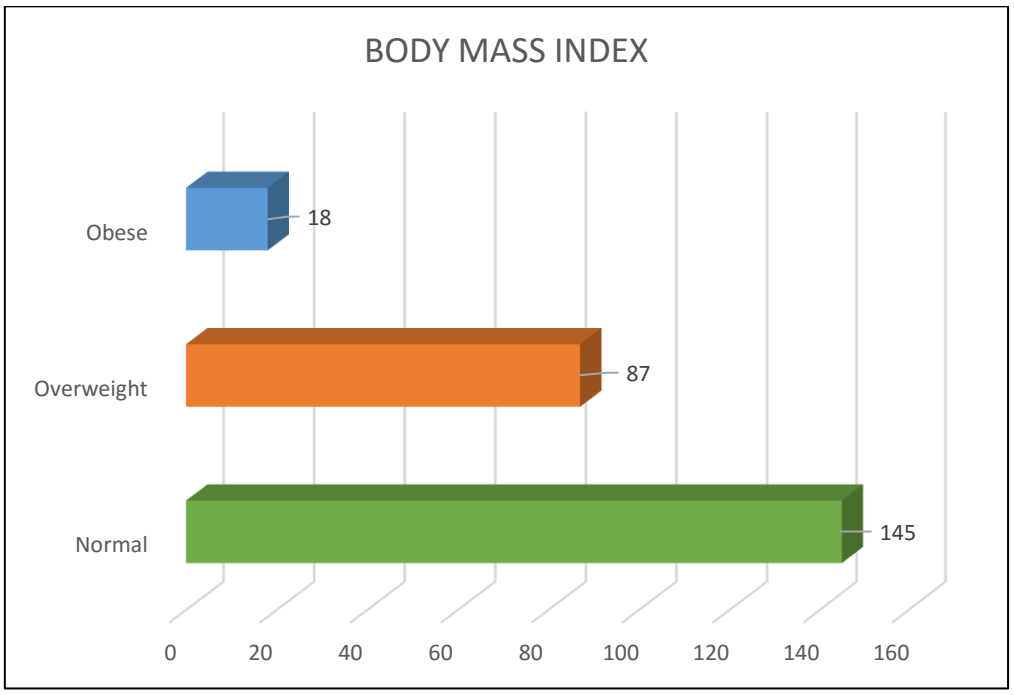

Fig 23: Distribution of the study subjects according to Body mass index (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO BIRTH ORDER:

About 65% of the study subjects belonged to first birth order. Majority of the patients were of the lower order i.e. first born child in their family and the remaining were of higher order i.e. 2nd or successive child in their family.

Table 6: Distribution of the study subjects according to birth order (n=250)

Birth order	Frequency	Percent
1st order	162	64.8
2nd order and above	88	35.2
Total	250	100.0

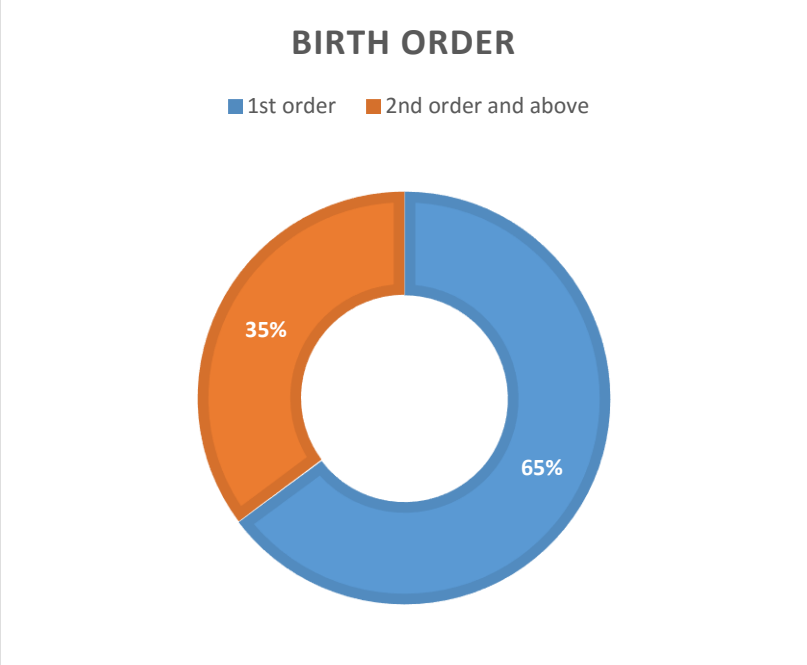

Fig 24: Distribution of the study subjects according to birth order (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO AGE OF ONSET OF ASTHMA:

About 76% of the study subjects had early age of onset of asthma. Major proportion of the study population had their asthma symptoms from early childhood and has recurrent exacerbations when they are prone to triggering factors

Table 7: Distribution of the study subjects according to Age of onset of asthma (n=250)

Age of onset	Frequency	Percent
Early onset	191	76.4
Late onset	59	23.6
Total	250	100.0

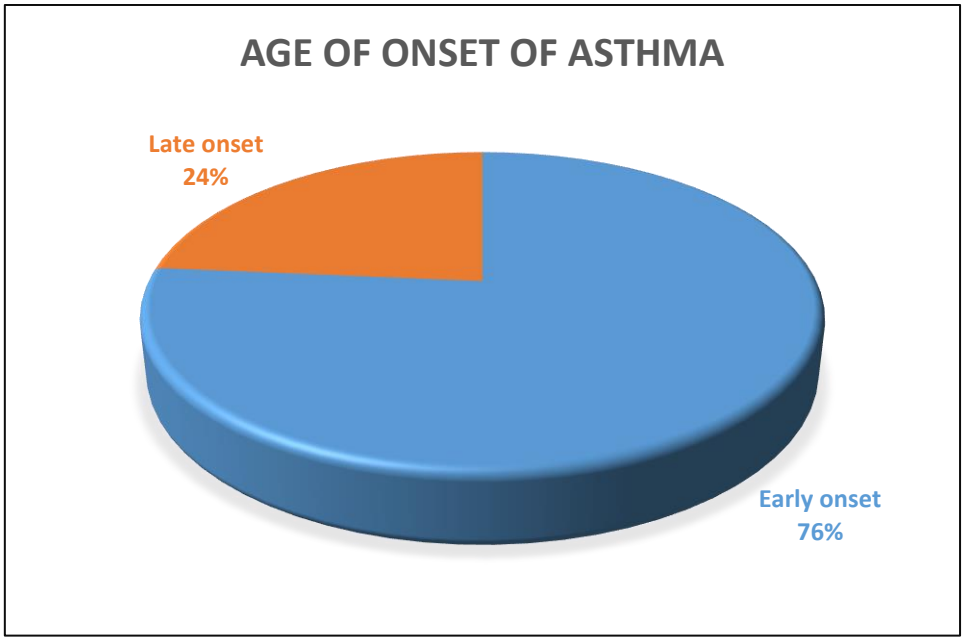

Fig 25: Distribution of the study subjects according to Age of onset of asthma (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF SMOKING:

About 25% of the study subjects were smokers and 20.8% were non-smokers. 54% of the study subjects were females and none of them were smokers and it is mainly due to the absence of influence of the westernized culture in our country.

Table 8: Distribution of the study subjects according to History of smoking (n=250)

History of smoking	Frequency	Percent
Yes	62	24.8
No	52	20.8
Not applicable	136	54.4
Total	250	100.0

Table 9: Distribution of the study subjects according to exposure to Passive smoking (n=250)

Exposure to passive smoking	Frequency	Percent
Yes	111	44.4
No	60	24.0
Not applicable	79	31.6
Total	250	100.0

About 44% of the study subjects were exposed to passive smoking. These 44% of the patients comprises of mostly females and children who are constantly exposed to passive or second hand smoking in their households and they give positive history of worsening of symptoms when they are exposed to these kind of triggering factors.

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF ATOPY:

About 52.8% of the study subjects had history of atopy in some forms. There is relatively a high proportion of patients who gives a positive history of atopy.

Table 10: Distribution of the study subjects according to History of atopy (n=250)

History of atopy	Frequency	Percent
Yes	132	52.8
No	118	47.2
Total	250	100.0

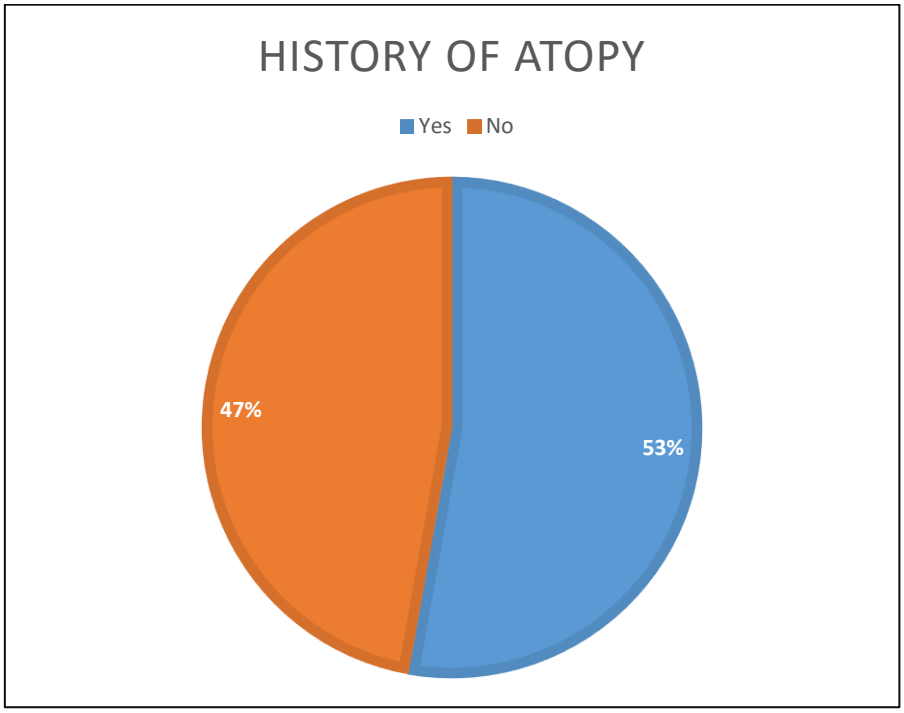

Fig 26: Distribution of the study subjects according to History of atopy (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO FAMILY HISTORY OF ASTHMA (HEREDITARY):

About 52% of the study subjects had family history of asthma (hereditary). There is relatively a high proportion of patients who gave a positive history of presence of asthma in their family members.

Table 11: Distribution of the study subjects according to Family history of asthma (Hereditary) (n=250)

Family history	Frequency	Percent
Yes	129	51.6
No	121	48.4
Total	250	100.0

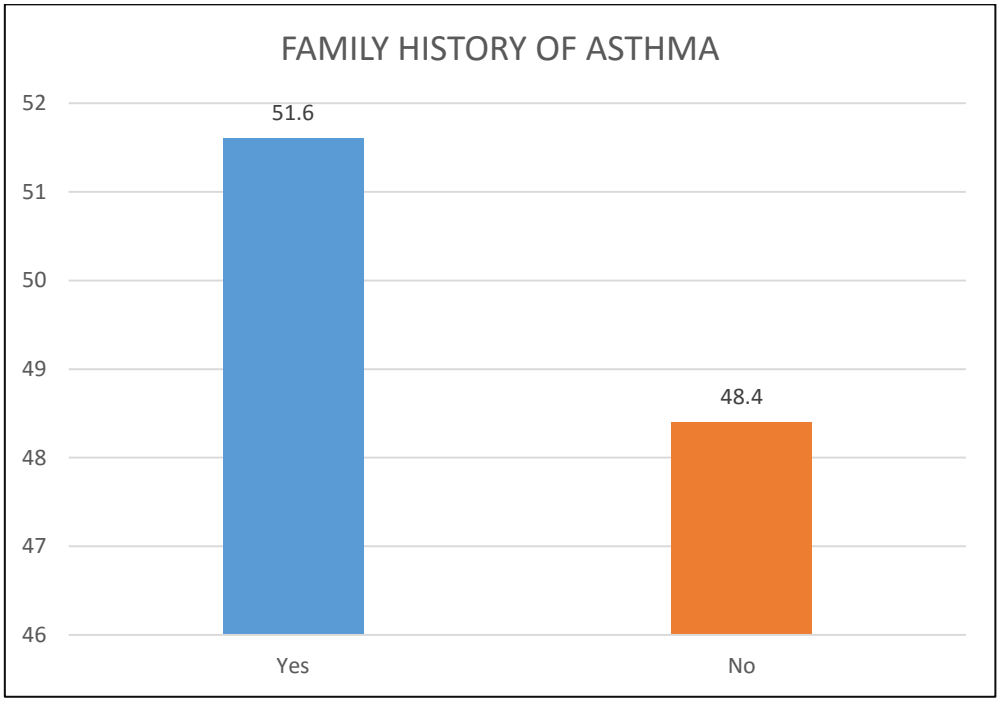

Fig 27: Distribution of the study subjects according to Family history of asthma (hereditary) (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HEREDITARY PATTERN:

Out of 129 subjects who gave positive history of asthma in their family members, about 30% of the study subjects inherited asthma from their mother while 16% from their father and a significant proportion of subjects comprising about 5% of the patients had given positive history of asthma in both the parents.

Table 12: Distribution of the study subjects according to hereditary pattern

Hereditary pattern	Frequency	Percent
Father	41	16.4
Mother	76	30.4
Both Father and Mother	12	4.8
Not applicable	121	48.4
Total	250	100.0

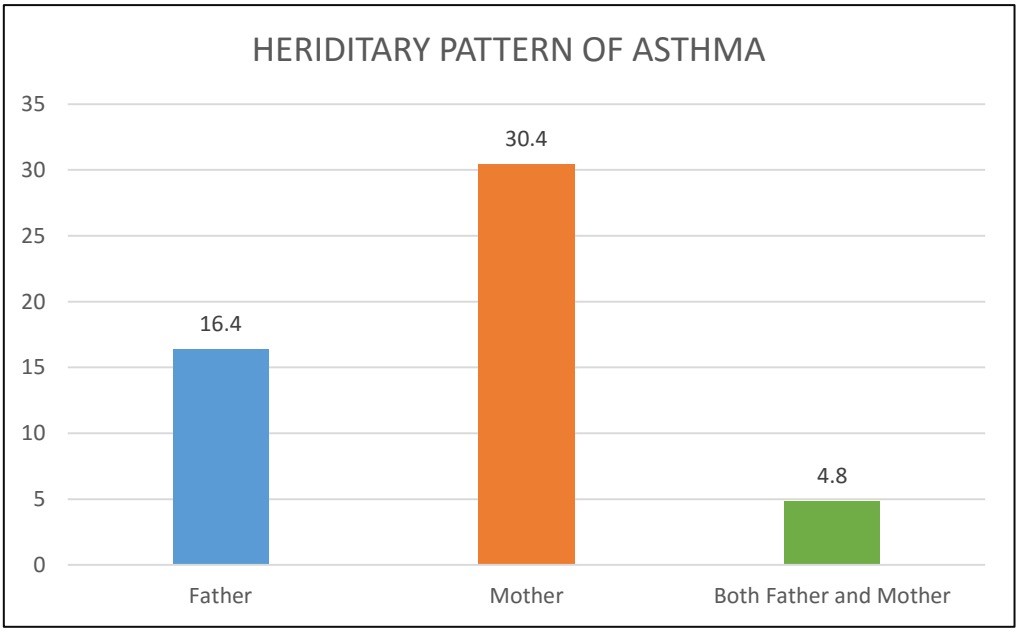

Fig 28: Distribution of the study subjects according to hereditary pattern (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO EXPOSURE TO ALLERGENS:

Out of 132 subjects who gave history of atopy about 20.8% of the study subjects were exposed to food allergen while 13% and 12% were exposed to animals and environmental dust and a significant proportion of subjects constituting 6% were allergic to pollen dust.

Table 13: distribution of the study subjects according to exposure to allergens

Allergen	Frequency	Percent
Pollen Dust	16	6.4
Environmental dust	31	12.4
Food Products	52	20.8
Animals	33	13.2
Nil	118	47.2
Total	250	100.0

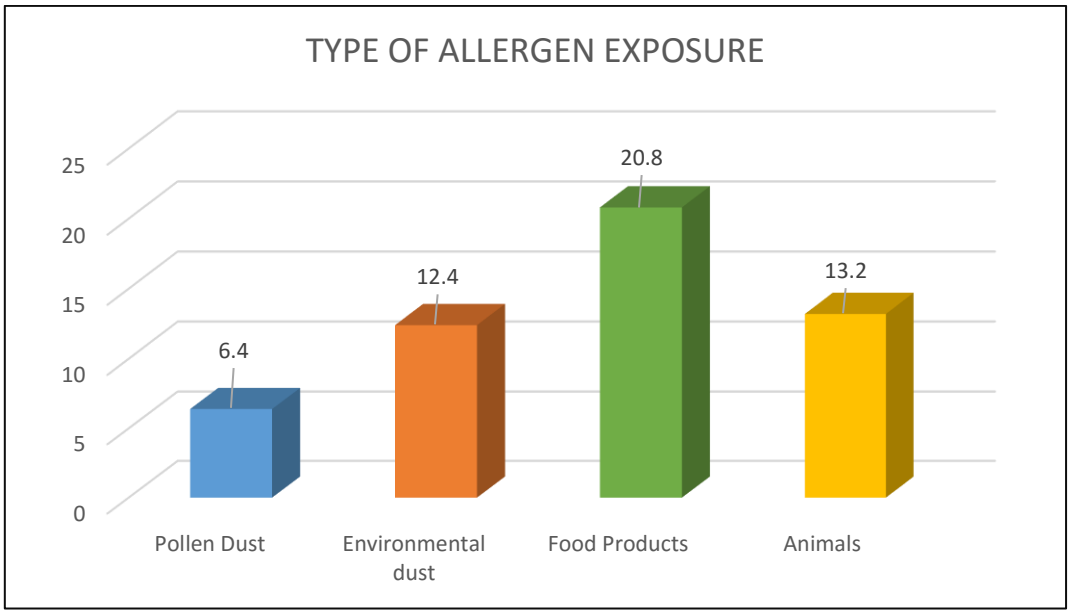

Fig 29: distribution of the study subjects according to exposure to allergens

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF EXPOSURE TO ASPIRIN/NSAIDs:

About 6% of the study subjects had exposure to aspirin. These proportion of patients gave positive history of exacerbation of asthma symptoms when they had ingestion of aspirin or NSAIDs for some form of illness.

Table 14: Distribution of the study subjects according to history of exposure to aspirin/NSAIDs (n=250)

Exposure to aspirin/NSAIDs	Frequency	Percent
Yes	16	6.4
No	234	93.6
Total	250	100.0

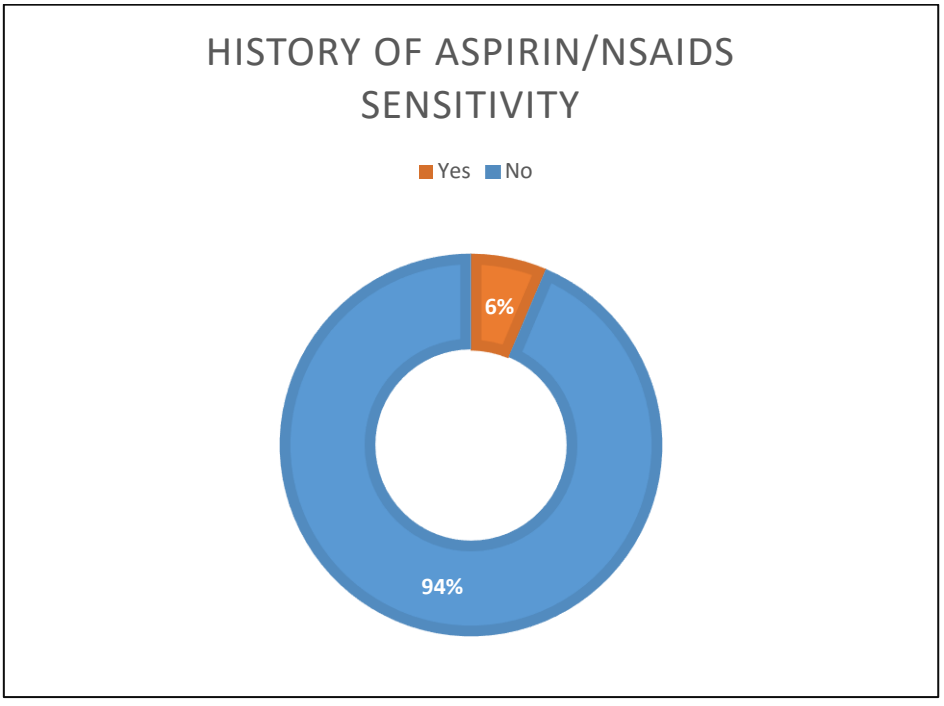

Fig 30: Distribution of the study subjects according to history of exposure to aspirin/NSAIDs (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO FAMILY SIZE:

About 63% of the study subjects belonged to small family size. Family size plays a role in the prevalence of asthma and in our study the prevalence of asthma is inversely proportional to the family size.

Table 15: Distribution of the study subjects according to Family size (n=250)

Family size	Frequency	Percent
Small	158	63.2
Large	92	36.8
Total	250	100.0

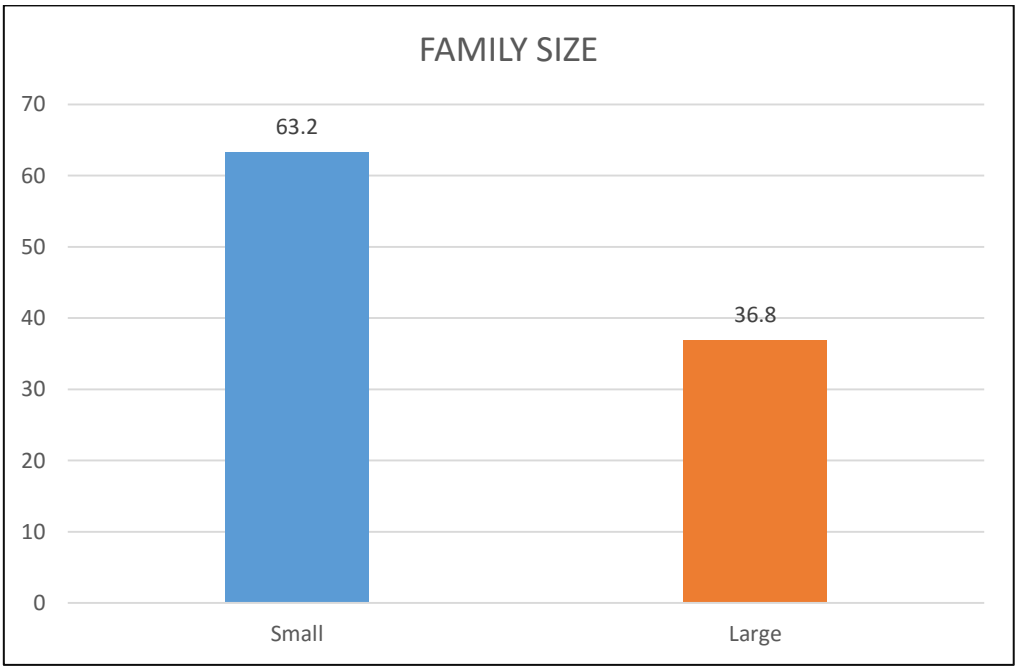

Fig 31: Distribution of the study subjects according to Family size (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF RECURRENT RESPIRATORY INFECTIONS (RRTI):

About 60% of the study subjects had history of recurrent respiratory infections. Majority of the patients gives positive history to the presence of recurrent respiratory tract infections which exacerbates their symptoms.

Table 16: Distribution of the study subjects according to history of recurrent respiratory infections (RRTI) (n=250)

H/o RRTI	Frequency	Percent
Yes	151	60.4
No	99	39.6
Total	250	100.0

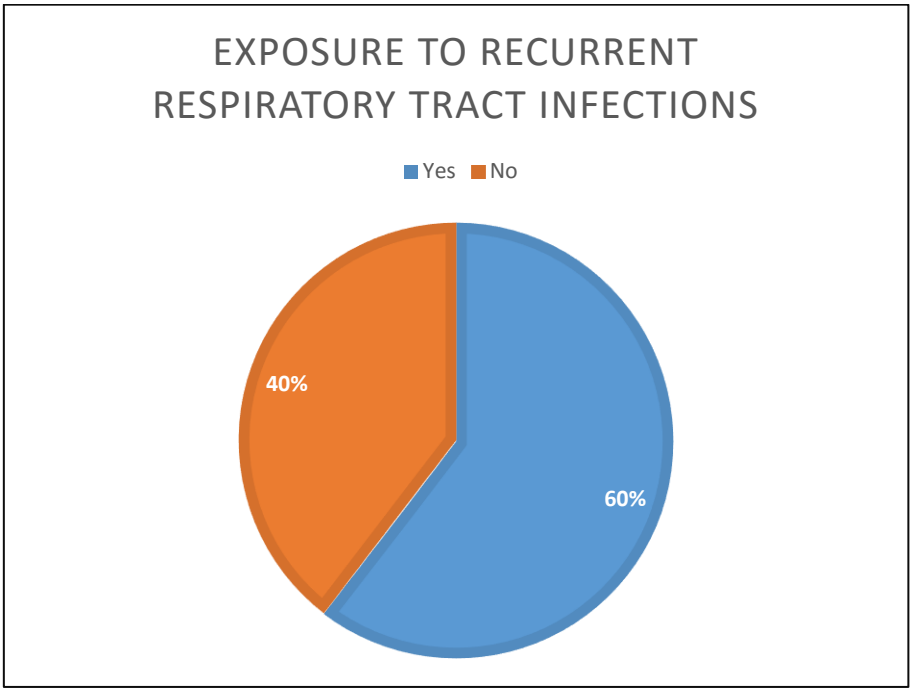

Fig 32: Distribution of the study subjects according to history of recurrent respiratory infections (RRTI) (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF GASTRO-ESOPHAGEAL REFLUX DISEASE (GERD):

About 19% of the study subjects had history of gastro-oesophageal reflux disease. Majority of these subjects were females and are obese which shows the impact of GERD in asthma provocation.

Table 17: Distribution of the study subjects according to history of gastro-oesophageal reflux disease (GERD) (n=250)

H/O GERD	Frequency	Percent
Yes	47	18.8
No	203	81.2
Total	250	100.0

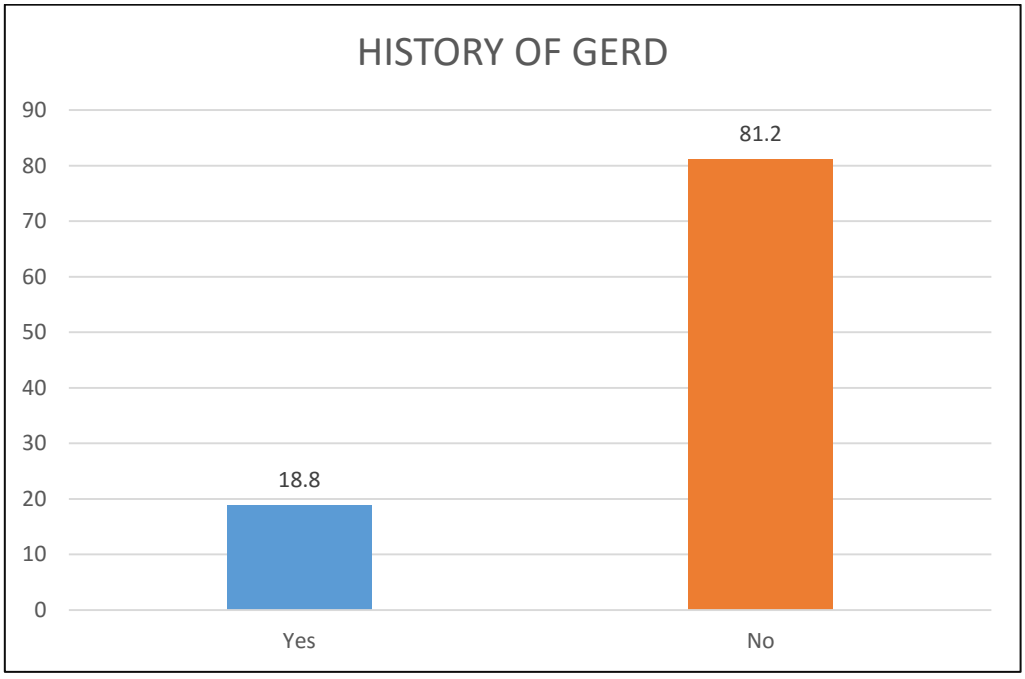

Fig 33: Distribution of the study subjects according to history of gastro-oesophageal reflux disease (GERD) (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SUSCEPTIBILITY TO WEATHER CHANGE:

About 70% of the study subjects were susceptible to develop asthma due to weather change. Weather changes play an important role in exacerbation of asthma symptoms and it is evident from this study very well.

Table 18: Distribution of the study subjects according to susceptibility to Weather change (n=250)

Susceptibility to weather change	Frequency	Percent
Yes	176	70.4
No	74	29.6
Total	250	100.0

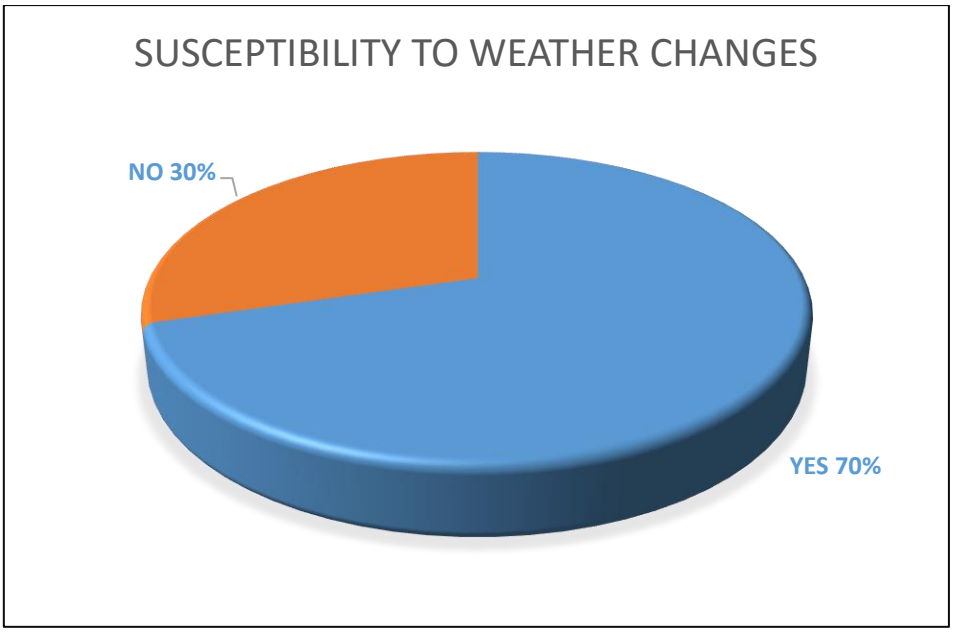

Fig 34: Distribution of the study subjects according to susceptibility to Weather change (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SUSCEPTIBILITY TO STRESS TO DEVELOP ASTHMA:

About 30% of the study subjects were susceptible to develop asthma due to stress/emotional conditions.

Table 19: Distribution of the study subjects according to susceptibility to Stress to develop asthma (n=250)

Susceptibility to stress	Frequency	Percent
Yes	72	28.8
No	178	71.2
Total	250	100.0

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO EXERCISE-INDUCED ASTHMA:

About 30% of the study subjects had exercise-induced asthma. They had developed exacerbation of asthma symptoms in some form of exertion in their day-to-day activities.

Table 20: Distribution of the study subjects according to Exercise-induced asthma (n=250)

Exercise-induced asthma	Frequency	Percent
Yes	74	29.6
No	176	70.4
Total	250	100.0

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO KNOWLEDGE ABOUT THE DIAGNOSIS:

About 77% of the study subjects had knowledge about diagnosis. Majority of the patients were aware of their nature of disease and how it had been diagnosed.

Table 21: Distribution of the study subjects according to Knowledge about the diagnosis (n=250)

Knowledge about diagnosis	Frequency	Percent
Yes	192	76.8
No	58	23.2
Total	250	100.0

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO KNOWLEDGE ABOUT THE MODE OF TREATMENT:

All the study subjects had knowledge about asthma. Almost all the patients were well aware of the mode of treatment they are undergoing.

Table 22: Distribution of the study subjects according to Knowledge about the mode of treatment (n=250)

Knowledge about asthma	Frequency	Percent
Yes	250	100.0
No	0	0
Total	250	100.0

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO MODE OF TREATMENT:

In our study majority of the subjects 205(82%) were on both oral drugs and inhalational drugs and remaining 45 subjects were taking only oral drugs.

Table 23: Distribution of the study subjects according to Mode of treatment (n=250)

Mode of treatment	Frequency	Percent
Oral drugs only	45	18
Oral drugs+Inhalational (LABA+ICS)	205	82
Total	250	100.0

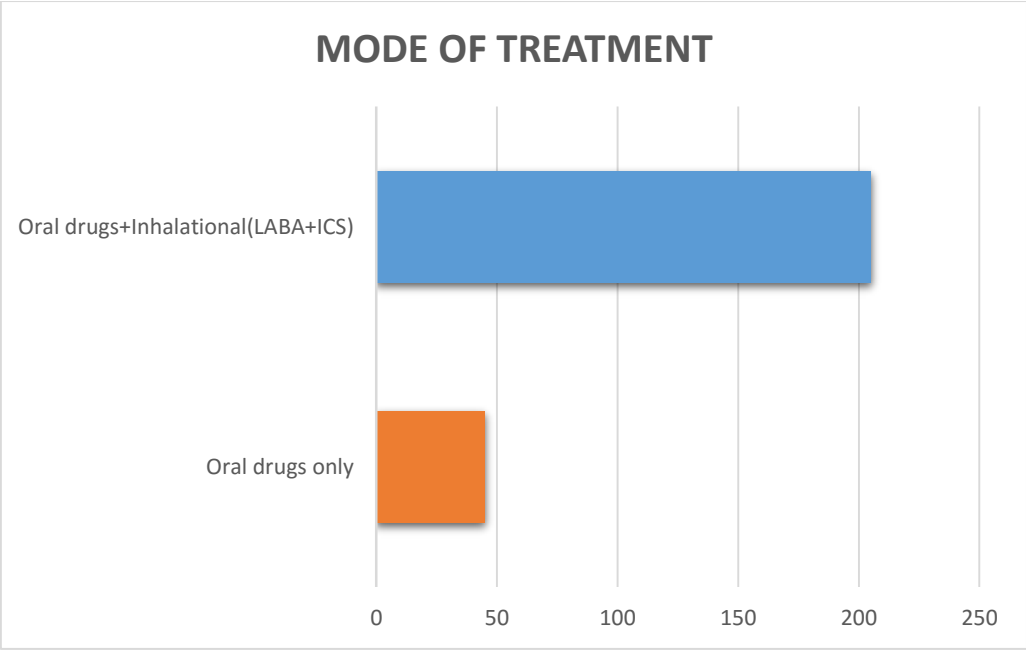

Fig 35: Distribution of the study subjects according to Mode of treatment (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SYMPTOMATIC CONTROL:

About 75% of the study subjects had symptomatic control. Among the subjects who were undergoing various methods of treatment as out patients, majority of the patients had optimal control of symptoms.

Table 24: Distribution of the study subjects according to Symptomatic control (n=250)

Symptomatic control	Frequency	Percent
Yes	188	75.2
No	62	24.8
Total	250	100.0

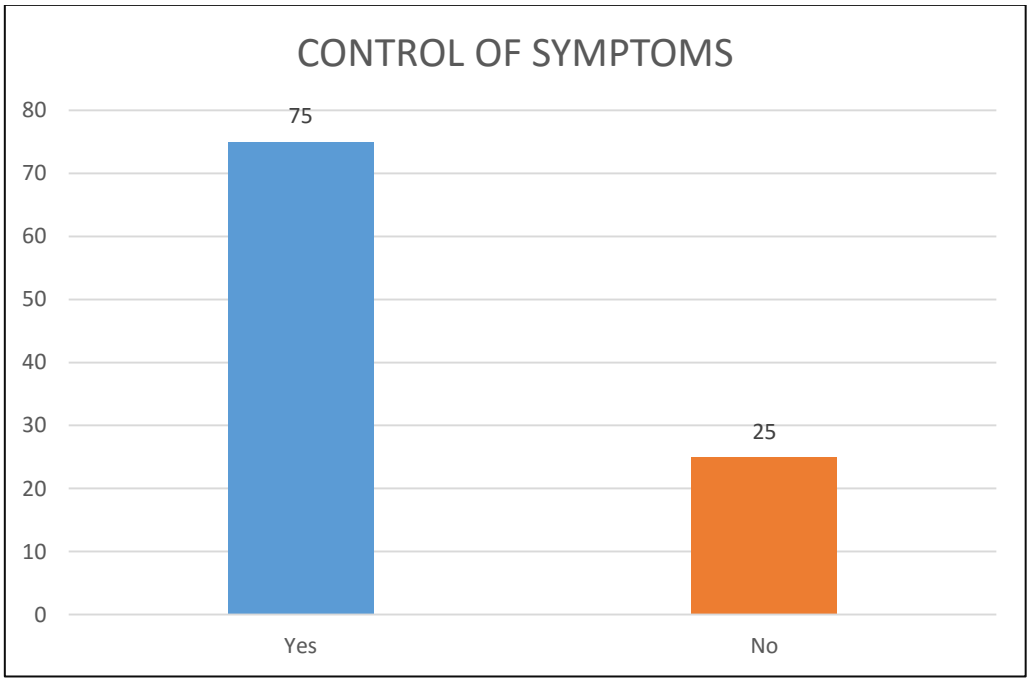

Fig 36: Distribution of the study subjects according to Symptomatic control (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO ABSOLUTE EOSINOPHILIC COUNT:

About 72% of the study subjects had elevated absolute eosinophilic count. This clearly reflects major proportion of the prevalence of atopy and allergic type asthma among the individuals living in urban population.

Table 25: Distribution of the study subjects according to Absolute eosinophilic count (AES) (n=250)

AEC	Frequency	Percent
Normal	69	27.6
Elevated (>350)	181	72.4
Total	250	100.0

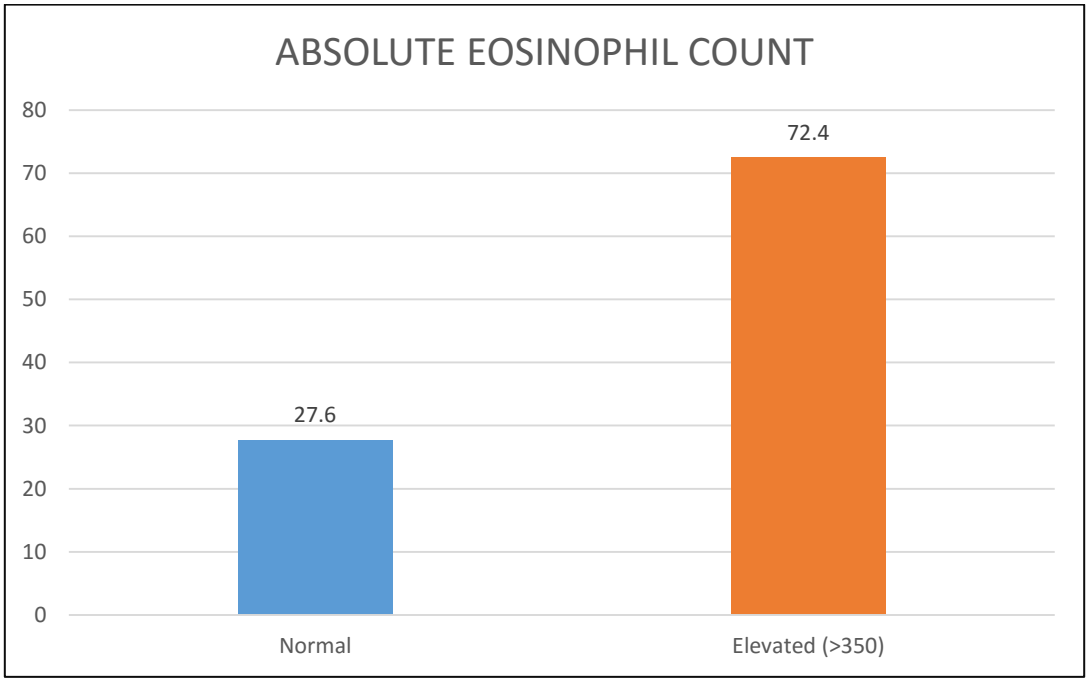

Fig 37: Distribution of the study subjects according to Absolute eosinophilic count (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SPUTUM CYTOLOGY:

About 73% of the study subjects had eosinophilic cytology while 19% and roughly 8% of the study subjects had neutrophilic and mixed neutrophilic and eosinophilic type, respectively.

Table 26: Distribution of the study subjects according to Sputum cytology (n=250)

Sputum cytology	Frequency	Percent
Eosinophilic	182	72.8
Neutrophilic	47	18.8
Mixed neutrophilic and eosinophilic	19	7.6
Pauci-granulocytic	2	0.8
Total	250	100.0

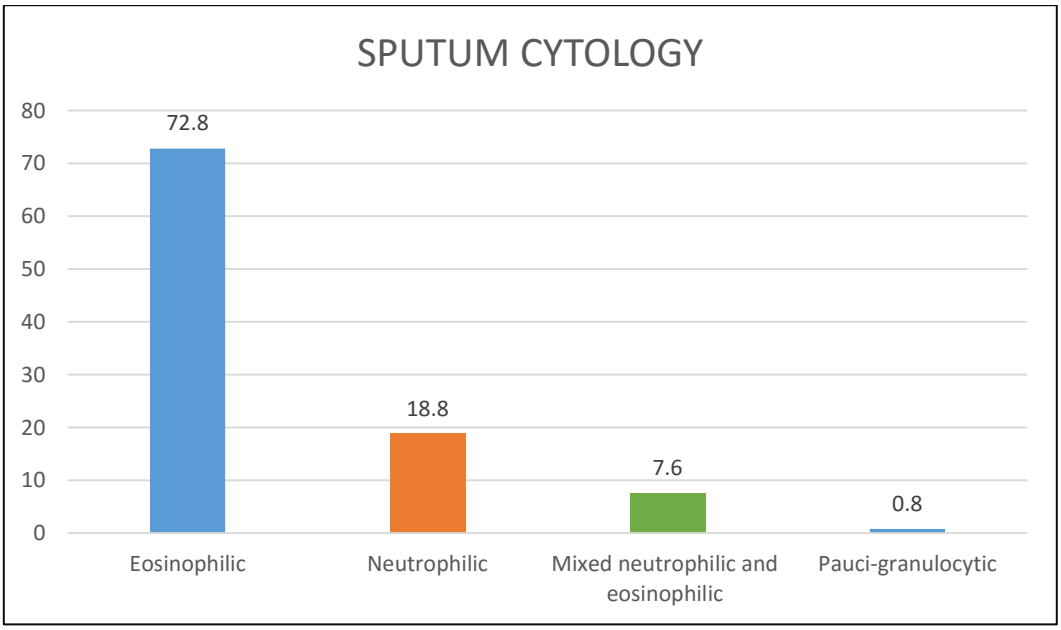

Fig 38: Distribution of the study subjects according to Sputum cytology (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO ASTHMA PHENOTYPE:

About 73% of the study subjects were of allergic phenotype while 15% and 8% of the study subjects had smoking related asthma and asthma with obesity, respectively.

Table 27: Distribution of the study subjects according to Asthma phenotype (n=250)

Asthma phenotype	Frequency	Percent
Allergic phenotype	182	72.8
Asthma with obesity	20	8.0
Aspirin intolerant asthma	10	4.0
Smoking related asthma	38	15.2
Total	250	100.0

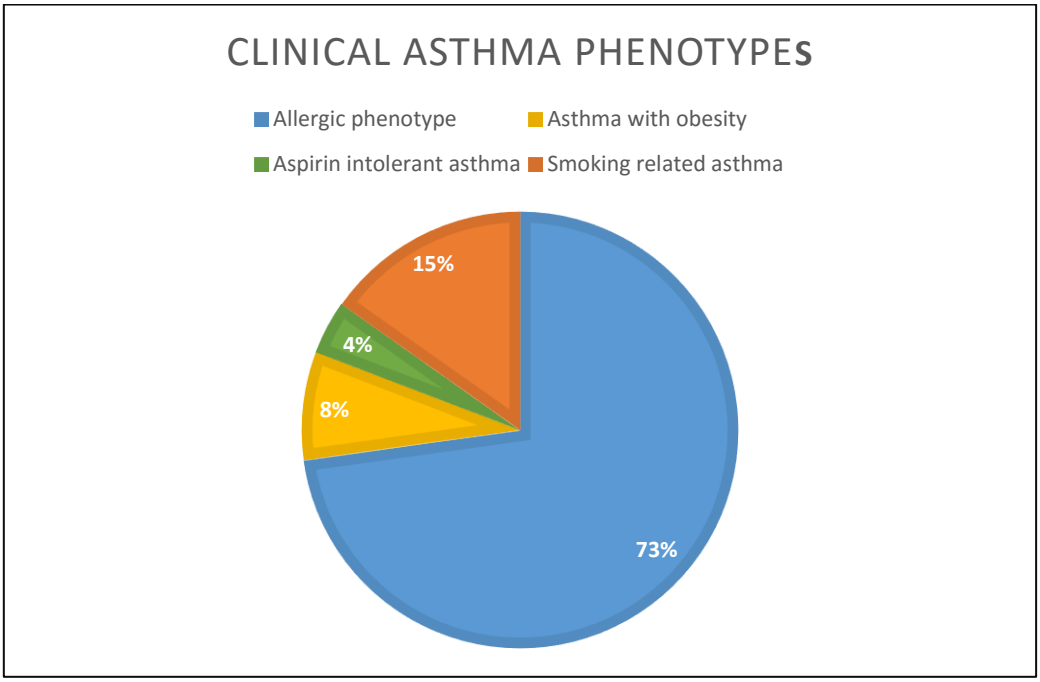

Fig 39: Distribution of the study subjects according to Asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO AGE AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype have a low mean age in comparison to non-allergic phenotype (29 years vs 41 years) and this difference in mean age was statistically significant.

Table 28: Distribution of the study subjects according to Age and asthma phenotype (n=250)

	Allergic phenotype		Non-Allergic phenotype		Mean difference	Student 't' test p value
	Mean	Standard Deviation	Mean	Standard Deviation		
AGE	29.47	9.15	41.09	6.37	-11.621	<0.001

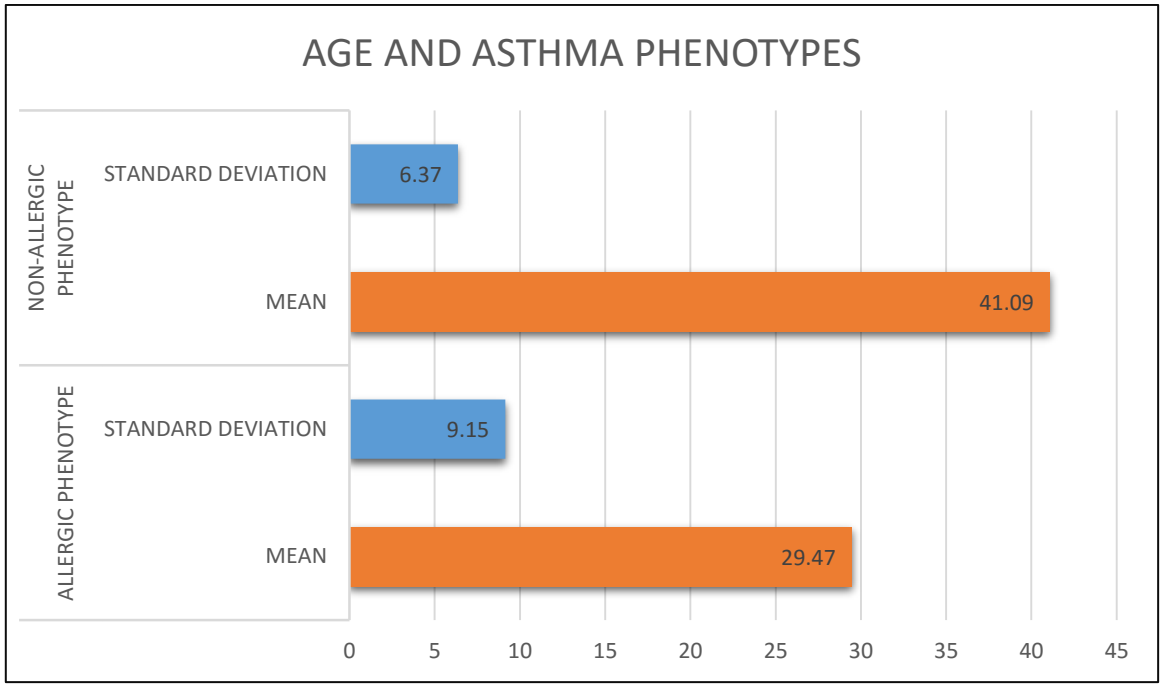

Fig 40: Distribution of the study subjects according to Age and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO GENDER AND ASTHMA PHENOTYPES:

The subjects with allergic phenotype are mostly females in comparison to non-allergic phenotype (65% vs 26%) and this difference in gender distribution was statistically significant.

Table 29: Distribution of the study subjects according to Gender and asthma phenotypes (n=250)

SEX	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Male	64	35.20%	50	73.50%
Female	118	64.80%	18	26.50%
Total	182	100.0%	68	100.0%

Chi-square value: 29.372 df =1 p value= <***0.001****

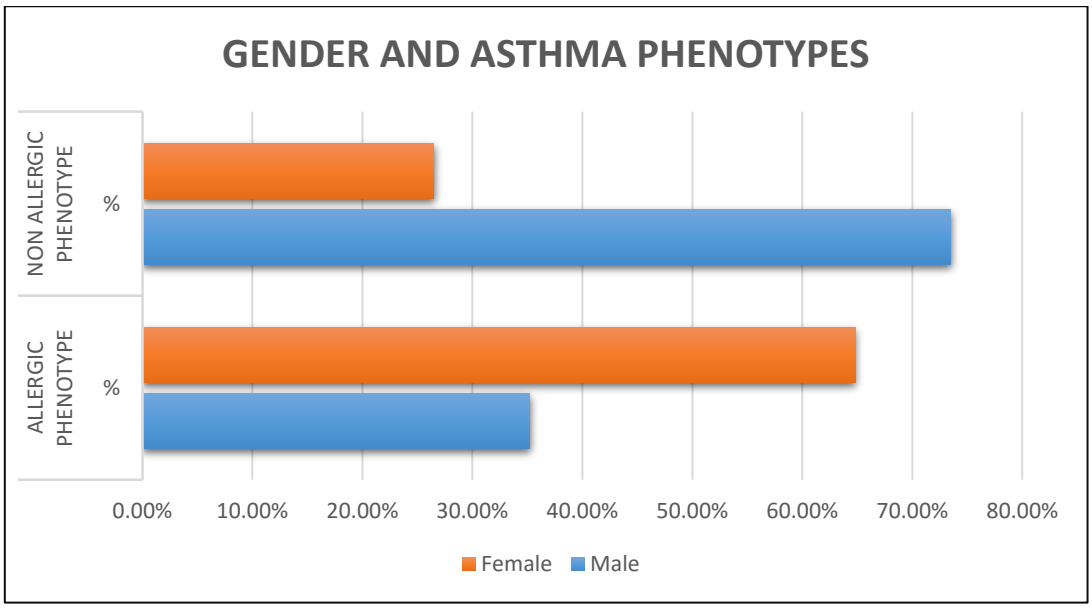

Fig 41: Distribution of the study subjects according to Gender and asthma phenotypes (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO ASTHMA PHENOTYPE AND BODY MASS INDEX:

The subjects with non-allergic phenotype had higher prevalence of overweight and obesity in comparison to allergic phenotype and this difference was statistically significant.

Table 30: Distribution of the study subjects according to asthma phenotype and Body mass index (n=250)

BMI	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Normal	118	64.8%	27	39.7%
Overweight	56	30.8%	31	45.6%
Obese	8	4.4%	10	14.7%
Total	182	100.0%	68	100.0%

Chi-square value: 15.823 df =2 p value= ***<0.001****

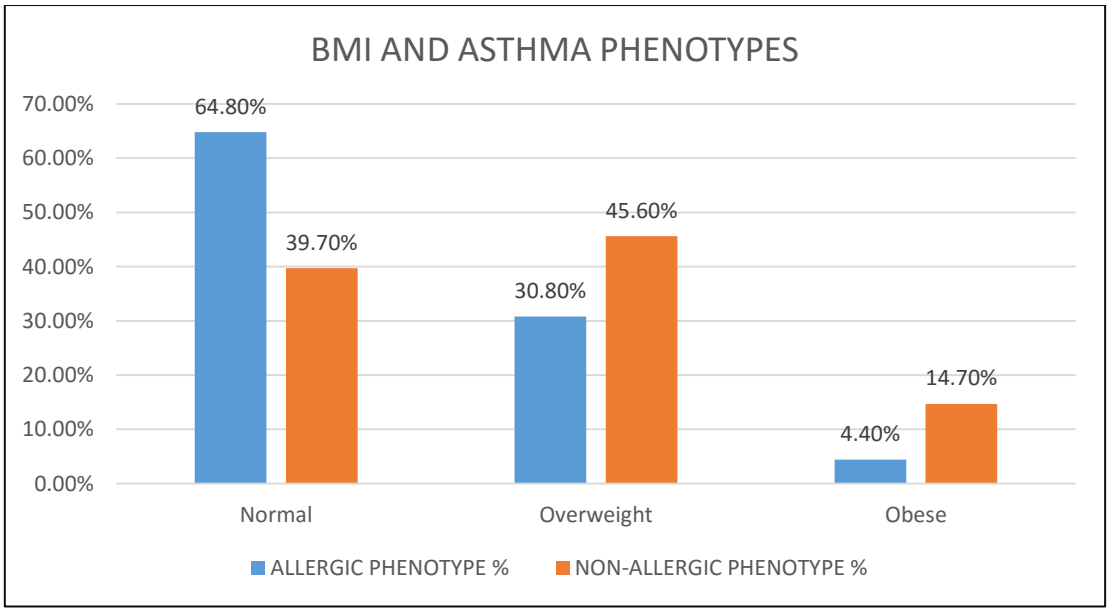

Fig 42: Distribution of the study subjects according to asthma phenotype and body mass index (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO ASTHMA PHENOTYPES AND BIRTH ORDER:

The subjects with allergic phenotype are mostly similar in comparison to non-allergic phenotype in terms of birth order (57% vs 43%).

Table 31: Distribution of the study subjects according to asthma phenotypes and Birth order (n=250)

Birth order	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
1st order	123	67.6%	39	57.4%
2nd order and above	59	32.4%	29	42.6%
Total	182	100.0%	68	100.0%

Chi-square value: 2.271 df =1 p value= 0.132

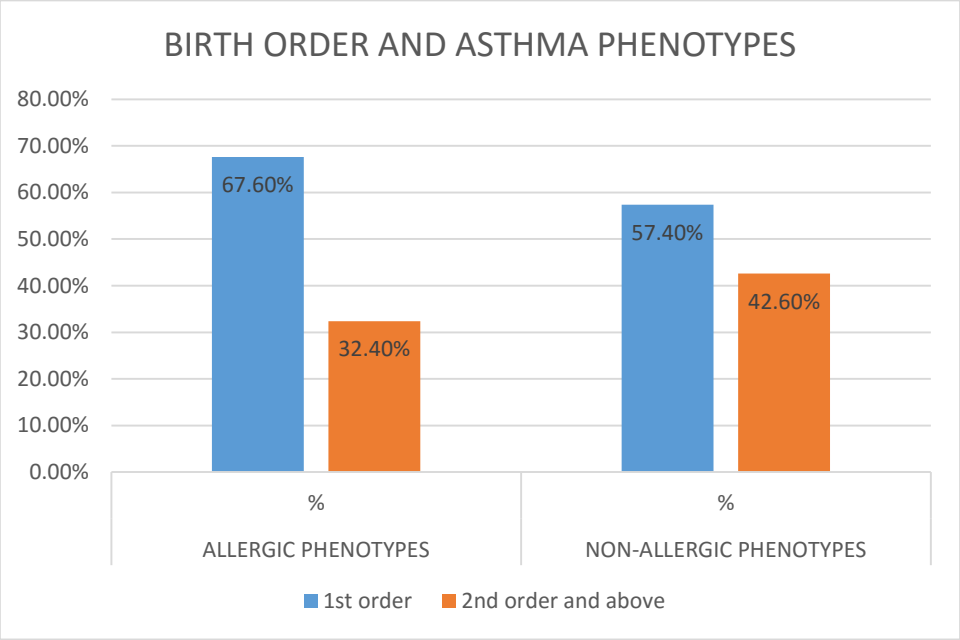

Fig 43: Distribution of the study subjects according to asthma phenotypes and Birth order (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO AGE OF ONSET AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had mostly early onset in comparison to non-allergic phenotype (87% vs 47%) and this difference was statistically significant.

Table 32: Distribution of the study subjects according to Age of onset and asthma phenotype (n=250)

Age of onset	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Early onset	159	87.4%	32	47.1%
Late onset	23	12.6%	36	52.9%
Total	182	100.0%	68	100.0%

Chi-square value: 44.599 df =1 p value= <***0.001****

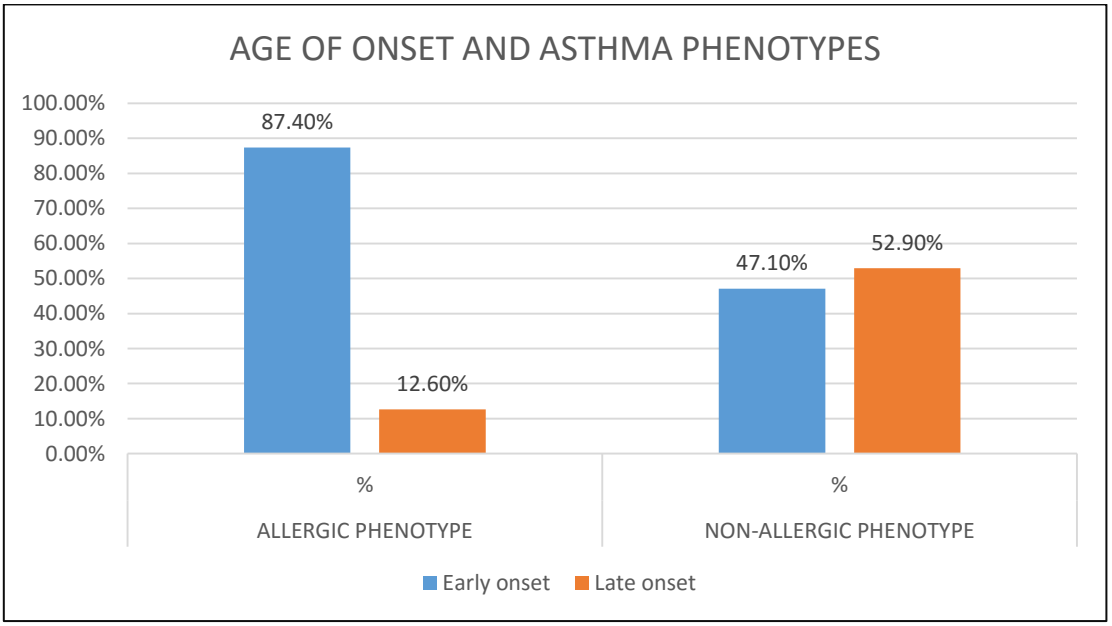

Fig 44: Distribution of the study subjects according to Age of onset and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF ATOPY AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had high proportion of atopy in comparison to non-allergic phenotype (72% vs 4%) and this difference was statistically significant.

Table 33: Distribution of the study subjects according to History of atopy and asthma phenotype (n=250)

History of atopy	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Present	132	72.5%	3	4.4%
Absent	50	27.5%	65	95.6%
Total	182	100.0%	68	100.0%

Chi-square value: 92.466 df =1 p value= <***0.001****

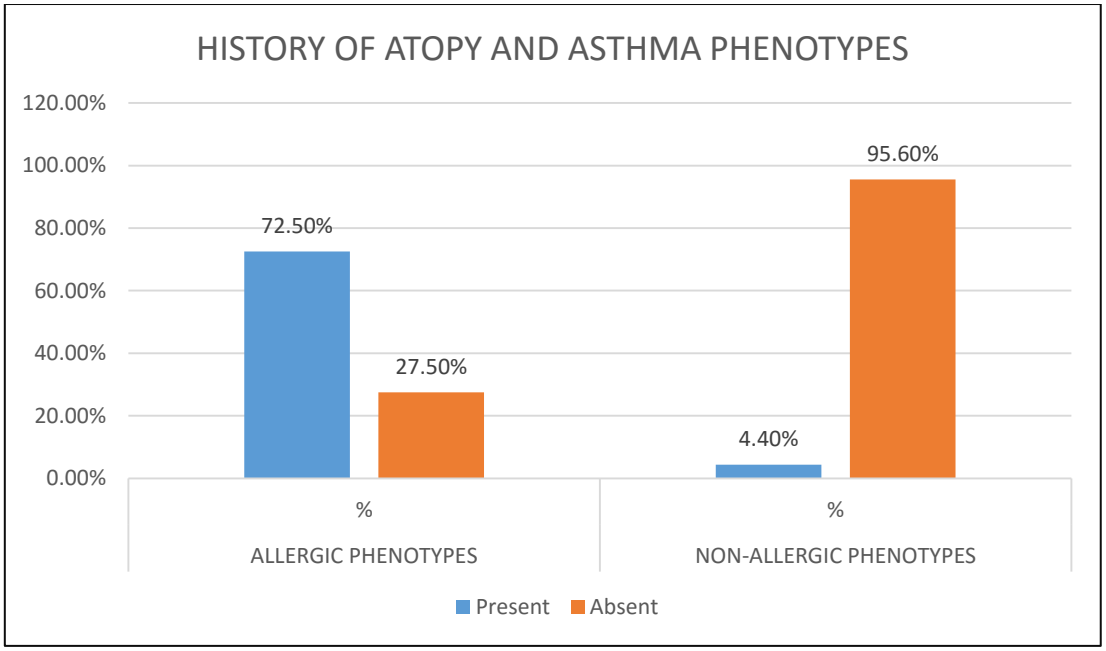

Fig 45: Distribution of the study subjects according to History of atopy and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HEREDITARY HISTORY AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had high proportion of inheritance in the family in comparison to non-allergic phenotype (64% vs 12%) and this difference was statistically significant.

Table 34: Distribution of the study subjects according to Hereditary history and asthma phenotype (n=250)

Inheritance	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Hereditary	117	64.3%	8	11.8%
Non hereditary	65	35.7%	60	88.2%
Total	182	100.0%	68	100.0%

Chi-square value: 54.622 df =1 p value= <***0.001****

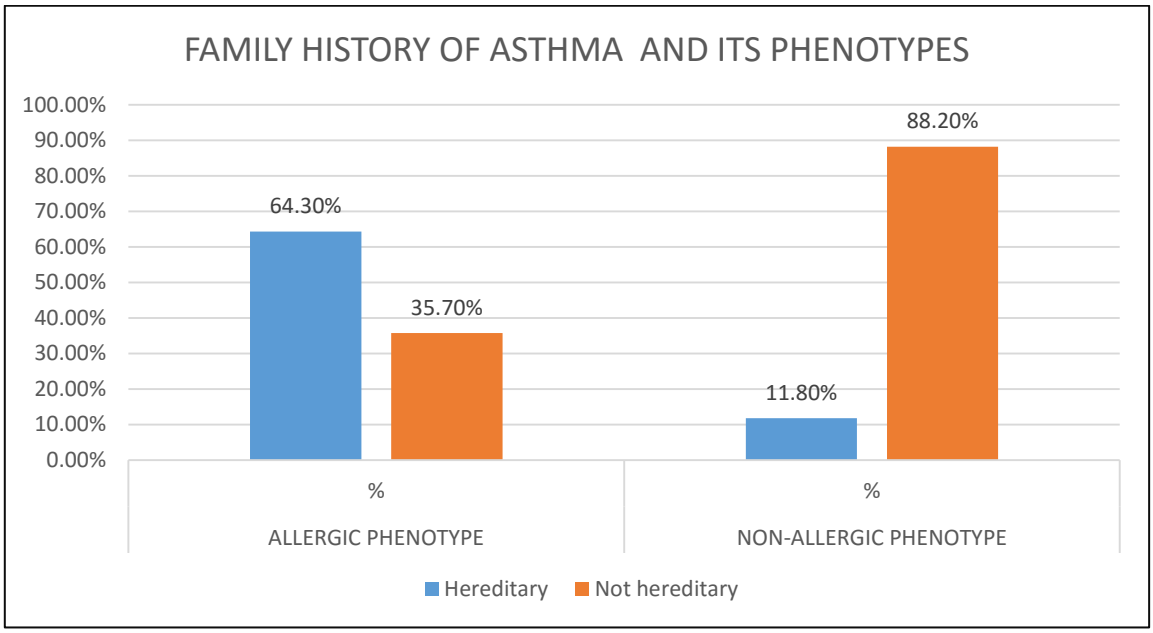

Fig 46: Distribution of the study subjects according to Hereditary history and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO HISTORY OF RECURRENT RESPIRATORY TRACT INFECTIONS (RRTI) AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had high proportion of recurrent respiratory tract infections in comparison to non-allergic phenotype (82% vs 3%) and this difference was statistically significant.

Table 35: Distribution of the study subjects according to History of RRTIs and asthma phenotype (n=250)

H/o RRTI	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Yes	149	81.9%	2	2.9%
No	33	18.1%	66	97.1%
Total	182	100.0%	68	100.0%

Chi-square value: 128.93 df =1 p value= <***0.001****

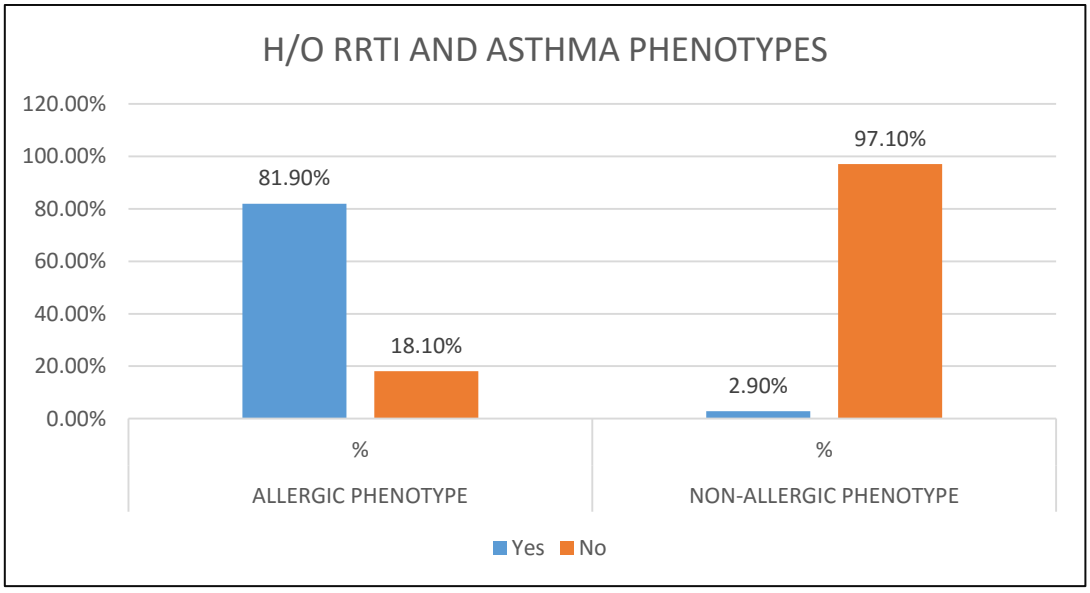

Fig 47: Distribution of the study subjects according to History of RRTIs and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SUSCEPTIBILITY TO WEATHER CHANGES AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had high proportion of susceptibility to weather change in comparison to non-allergic phenotype (82% vs 40%) and this difference was statistically significant.

Table 36: Distribution of the study subjects according to susceptibility to Weather change and asthma phenotype (n=250)

Weather changes	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Susceptible	149	81.9%	27	39.7%
Not susceptible	33	18.1%	41	60.3%
Total	182	100.0%	68	100.0%

Chi-square value: 42.230 df =1 p value= <***0.001****

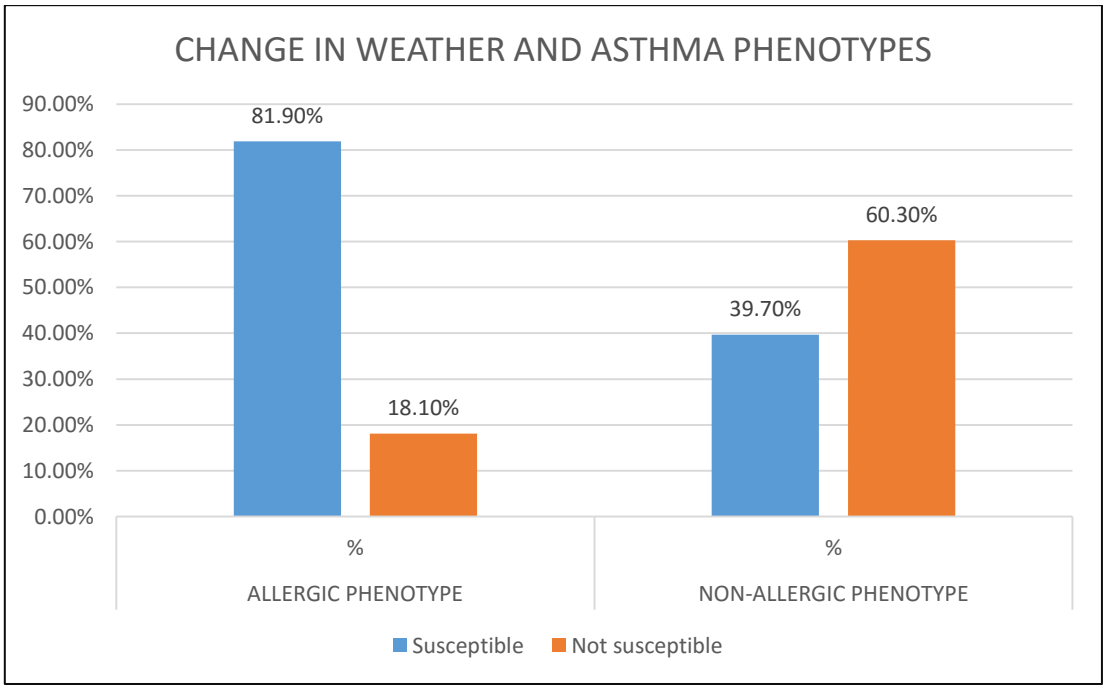

Fig 48: Distribution of the study subjects according to susceptibility to Weather change and asthma phenotype

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO ABSOLUTE EOSINOPHIL COUNT (AEC) AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had high mean absolute eosinophil count in comparison to non-allergic phenotype (587 vs 231) and this difference in mean absolute eosinophil count was statistically significant.

Table 37: Distribution of the study subjects according to Absolute eosinophil count and asthma phenotype (n=250)

	Allergic phenotype		Non-Allergic phenotype		Mean difference	Student 't' test p value
	Mean	Standard Deviation	Mean	Standard Deviation		
AEC	587.03	155.382	231.47	70.927	355.56	<0.001

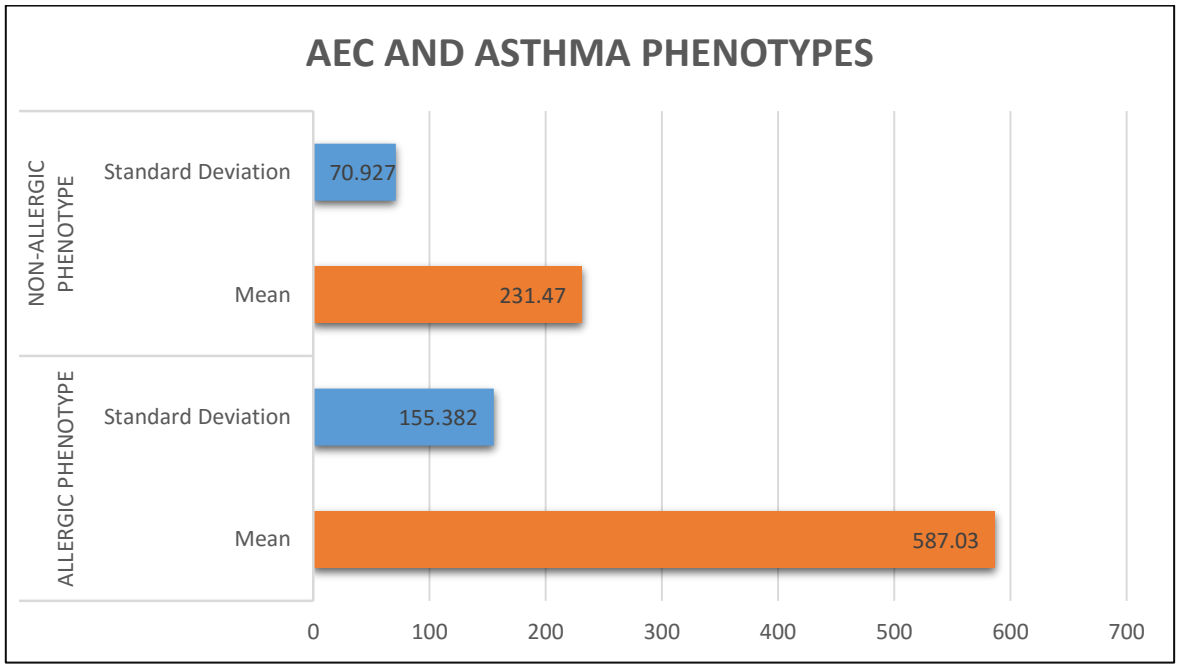

Fig 49: Distribution of the study subjects according to Absolute eosinophil count and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO MODE OF TREATMENT AND ASTHMA PHENOTYPE:

In our population, almost all patients were consuming oral medications on a regular basis. Delineation of patients taking inhalational drugs alone could not be made out. This is mainly due to the low socioeconomic status, poor literacy and lack of knowledge of the disease they continue to demand for oral drugs. But it has been found that majority of the subjects with allergic phenotype 150(60%) taking inhalational (controller medications) with oral had better response to treatment than oral drugs only.

Table 38: Distribution of the study subjects according to Mode of treatment and asthma phenotype (n=250)

Mode of treatment	Allergic phenotype	Non-Allergic phenotype	Total
Oral Drugs only (%)	32 (12.8%)	13 (19.1)	45 (18.0%)
Oral Drugs+Inhalational (LABA+ICS) (%)	150 (60%)	55 (22.0%)	205 (82%)
Total (%)	182	68	250 (100%)

Chi-square value: 140.30 df =3 p value= <***0.001****

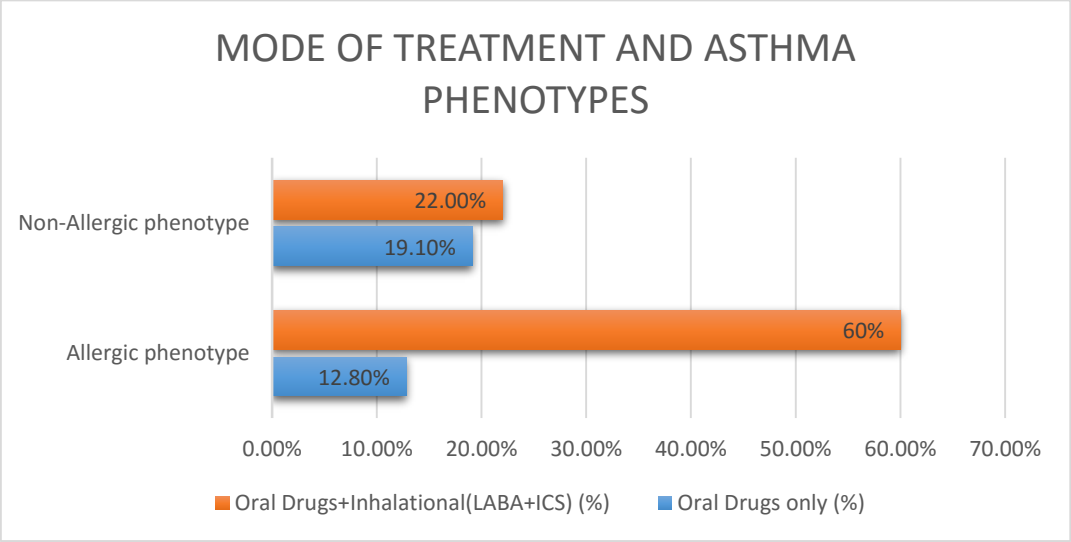

Fig 50: Distribution of the study subjects according to Mode of treatment and asthma phenotype (n=250)

DISTRIBUTION OF THE STUDY SUBJECTS ACCORDING TO SYMPTOM CONTROL AND ASTHMA PHENOTYPE:

The subjects with allergic phenotype had high proportion of symptom control in comparison to non-allergic phenotype (96% vs 59%) and this difference was statistically significant.

Table 39: Distribution of the study subjects according to Symptom control and asthma phenotype (n=250)

Symptom control	Allergic Phenotype		Non-Allergic Phenotype	
	N	%	N	%
Achieved	175	96.2%	40	58.8%
Not achieved	7	3.8%	28	41.2%
Total	182	100.0%	68	100.0%

Chi-square value: 57.298 df =1 p value= <***0.001****

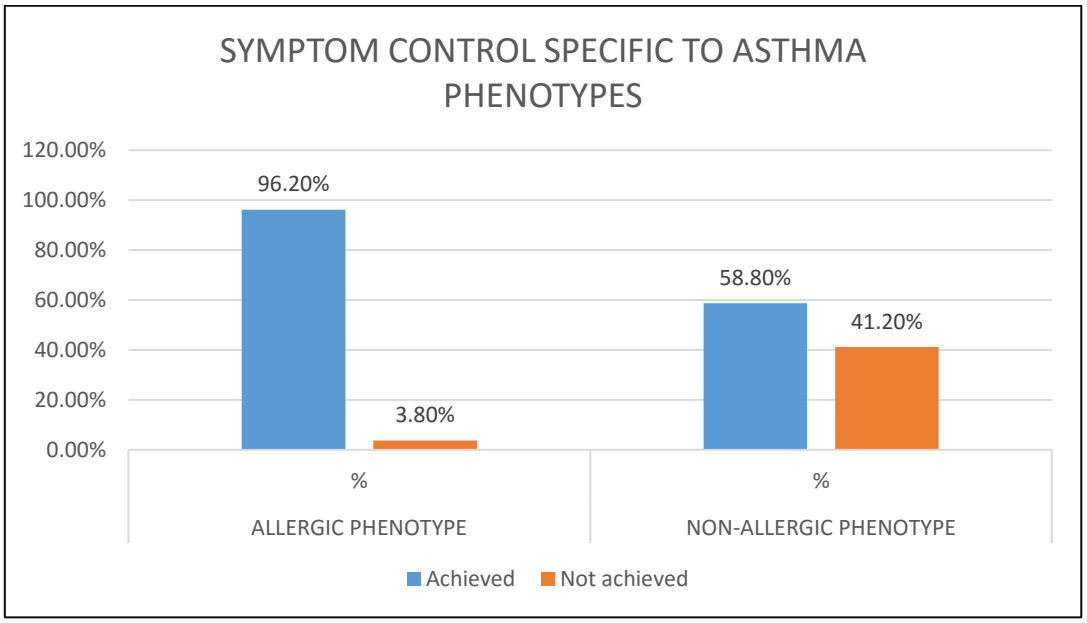

Fig 51: Distribution of the study subjects according to Symptomatic control and asthma phenotype (n=250)

DISCUSSION

Age distribution of study population:

The age distribution in our study population ranged from 10-56 years. The mean age is 32.63. The number of patients in the age group 10-20, 21-30, 31-40, 41-50 and 51-60 were 31(12.4%), 84(33.6%), 78(31.2%), 50(20%) and 7(2.8%) respectively. About 65% of the study subjects were in the age group of 21 to 40 years which denotes a higher prevalence of asthma in 2nd and 3rd decade of life which is usually documented in literature. When comparing the age with asthma phenotypes, the subjects with allergic phenotype have a low mean age in comparison to non-allergic phenotype (29 years vs 41 years) and this difference in mean age (-11.621; Student 't' test p value,0.001) was statistically significant.

Gender distribution of the study population:

Out of 250 study subjects, males were 114(45.6%) and females were 136(54.4%). The subjects with allergic phenotype are mostly females in comparison to non-allergic phenotype (65% vs 26%) and this difference in gender distribution was statistically significant (chi-square value: 29.372, P,0.001).

Distribution of study subjects according to the level of education:

Among the total number of 250 subjects included in our study only 88 (35.2%) were literates and 162 (64.8%) were illiterates. Though asthma is more prevalent among literates as proved by various studies, our study shows high prevalence of asthma among illiterates. This may be a limitation in our study due to the demographic influence in our study population.

Distribution of the study subjects according to socio-economic status:

Among the total number of 250 subjects included in our study, 160 (64%) subjects were classified under low socio-economic status according to modified Kuppuswamy scale and 90 (36%) subjects were classified under middle socio economic status. None of the study subjects were under high socio-economic status. Although asthma is considered as a disease of westernised population, it is more prevalent among the low socio-economic groups in our study. This may be a limitation in our study due to the demographic influence in our study population.

Distribution of study subjects according to body mass index:

The subjects were classified according to body mass index, we found that the number of subjects belonged to normal, overweight and obese were 145 (58%), 87 (34%) and 18 (7.2%) respectively. The subjects with non-allergic phenotype are having a higher prevalence of overweight and obesity (Chi-square value: 15.823; $P<0.001$) in comparison to allergic phenotype and this difference was statistically significant. Mostly asthma with obesity were females, late onset symptoms, less atopy and less significant hereditary pattern. Hence asthma with obesity is considered as a separate phenotype.

Distribution of study subjects according to birth order:

Out of 250 subjects included in our study, 162(64.8%) subjects were first born children in their families and 88 (35.2%) subjects were belonged to higher birth order i.e. 2^{nd} and above. When comparing the birth order with asthma phenotypes, the prevalence of asthma is high in the first born children (Chi-square value: 2.271;

P=0.132) irrespective of the phenotypes. But it was found to be statistically insignificant. This may be due to a small sample size of our study.

Distribution of study subjects according to age of onset:

About 191 (76.4%) subjects of the study population had developed asthma at an early age i.e. before the age of 40 years and 59 (23.6%) subjects had developed their symptoms late in their life i.e. 40 years and above. When comparing the age of onset of asthma symptoms and the asthma phenotypes, the subjects with allergic phenotype had mostly early onset in comparison to non-allergic phenotype (87% vs 47%) and this difference was statistically significant with Chi-square value of 44.599 and P value<0.001. Since they possess distinct clinical features and management strategies they are considered as a separate phenotype in asthma.

Distribution of study subjects according to history of smoking:

All females in our study group are non-smokers. This may be due to the fact that our population had not yet exposed to the westernized culture. Among the total males, 52 patients (20.8%) were non-smokers and 62 (24.8%) were smokers. The patients with history of smoking were predominantly in the older age group and are associated with decreased blood eosinophilia.

Distribution of study subjects according to the exposure of passive smoking:

Considering the children and female subjects, who had asthmatic symptoms, 111 (44.4%) patients had given positive history of exposure to second hand smoking. This is considerably high when compared to the prevalence found in previous literatures. This shows high proportion of females and children also had passive smoking as a triggering factor for the worsening of asthma symptoms.

Distribution of study subjects according to history of atopy:

Among 250 patients included in our study, 132 (52.8%) subjects had history of atopy and 118 (47.2%) patients had no history of atopy. Subjects with allergic phenotype had high proportion of atopy in comparison to non-allergic phenotype (72% vs 4%) and this difference was statistically significant (Chi-square value: 92.466; P<0.001) which is usually documented in the literatures. The presence of atopy to food products, animal products, environmental dust and pollen dust were 52 (20.8%), 33(13.2%), 31(12.4%), and 16(6.4%) respectively.

Distribution of study subjects according to the hereditary pattern of asthma:

Among the 250 patients included in our study, 129 (51.6%) patients had given positive history for the presence of asthma in family members and 121 (48.4%) patients did not have a positive history of asthma in family. Out of 129 patients, positive history of asthma in mother, father and both were 76 (30.4%), 41 (16.4%) and 12 (4.8%) respectively. Subjects with allergic phenotype had increased prevalence of asthma in their family members in comparison to non-allergic phenotype (64% vs 12%) and this difference was statistically significant (Chi-square value: 54.622; P<0.001). The inheritance from mothers is high than fathers and this difference was also statistically significant (Chi-square value: 59.779; P<0.001).

Distribution of study subjects according to history of exposure to aspirin:

Among the 250 patients included in our study, 16 patients had given positive history of increase in the asthmatic attacks after the ingestion of aspirin or other non-steroidal anti-inflammatory drugs. They do not show any history of atopy or family

history of asthma. This prevalence is usually documented in literatures. Due to its specific characteristics of asthma pattern they are considered as a separate phenotype.

Distribution of study subjects according to family size:

Out of 250 subjects, 158 (63.2%) patients were living in a small family and 92 (36.8%) patients were living in a large family. Subjects with non-allergic phenotype belonged to high proportion of small family size in comparison to allergic phenotype (76% vs 58%) and this difference was statistically significant (Chi-square value: 7.073; P=0.008).

Distribution of the study subjects according to history of recurrent respiratory infections (RRTI):

Out of 250 subjects, history of recurrent respiratory tract infections was positive in 151 (60.4%) subjects. Subjects with allergic phenotype had high proportion of recurrent respiratory tract infections in comparison to non-allergic phenotype (82% vs 3%; Chi-square value-128.93; P<0.001) and this difference was statistically significant.

Distribution of the study subjects according to susceptibility to weather changes:

Out of 250 subjects, 176 (70.4%) were susceptible to develop asthma symptoms due to weather changes. Subjects with allergic phenotype had increased susceptibility to weather changes in comparison to non-allergic phenotype (80% vs 40%; Chi-square value-42.230; P<0.001) and this difference was statistically significant.

Distribution of study subjects according to history of GERD:

Out of 250 patients, 47 (18.8%) patients were suffering from gastro-oesophageal reflux disorder. Most of the subjects are elderly females and with increased BMI. Hence GERD plays a role in the exacerbation of symptoms.

Distribution of study subjects according to susceptibility to stress to develop asthma:

Among the 250 patients, 72 (28.8%) patients had symptoms susceptible secondary to stress or emotional liability. This reflects the amount of stress undergone by the urban population which acts as a trigger for asthma symptoms.

Distribution of study subjects according to the knowledge of diagnosis and mode of treatment:

Out of 250 subjects, patients taking only oral drugs were 45 (18.0%) and both oral with inhalational (LABA+ICS) were 205 (82.0%). This is mainly due to the low socioeconomic status, poor literacy and lack of knowledge of the disease they continue to demand for oral drugs. Among 182 subjects of allergic phenotype, symptom control was achieved in 175 (96.2%). Among 68 subjects of non-allergic phenotype, symptom control was achieved in 40 (58.8%). Subjects with allergic phenotype had high proportion of symptomatic control in comparison to non-allergic phenotype (96% vs 59%) and this difference was statistically significant (Chi-square value-57.298; $P<0.001$).

Distribution of the study subjects according to absolute eosinophil count:

Among the 250 patients, 181 (72.4%) patients had elevated absolute eosinophil count. Absolute eosinophil count is the indirect measure of susceptibility of the subjects to atopy and it has been found that these subjects with allergic phenotype has high mean

absolute eosinophil count in comparison to non-allergic phenotype (587 vs 231) and this difference in mean absolute eosinophil count was statistically significant (Mean Difference-355.56; P<0.001).

Distribution of the study subjects according to sputum cytology:

Out of 250 patients, the total number of patients classified under eosinophilic, neutrophilic, mixed neutrophilic and eosinophilic and Pauci-granulocytic were 182(72.8%), 47(18.8%), 19(7.6%) and 2(0.8%) respectively. This reflects the high proportion of patients with allergic phenotype had predominantly eosinophilic sputum cytology which is usually documented in the literature as the cellular phenotypes of asthma.

Distribution of study subjects according to the clinical asthma phenotypes:

Among the 250 subjects included in our study, four distinct clinical phenotypes were allergic 182(72.8%), asthma with obesity 20(8.0%), aspirin evoked 10(4.0%) and smoking related 38(15.2%) were found. In our study it is clearly evident that major proportion of the study subjects had elevated AEC and high sputum eosinophilia which shows high prevalence of atopy among the individuals living in the urban area who are constantly exposed to high amount of environmental air pollution.

CONCLUSION

- Phenotyping of asthma serves as a stepping stone toward the practice of personalised treatment for asthma.
- Recent treatment guidelines are aimed at reversing the bronchospasm with SABA and LABA and decreasing the airway inflammation with ICS would help to achieve asthma control among the allergic phenotypes only
- The treatment of asthma based on phenotype would reduce the likelihood of prescribing wrong drugs to wrong patients. It will also decrease the number of "difficult to treat" asthmatics and minimising the burden of this chronic heterogeneous inflammatory disease in a community.
- Although asthma is a disease of westernised population as proved by various studies, our study concludes that asthma is a disease of all irrespective of socio-economic status.
- Hence untangling asthma phenotypes is a right direction towards a tailored management of asthma.

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LIST OF ABBREVIATIONS USED

Th2	-Type 2 helper cells
LRI	-Lower Respiratory Infections
ICS	-Inhaled Corticosteroids
IgE	-Immunoglobulin E
BMI	-Body Mass Index
SES	-Socio-economic status
SHS	-Second Hand Smoke
EGEA	-Epidemiological study on the genetics and environment of asthma
NSAID	-Non-steroidal anti-inflammatory drugs
AIA	-Aspirin intolerant asthma
GINA	-Global Initiative for Asthma
COPD	-Chronic Obstructive Pulmonary Disease
GERD	-Gastro-oesophageal reflux disease
AEC	-Absolute eosinophil count
RRTI	-Recurrent respiratory tract infection
SABA	-Short acting Beta 2 agonist
LABA	-Long acting Beta 2 agonist

ETHICAL COMMITTEE APPROVAL CERTIFICATE

INSTITUTIONAL ETHICS COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No. 18/2016 Dt: 04.04.2016
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval " **To study the epidemiological aspects in determining the prevalence and expression of asthma phenotype in the urban population of north chennai** " - For Project Work submitted by **Dr.P.Dhamodharan**, PG TB and Respiratory Diseases, Govt. Kilpauk Medical College, Chennai – 10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

DEAN,
Govt.Kilpauk Medical College,
Chennai – 10.

PC 2cME I Section<Ethical Committee 1

PROFORMA

PATIENT'S DEMOGRAPHY

1. Serial No. 2. Date:

3. Name:

4. Age:

5. Gender:

6. Address:

7. Phone:

8. Religion: Hindu/Christian/Muslim/Others
9. Habitat/Locality: Rural/Urban
10. Level of education: literate/Illiterate
11. Occupation: _____

12. Socio-economic status Low/Middle/Upper
13. BMI Underweight (Below 18.5)
Normal (18.5-24.9)
Overweight (25.0-29.9)
Obese (30 and above)

CHILDHOOD HISTORY

14. Birth Order _____
15. Exposure to recurrent infection RS/GIT
16. Immunisation status _____
17. Exposure to Farm Products Yes/No
18. Exposure to Smoke Yes/No

RISK FACTORS/PREDISPOSING FACTORS

19. Allergy/Atopy Yes/No
20. Hereditary Yes/No

If yes Mother/Father/Both
Grandparents
Siblings

CAUSAL FACTORS

21. Exposure to allergens	Pollen dust/Environmental dust Food products/Animals
22. Exposure to chemicals in work place	Yes/No
23. H/O drug intake (Aspirin/NSAIDS)	Yes/No

CONTRIBUTING FACTORS

24. H/O Environmental Tobacco Smoke	Yes/No
25. H/O Upper respiratory infection	Yes/No
26. H/O Exposure to Air Pollution	Yes/No

PROTECTING FACTORS

27. Food habits	Veg/Non-Veg
28. Habitation	Rural/Urban
29. Family Size	Large/Small

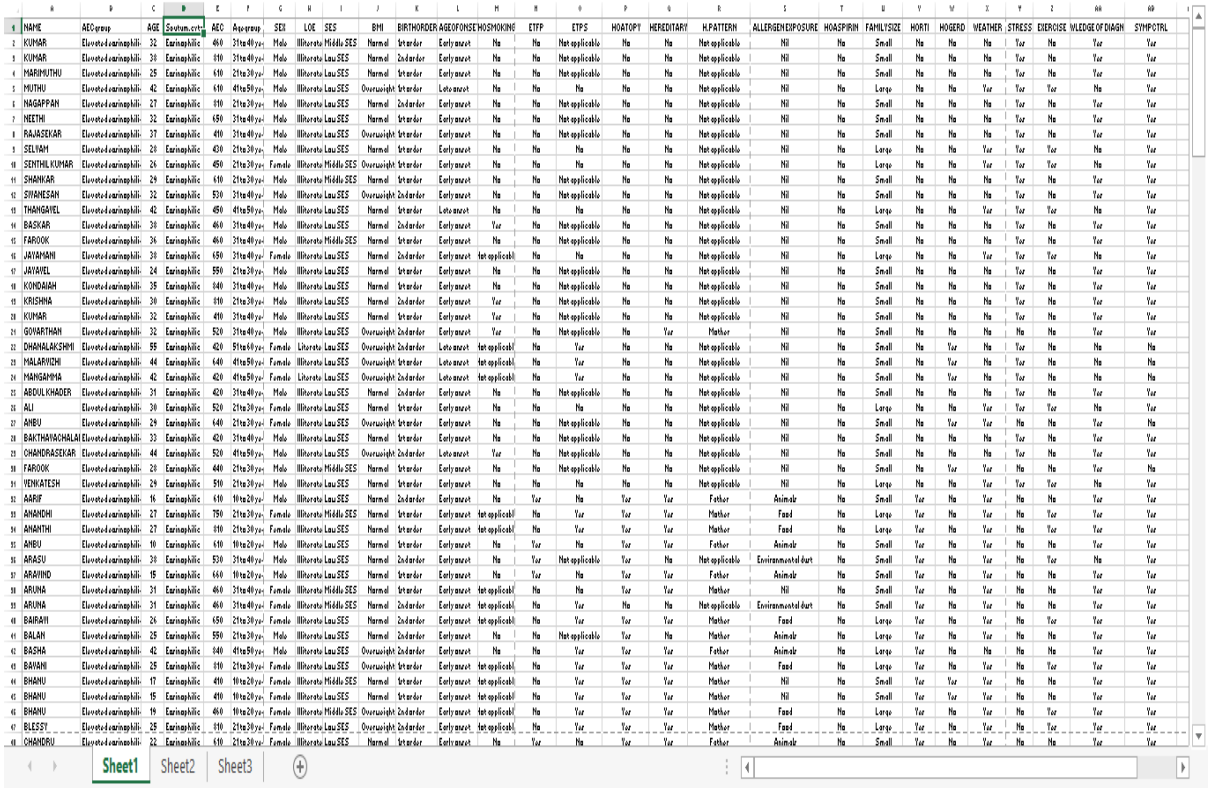
TRIGGERING FACTORS

30. H/O Respiratory infection	Yes/No
31. H/O GERD	Yes/No
32. Change in weather conditions	Yes/No
33. Pregnancy	Yes/no
34. H/O Stress and Emotional conditions	Yes/No
35. Whether exercise induced	Yes/No

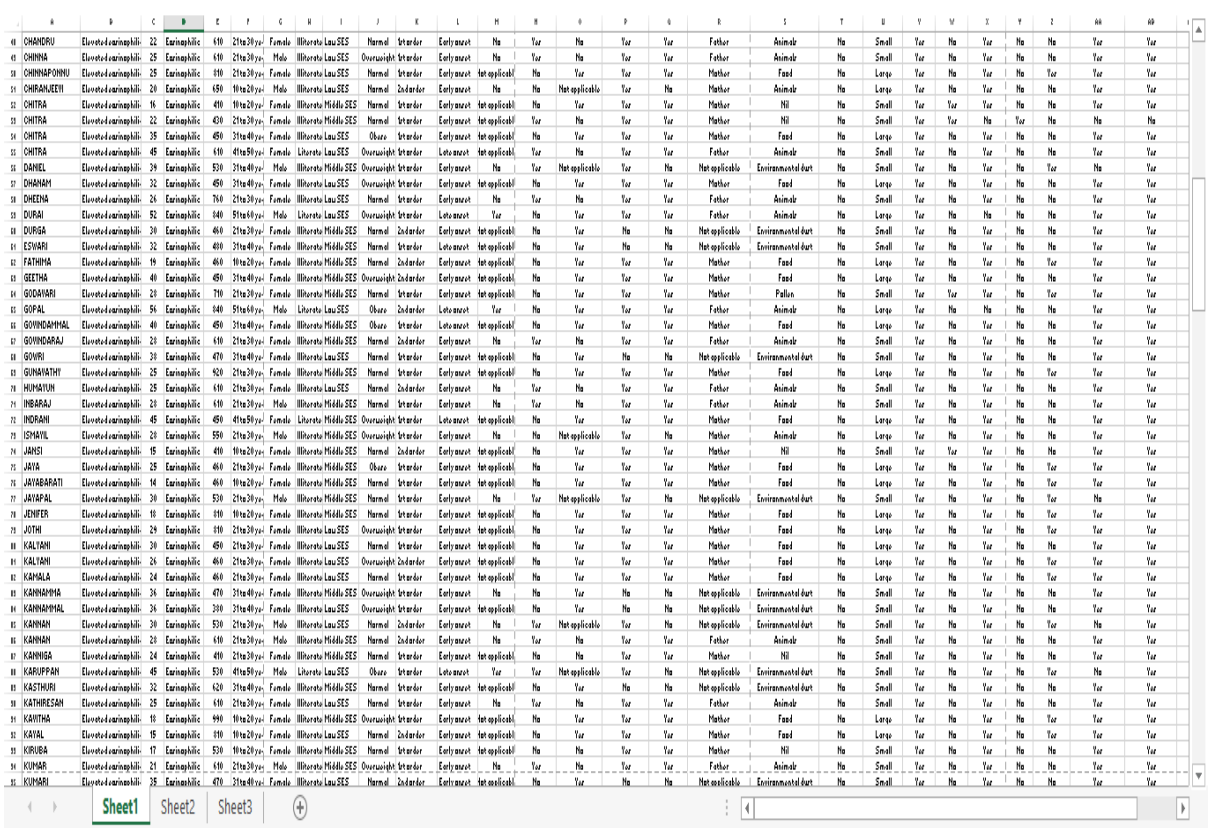
PATIENT'S KNOWLEDGE

36. Knowledge about the disease	Yes/No
37. Knowledge about the diagnosis	Yes/No
If Yes	Spirometry/Clinical
38. Mode of treatment undertaken	_____
39. Knowledge about the treatment outcome	Yes/No
40. Whether symptoms controlled	Yes/No
41. Absolute eosinophil count	_____

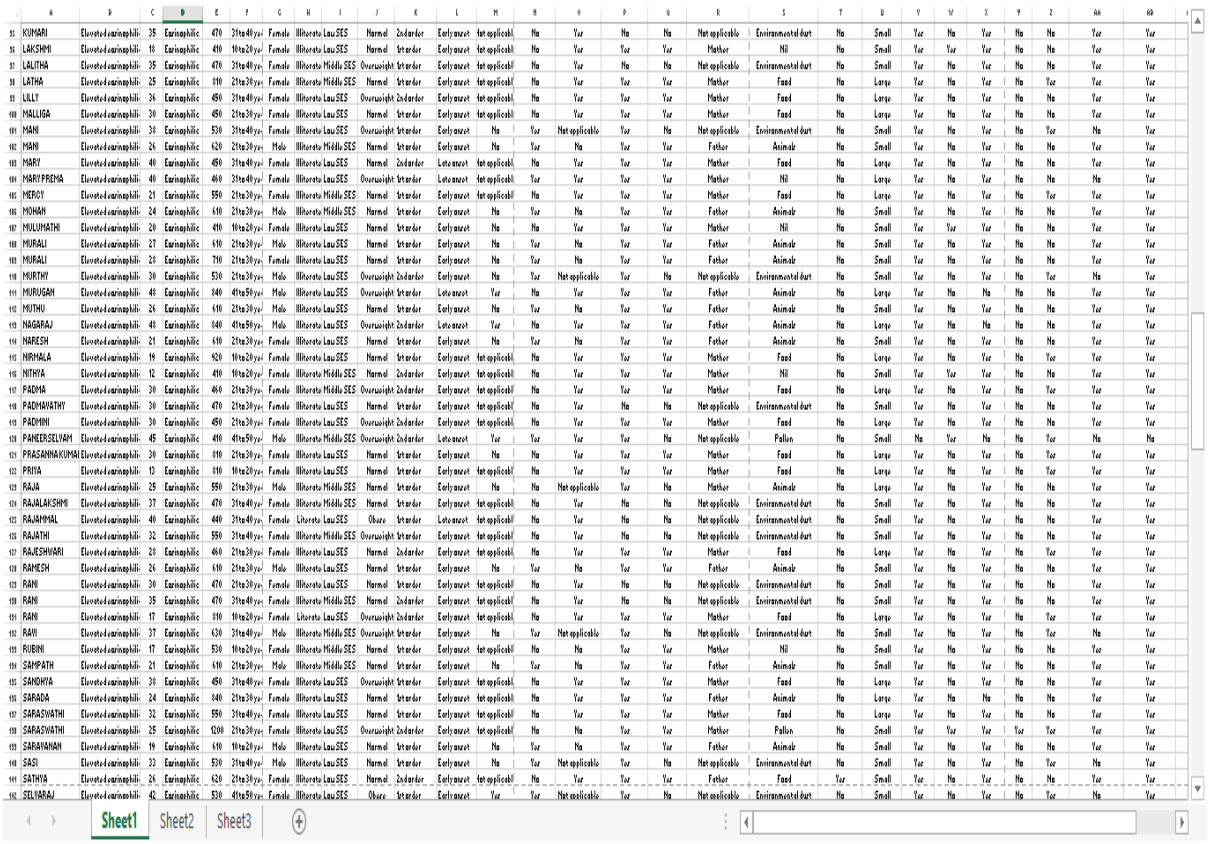
MASTER CHARTS



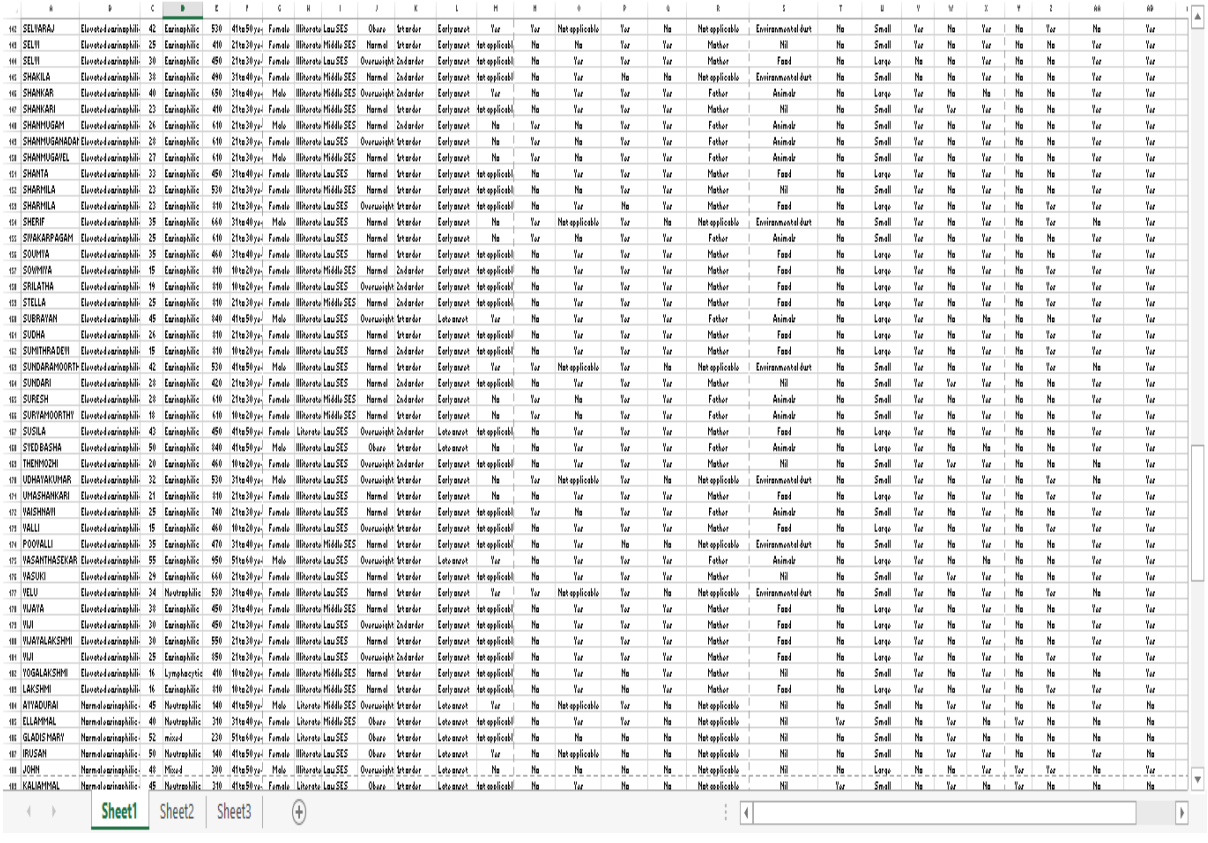
NAME	AEGroup	AGE	Sautum.crit	AEC	Agegroup	SEX	LOE	SES	BMI	BIRTHORDER	AGEOFONSET	HOSMOKING	ETFP	ETPS	HOATOPY	HEREDITARY	H.PATTERN	ALLERGENEXPOSURE	HOASPIRIN	FAMILYSIZE	HORTI	HOGERD	WEATHER	STRESS	EXERCISE	WLEDGE OF DIAGN	SYMPOTRL
KUMAR	Elevated earinophili	32	Earinophilic	460	31to40yrs	Male	Illiterate	Middle SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
KUMAR	Elevated earinophili	38	Earinophilic	610	31to40yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
MARIMUTHU	Elevated earinophili	25	Earinophilic	610	21to30yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
MUTHU	Elevated earinophili	42	Earinophilic	610	41to50yrs	Male	Illiterate	Low SES	Overweight	1ntordor	Late onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
NAGAPPAN	Elevated earinophili	27	Earinophilic	610	21to30yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
NEETHI	Elevated earinophili	32	Earinophilic	650	31to40yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
RAJASEKAR	Elevated earinophili	37	Earinophilic	410	31to40yrs	Male	Illiterate	Low SES	Overweight	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
SELVAM	Elevated earinophili	20	Earinophilic	420	21to30yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
SENTHILKUMAR	Elevated earinophili	26	Earinophilic	450	21to30yrs	Female	Illiterate	Middle SES	Overweight	1ntordor	Early onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
SHANKAR	Elevated earinophili	29	Earinophilic	610	21to30yrs	Male	Illiterate	Middle SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
SIVANESAN	Elevated earinophili	32	Earinophilic	530	31to40yrs	Male	Illiterate	Low SES	Overweight	2ndordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
THANGAVEL	Elevated earinophili	42	Earinophilic	450	41to50yrs	Male	Illiterate	Low SES	Normal	1ntordor	Late onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
BASKAR	Elevated earinophili	38	Earinophilic	460	31to40yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	Yes	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
FAROOK	Elevated earinophili	36	Earinophilic	460	31to40yrs	Male	Illiterate	Middle SES	Normal	2ndordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
JAYAMANI	Elevated earinophili	38	Earinophilic	650	31to40yrs	Female	Illiterate	Low SES	Normal	2ndordor	Early onset	not applicable	No	No	No	No	Not applicable	Nil	No	Large	No	No	No	Yes	Yes	No	Yes
JAYAVEL	Elevated earinophili	24	Earinophilic	550	21to30yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
KONDAIAH	Elevated earinophili	35	Earinophilic	640	31to40yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
KRISHNA	Elevated earinophili	30	Earinophilic	610	21to30yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	Yes	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
KUMAR	Elevated earinophili	32	Earinophilic	410	31to40yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	Yes	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
GOVARTHAN	Elevated earinophili	32	Earinophilic	520	31to40yrs	Male	Illiterate	Low SES	Overweight	2ndordor	Early onset	Yes	No	Not applicable	No	Yes	Mother	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
DHAMALAKSHMI	Elevated earinophili	59	Earinophilic	420	51to60yrs	Female	Literate	Low SES	Overweight	2ndordor	Late onset	not applicable	No	Yes	No	No	Not applicable	Nil	No	Small	No	Yes	No	No	No	No	No
MALARVIZHI	Elevated earinophili	44	Earinophilic	640	41to50yrs	Female	Illiterate	Low SES	Overweight	2ndordor	Late onset	not applicable	No	Yes	No	No	Not applicable	Nil	No	Small	No	Yes	No	No	No	No	No
MANGAMMA	Elevated earinophili	42	Earinophilic	420	41to50yrs	Female	Literate	Low SES	Overweight	2ndordor	Late onset	not applicable	No	Yes	No	No	Not applicable	Nil	No	Small	No	Yes	No	No	No	No	No
ABDULKHADER	Elevated earinophili	31	Earinophilic	420	31to40yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
ALI	Elevated earinophili	30	Earinophilic	520	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
ANBU	Elevated earinophili	29	Earinophilic	640	21to30yrs	Female	Illiterate	Low SES	Overweight	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	Yes	Yes	No	No	Yes
BAKTHAVACHALAI	Elevated earinophili	33	Earinophilic	420	31to40yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
CHANDRASEKAR	Elevated earinophili	44	Earinophilic	520	41to50yrs	Male	Illiterate	Low SES	Overweight	2ndordor	Late onset	Yes	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	No	No	Yes	No	Yes	Yes
FAROOK	Elevated earinophili	28	Earinophilic	440	21to30yrs	Male	Illiterate	Middle SES	Normal	1ntordor	Early onset	No	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	Yes	Yes	No	No	Yes	No
VENKATESH	Elevated earinophili	29	Earinophilic	510	21to30yrs	Male	Illiterate	Middle SES	Normal	1ntordor	Early onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
AARIF	Elevated earinophili	16	Earinophilic	610	16to20yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
AMANDHI	Elevated earinophili	27	Earinophilic	750	21to30yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
AMANTHI	Elevated earinophili	27	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
ANBU	Elevated earinophili	10	Earinophilic	610	16to20yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
ARASU	Elevated earinophili	38	Earinophilic	530	31to40yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	Yes	Not applicable	No	Yes	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
ARAVIND	Elevated earinophili	15	Earinophilic	660	16to20yrs	Male	Illiterate	Low SES	Normal	1ntordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
ARUNA	Elevated earinophili	31	Earinophilic	460	31to40yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	No	Yes	Yes	Nil	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
ARUNA	Elevated earinophili	31	Earinophilic	460	31to40yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
BAIRAVI	Elevated earinophili	26	Earinophilic	650	21to30yrs	Female	Illiterate	Low SES	Normal	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
BALAN	Elevated earinophili	25	Earinophilic	550	21to30yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	No	Not applicable	Yes	No	Mother	Animals	No	Large	Yes	No	Yes	No	No	Yes	Yes
BASHA	Elevated earinophili	42	Earinophilic	640	41to50yrs	Male	Illiterate	Low SES	Overweight	2ndordor	Early onset	No	No	Yes	Yes	No	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
BAVANI	Elevated earinophili	25	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Overweight	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
BHANU	Elevated earinophili	17	Earinophilic	410	16to20yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
BHANU	Elevated earinophili	15	Earinophilic	410	16to20yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
BHANU	Elevated earinophili	19	Earinophilic	460	16to20yrs	Female	Illiterate	Middle SES	Overweight	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	No	No	Yes	Yes	Yes
BLESSY	Elevated earinophili	25	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Overweight	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	No	No	Yes	Yes	Yes
CHANDRU	Elevated earinophili	22	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	No	No	Yes	No	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes



CHANDRU	Elevated earinophili	22	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
CHINNA	Elevated earinophili	25	Earinophilic	610	21to30yrs	Male	Illiterate	Low SES	Overweight	1ntordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
CHINNAPONNU	Elevated earinophili	25	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
CHIRANJEEVI	Elevated earinophili	20	Earinophilic	650	16to20yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	No	Not applicable	Yes	No	Mother	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
CHITRA	Elevated earinophili	16	Earinophilic	410	16to20yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
CHITRA	Elevated earinophili	22	Earinophilic	430	21to30yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	Yes	No	Yes	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	No	No
CHITRA	Elevated earinophili	35	Earinophilic	450	31to40yrs	Female	Illiterate	Low SES	Obese	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
CHITRA	Elevated earinophili	45	Earinophilic	610	41to50yrs	Female	Literate	Low SES	Overweight	1ntordor	Late onset	not applicable	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
DANIEL	Elevated earinophili	39	Earinophilic	530	31to40yrs	Male	Illiterate	Middle SES	Overweight	1ntordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	No
DHANAM	Elevated earinophili	32	Earinophilic	450	31to40yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
DHEENA	Elevated earinophili	26	Earinophilic	760	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
DURAI	Elevated earinophili	52	Earinophilic	840	51to60yrs	Male	Literate	Low SES	Overweight	1ntordor	Late onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
DURGA	Elevated earinophili	30	Earinophilic	460	21to30yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
ESWARI	Elevated earinophili	32	Earinophilic	490	31to40yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
FATHIMA	Elevated earinophili	19	Earinophilic	460	16to20yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
GEETHA	Elevated earinophili	40	Earinophilic	450	31to40yrs	Female	Illiterate	Middle SES	Overweight	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
GODAVARI	Elevated earinophili	20	Earinophilic	710	21to30yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Pollen	No	Small	Yes	No	Yes	No	No	Yes	Yes
GOPAL	Elevated earinophili	56	Earinophilic	840	51to60yrs	Male	Literate	Low SES	Obese	2ndordor	Late onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
GOVINDAMMAL	Elevated earinophili	40	Earinophilic	450	31to40yrs	Female	Illiterate	Middle SES	Obese	2ndordor	Late onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
GOVINDARAJ	Elevated earinophili	28	Earinophilic	610	21to30yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
GOVRI	Elevated earinophili	38	Earinophilic	470	31to40yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
GUNAYATHY	Elevated earinophili	25	Earinophilic	520	21to30yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
HUMAYUN	Elevated earinophili	25	Earinophilic	610	21to30yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
INBARAJ	Elevated earinophili	20	Earinophilic	610	21to30yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
INDRANI	Elevated earinophili	45	Earinophilic	450	41to50yrs	Female	Literate	Middle SES	Overweight	1ntordor	Late onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
ISMAYIL	Elevated earinophili	28	Earinophilic	550	21to30yrs	Male	Illiterate	Middle SES	Overweight	1ntordor	Early onset	No	No	Not applicable	Yes	Yes	Mother	Animals	No	Large	Yes	No	Yes	No	No	Yes	Yes
JANSI	Elevated earinophili	15	Earinophilic	410	16to20yrs	Female	Illiterate	Middle SES	Normal	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
JAYA	Elevated earinophili	25	Earinophilic	460	21to30yrs	Female	Illiterate	Middle SES	Obese	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
JAYABARATI	Elevated earinophili	14	Earinophilic	460	16to20yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
JAYAPAL	Elevated earinophili	30	Earinophilic	520	21to30yrs	Male	Illiterate	Middle SES	Normal	1ntordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
JENIFER	Elevated earinophili	18	Earinophilic	610	16to20yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
JOTHI	Elevated earinophili	29	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Overweight	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
KALYANI	Elevated earinophili	30	Earinophilic	450	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
KALYANI	Elevated earinophili	26	Earinophilic	460	21to30yrs	Female	Illiterate	Low SES	Overweight	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
KAMALA	Elevated earinophili	24	Earinophilic	460	21to30yrs	Female	Illiterate	Low SES	Normal	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
KANNAMMA	Elevated earinophili	36	Earinophilic	470	31to40yrs	Female	Illiterate	Middle SES	Overweight	1ntordor	Early onset	No	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
KANNAMMAL	Elevated earinophili	36	Earinophilic	300	31to40yrs	Female	Illiterate	Low SES	Overweight	1ntordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
KANNAN	Elevated earinophili	30	Earinophilic	530	21to30yrs	Male	Illiterate	Low SES	Normal	2ndordor	Early onset	No	Yes	Not applicable	No	No	Not applicable	Environmental dust	No	Small	Yes	No	No	No	Yes	No	Yes
KANNAN	Elevated earinophili	20	Earinophilic	610	21to30yrs	Male	Illiterate	Middle SES	Normal	2ndordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
KANNIGA	Elevated earinophili	24	Earinophilic	410	21to30yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	No	Yes	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
KARUPPAN	Elevated earinophili	45	Earinophilic	530	41to50yrs	Male	Literate	Low SES	Obese	1ntordor	Late onset	Yes	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
KASTHURI	Elevated earinophili	32	Earinophilic	620	31to40yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
KATHIRESAN	Elevated earinophili	25	Earinophilic	610	21to30yrs	Female	Illiterate	Low SES	Normal	2ndordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
KAVITHA	Elevated earinophili	18	Earinophilic	990	16to20yrs	Female	Illiterate	Low SES	Overweight	1ntordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
KAYAL	Elevated earinophili	15	Earinophilic	610	16to20yrs	Female	Illiterate	Low SES	Normal	2ndordor	Early onset	not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
KIRUBA	Elevated earinophili	17	Earinophilic	520	16to20yrs	Female	Illiterate	Middle SES	Normal	1ntordor	Early onset	not applicable	No	No	No	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
KUMAR	Elevated earinophili	21	Earinophilic	610	21to30yrs	Male	Illiterate	Middle SES	Overweight	1ntordor	Early onset	No	No	Yes	No	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
KUMARI	Elevated earinophili	38	Earinophilic	470	31to40yrs	Female	Illiterate	Low SES	Normal	2ndordor	Early onset	not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes



	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB
95	KUMARI	Elevated eosinophilic	35	Eosinophilic	470	31to40 yrs	Female	Illiterate	Low SES	Normal	Underdor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
96	LAKSHMI	Elevated eosinophilic	18	Eosinophilic	410	19to20 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	No	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
97	LALITHA	Elevated eosinophilic	35	Eosinophilic	470	31to40 yrs	Female	Illiterate	Middle SES	Overweight	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
98	LATHA	Elevated eosinophilic	25	Eosinophilic	510	21to30 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
99	LILLY	Elevated eosinophilic	36	Eosinophilic	450	31to40 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
100	MALLIGA	Elevated eosinophilic	30	Eosinophilic	450	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
101	MANI	Elevated eosinophilic	38	Eosinophilic	530	31to40 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
102	MANI	Elevated eosinophilic	26	Eosinophilic	420	21to30 yrs	Male	Illiterate	Middle SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
103	MARY	Elevated eosinophilic	40	Eosinophilic	450	31to40 yrs	Female	Illiterate	Low SES	Normal	Underdor	Late onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
104	MARYPREMA	Elevated eosinophilic	40	Eosinophilic	460	31to40 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Late onset	Not applicable	Yes	Yes	Yes	Yes	Mother	Nil	No	Large	Yes	No	Yes	No	No	No	Yes
105	MERCY	Elevated eosinophilic	21	Eosinophilic	550	21to30 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
106	MOHAN	Elevated eosinophilic	24	Eosinophilic	410	21to30 yrs	Male	Illiterate	Middle SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
107	MULUMATHI	Elevated eosinophilic	20	Eosinophilic	410	19to20 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
108	MURALI	Elevated eosinophilic	27	Eosinophilic	410	21to30 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
109	MURALI	Elevated eosinophilic	26	Eosinophilic	710	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
110	MURTHY	Elevated eosinophilic	30	Eosinophilic	530	21to30 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
111	MURUGAN	Elevated eosinophilic	48	Eosinophilic	840	41to50 yrs	Male	Illiterate	Low SES	Overweight	Intordor	Late onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
112	MUTHU	Elevated eosinophilic	26	Eosinophilic	410	21to30 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
113	NAGARAJ	Elevated eosinophilic	48	Eosinophilic	840	41to50 yrs	Male	Illiterate	Low SES	Overweight	Underdor	Late onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
114	NARESH	Elevated eosinophilic	21	Eosinophilic	410	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
115	NIRMALA	Elevated eosinophilic	19	Eosinophilic	420	19to20 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
116	NITHYA	Elevated eosinophilic	12	Eosinophilic	410	19to20 yrs	Female	Illiterate	Middle SES	Normal	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
117	PADMA	Elevated eosinophilic	30	Eosinophilic	460	21to30 yrs	Female	Illiterate	Middle SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
118	PADMAVATHY	Elevated eosinophilic	30	Eosinophilic	470	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
119	PADMINI	Elevated eosinophilic	30	Eosinophilic	450	21to30 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	No	No	No	Yes	Yes
120	PANEERSELVAM	Elevated eosinophilic	45	Eosinophilic	410	41to50 yrs	Male	Illiterate	Middle SES	Overweight	Underdor	Late onset	Yes	Yes	Yes	Yes	No	Not applicable	Pollen	No	Small	No	Yes	No	No	No	Yes	No
121	PRASANNAKUMAR	Elevated eosinophilic	30	Eosinophilic	510	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
122	PRIYA	Elevated eosinophilic	13	Eosinophilic	510	19to20 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
123	RAJA	Elevated eosinophilic	25	Eosinophilic	550	21to30 yrs	Male	Illiterate	Middle SES	Normal	Intordor	Early onset	No	No	Not applicable	Yes	No	Mother	Animals	No	Large	Yes	No	Yes	No	No	Yes	Yes
124	RAJALAKSHMI	Elevated eosinophilic	37	Eosinophilic	470	31to40 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
125	RAJAMMAL	Elevated eosinophilic	40	Eosinophilic	440	31to40 yrs	Female	Literate	Low SES	Obese	Intordor	Late onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
126	RAJATHI	Elevated eosinophilic	32	Eosinophilic	550	31to40 yrs	Female	Illiterate	Middle SES	Overweight	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
127	RAJESHWARI	Elevated eosinophilic	20	Eosinophilic	460	21to30 yrs	Female	Illiterate	Low SES	Normal	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
128	RAMESH	Elevated eosinophilic	26	Eosinophilic	410	21to30 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
129	RANI	Elevated eosinophilic	30	Eosinophilic	470	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
130	RANI	Elevated eosinophilic	35	Eosinophilic	470	31to40 yrs	Female	Illiterate	Middle SES	Normal	Underdor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
131	RANI	Elevated eosinophilic	17	Eosinophilic	510	19to20 yrs	Female	Literate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
132	RAVI	Elevated eosinophilic	37	Eosinophilic	630	31to40 yrs	Male	Illiterate	Middle SES	Overweight	Intordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
133	RUBINI	Elevated eosinophilic	17	Eosinophilic	530	19to20 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	No	Yes	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
134	SAMPATH	Elevated eosinophilic	21	Eosinophilic	410	21to30 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
135	SANDHYA	Elevated eosinophilic	38	Eosinophilic	450	31to40 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
136	SARADA	Elevated eosinophilic	24	Eosinophilic	840	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
137	SARASWATHI	Elevated eosinophilic	32	Eosinophilic	550	31to40 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
138	SARASWATHI	Elevated eosinophilic	25	Eosinophilic	1200	21to30 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	No	Yes	Yes	Mother	Pollen	No	Small	Yes	Yes	Yes	Yes	Yes	Yes	Yes
139	SARAVANAN	Elevated eosinophilic	19	Eosinophilic	410	19to20 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
140	SASI	Elevated eosinophilic	33	Eosinophilic	530	31to40 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
141	SATHYA	Elevated eosinophilic	24	Eosinophilic	620	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Father	Food	Yes	Small	Yes	No	Yes	No	No	Yes	Yes
142	SELVARAJ	Elevated eosinophilic	42	Eosinophilic	520	41to50 yrs	Female	Literate	Middle SES	Obese	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes



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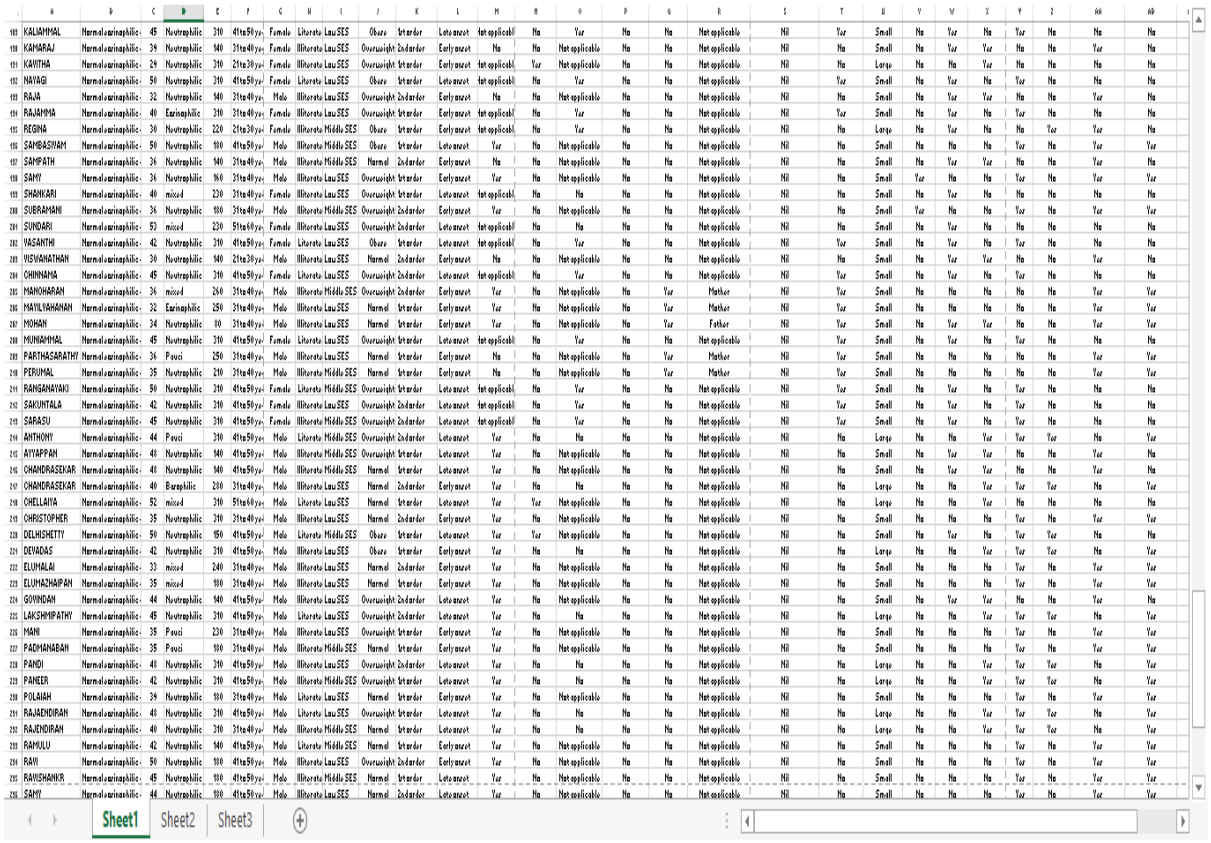
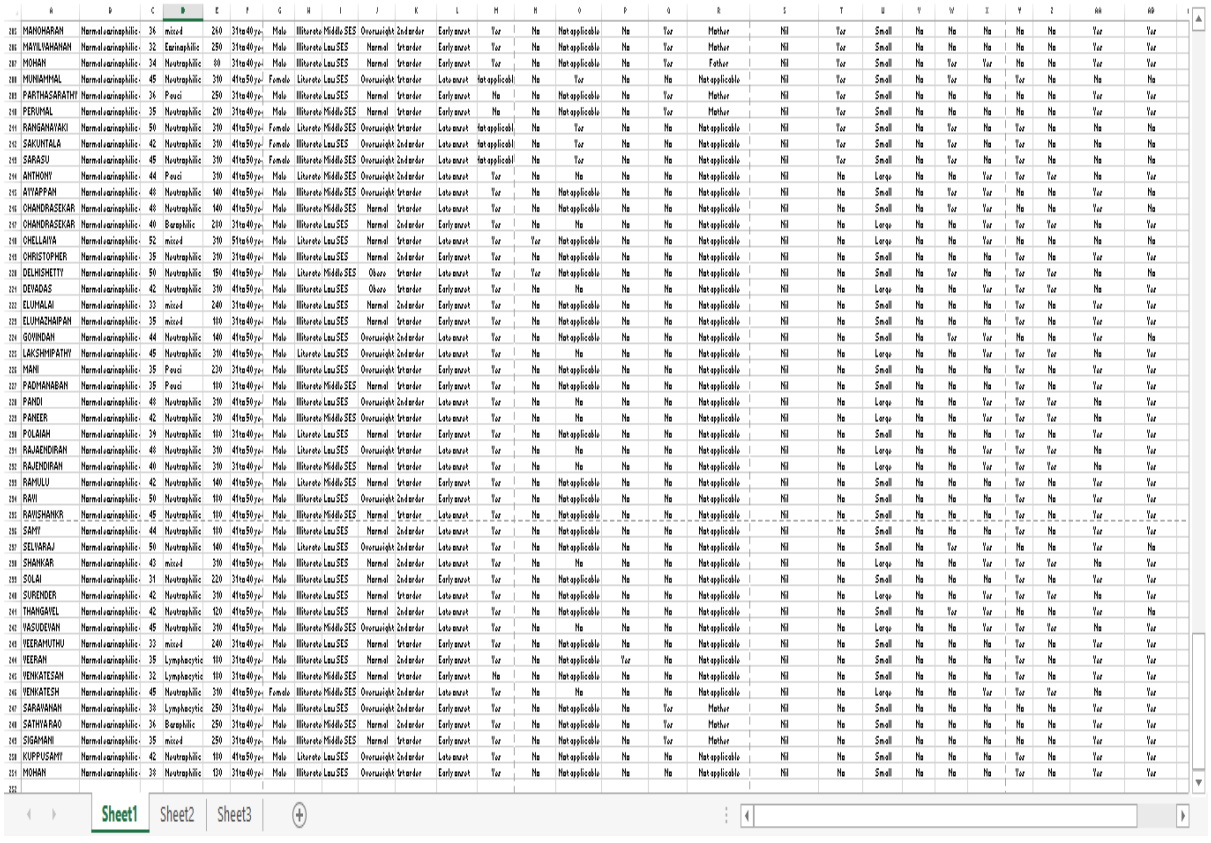
142	SELVARAJ	Elevated eosinophilic	42	Eosinophilic	520	41to50 yrs	Female	Illiterate	Low SES	Obese	Intordor	Early onset	Yes	No	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
143	SELVI	Elevated eosinophilic	25	Eosinophilic	410	21to30 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	No	Yes	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
144	SELVI	Elevated eosinophilic	30	Eosinophilic	450	21to30 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	No	No	Yes	No	No	Yes	Yes
145	SHAKILA	Elevated eosinophilic	38	Eosinophilic	490	31to40 yrs	Female	Illiterate	Middle SES	Normal	Underdor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	No	No	Yes	No	No	Yes	Yes
146	SHANKAR	Elevated eosinophilic	40	Eosinophilic	650	31to40 yrs	Male	Illiterate	Middle SES	Overweight	Underdor	Early onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
147	SHANKARI	Elevated eosinophilic	23	Eosinophilic	410	21to30 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
148	SHANMUGAM	Elevated eosinophilic	26	Eosinophilic	610	21to30 yrs	Male	Illiterate	Middle SES	Normal	Underdor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
149	SHANMUGANADAR	Elevated eosinophilic	28	Eosinophilic	610	21to30 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
150	SHANMUGAVEL	Elevated eosinophilic	27	Eosinophilic	610	21to30 yrs	Male	Illiterate	Middle SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
151	SHANTA	Elevated eosinophilic	33	Eosinophilic	450	31to40 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
152	SHARMILA	Elevated eosinophilic	23	Eosinophilic	530	21to30 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	No	Yes	Yes	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
153	SHARMILA	Elevated eosinophilic	23	Eosinophilic	510	21to30 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Early onset	Not applicable	No	Yes	No	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
154	SHERIF	Elevated eosinophilic	35	Eosinophilic	660	31to40 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
155	SIVAKARPAGAM	Elevated eosinophilic	25	Eosinophilic	610	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
156	SOUMYA	Elevated eosinophilic	35	Eosinophilic	460	31to40 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
157	SOWMYA	Elevated eosinophilic	15	Eosinophilic	510	19to20 yrs	Female	Illiterate	Low SES	Normal	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
158	SRILATHA	Elevated eosinophilic	19	Eosinophilic	510	19to20 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
159	STELLA	Elevated eosinophilic	25	Eosinophilic	510	21to30 yrs	Female	Illiterate	Middle SES	Normal	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
160	SUBRAYAN	Elevated eosinophilic	45	Eosinophilic	840	41to50 yrs	Male	Illiterate	Low SES	Overweight	Intordor	Late onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
161	SUDHA	Elevated eosinophilic	26	Eosinophilic	510	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
162	SUMITHRADEVI	Elevated eosinophilic	15	Eosinophilic	510	19to20 yrs	Female	Illiterate	Low SES	Normal	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
163	SUNDARAMOORTH	Elevated eosinophilic	42	Eosinophilic	530	41to50 yrs	Male	Illiterate	Low SES	Normal	Intordor	Early onset	Yes	No	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
164	SUNDARI	Elevated eosinophilic	28	Eosinophilic	420	21to30 yrs	Female	Illiterate	Low SES	Normal	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
165	SURESH	Elevated eosinophilic	28	Eosinophilic	610	21to30 yrs	Male	Illiterate	Middle SES	Normal	Underdor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
166	SURYAMOORTHY	Elevated eosinophilic	18	Eosinophilic	610	19to20 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
167	SUSILA	Elevated eosinophilic	43	Eosinophilic	450	41to50 yrs	Female	Literate	Low SES	Overweight	Underdor	Late onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
168	SYED BASHA	Elevated eosinophilic	50	Eosinophilic	840	41to50 yrs	Male	Illiterate	Low SES	Obese	Intordor	Early onset	No	Yes	No	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
169	THENMOZHI	Elevated eosinophilic	20	Eosinophilic	460	19to20 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
170	UDHAYAKUMAR	Elevated eosinophilic	32	Eosinophilic	530	31to40 yrs	Male	Illiterate	Low SES	Overweight	Intordor	Early onset	No	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
171	UMASHANKARI	Elevated eosinophilic	21	Eosinophilic	510	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	No	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
172	VAISHNAVI	Elevated eosinophilic	25	Eosinophilic	740	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Small	Yes	No	Yes	No	No	Yes	Yes
173	VALLI	Elevated eosinophilic	15	Eosinophilic	460	19to20 yrs	Female	Illiterate	Low SES	Overweight	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
174	POOVALLI	Elevated eosinophilic	35	Eosinophilic	470	31to40 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	No	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	No	Yes	Yes
175	VASANTHASEKAR	Elevated eosinophilic	55	Eosinophilic	950	51to60 yrs	Male	Illiterate	Low SES	Overweight	Intordor	Late onset	Yes	No	Yes	Yes	Yes	Father	Animals	No	Large	Yes	No	No	No	No	Yes	Yes
176	VASUKI	Elevated eosinophilic	29	Eosinophilic	660	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Nil	No	Small	Yes	Yes	No	No	No	Yes	Yes
177	VELU	Elevated eosinophilic	34	Neutrophilic	530	31to40 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Yes	Yes	Not applicable	Yes	No	Not applicable	Environmental dust	No	Small	Yes	No	Yes	No	Yes	No	Yes
178	VIJAYA	Elevated eosinophilic	38	Eosinophilic	450	31to40 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
179	VIJI	Elevated eosinophilic	30	Eosinophilic	450	21to30 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
180	VIJAYALAKSHMI	Elevated eosinophilic	30	Eosinophilic	550	21to30 yrs	Female	Illiterate	Low SES	Normal	Intordor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	No	Yes	Yes
181	VIJI	Elevated eosinophilic	25	Eosinophilic	650	21to30 yrs	Female	Illiterate	Low SES	Overweight	Underdor	Early onset	Not applicable	No	Yes	Yes	Yes	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
182	YOGALAKSHMI	Elevated eosinophilic	16	Lymphocytic	410	19to20 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	No	No	Mother	Nil	No	Small	Yes	No	Yes	No	No	Yes	Yes
183	LAKSHMI	Elevated eosinophilic	16	Eosinophilic	510	19to20 yrs	Female	Illiterate	Middle SES	Normal	Intordor	Early onset	Not applicable	No	Yes	No	No	Mother	Food	No	Large	Yes	No	Yes	No	Yes	Yes	Yes
184	AIYYADURAI	Normal eosinophilic	45	Neutrophilic	940	41to50 yrs	Male	Literate	Middle SES	Overweight	Intordor	Late onset	Yes	No	Not applicable	Yes	No	Not applicable	Nil	No	Small	No	Yes	No	No	No	Yes	No
185	ELLAMMAL	Normal eosinophilic	40	Neutrophilic	310	31to40 yrs	Female	Literate	Middle SES	Obese	Intordor	Late onset	Not applicable	No	Yes	No	No	Not applicable	Nil	Yes	Small	No	Yes	No	Yes	No	No	No
186	GLADIS MARY	Normal eosinophilic	52	mixed	220	51to60 yrs	Female	Literate	Low SES	Obese	Intordor	Late onset	Not applicable	No	No	No	No	Not applicable	Nil	No	Small	No	Yes	No	No	No	No	No
187	IRUSAN	Normal eosinophilic	50	Neutrophilic	940	41to50 yrs	Female	Illiterate	Low SES	Obese	Intordor	Late onset	Yes	No	Not applicable	No	No	Not applicable	Nil	No	Small	No	Yes	Yes	No	No	Yes	No
188	JOHN	Normal eosinophilic	40	Mixed	300	41to50 yrs	Male	Illiterate	Low SES	Overweight	Intordor	Late onset	No	No	No	No	No	Not applicable	Nil	No	Large	No	No	Yes	Yes	Yes	No	Yes
189	KALIAMMAL	Normal eosinophilic	45	Neutrophilic	310	41to50 yrs	Female	Literate	Low SES	Obese	Intordor	Late onset	Not applicable	No	Yes	No	No	Not applicable	Nil	Yes	Small	No	Yes	No	Yes	No	No	No





TURITIN ORIGINALITY CERTIFICATE