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Environmental Risk Factors of Bronchial Asthma among Primary Schoolchildren in Abu Dhabi City

Fareed Hussain Saleh H. Jalabi

United Arab Emirates University
Deanship of Graduate Studies
M.Sc. Program in Environmental sciences

ENVIRONMENTAL RISK FACTORS OF BRONCHIAL ASTHMA AMONG PRIMARY SCHOOLCHILDREN IN ABU DHABI CITY

By

Fareed Hussain Saleh H. Jalabi (M.B.B.S.)

A Thesis
Submitted to

United Arab Emirates University
in partial fulfillment of the requirements
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Supervisors:

Prof. Dr. Sayenna Uduman Faculty of Medicine and Health Science UAE University	Dr. Fatima Shad Kaneez Faculty of science UAE University

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The Thesis of Fareed Hussain Saleh H. Jalbi for the Degree of Master of Science in Environmental is approved.

31-5-2004

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Examining Committee Member, Dr. Fatima Shad Kaneez

31/5/04

.....

Examining Committee Member, Dr. Jose Joseph

31/5/2004

.....

Examining Committee Member, Prof. Phillip Williams

.....

Dean of the Graduate Studies, Dr. Hadef Rashed Al-Owais

United Arab Emirates University
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"So many people in our life come in and out, but very few of them leave foot prints, and change our destiny, you are one of them"

As all I wanted you to be here today, share me these sensitive moments and success, But no worry you never disappear, you where always the guide and the candle that brings me here.

To my grateful

the late Mr. Hussain Saleh teacher, friend and my father

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Abstract

The aim of this study was to describe the current epidemiological pattern of bronchial asthma among the schoolchildren aged 7-12 years old in 60 government primary schools at Abu Dhabi city –UAE.

The analytical cross-section design (ACSD) was chosen as a suitable method to investigate the exposure variables (environmental risk factors) and prevalence of bronchial asthma simultaneously in a representative samples.

A self-administered questionnaire was completed by the parents of the school children to collect information regarding bronchial asthma, other related allergic conditions and family history of respiratory allergy.

Analyses of the effect of the different socio-demographic variables and clinical history data of 3521 cases have been done. Out of which 503 (14.28%) asthmatic cases have been found. The socio-demographic data finding that father's educational level were preparatory education (35.8%) of asthmatic and (31.7%) of non-asthmatic cases with significant difference was found ($P<0.05$).

Asthmatic male were higher (54.67%) than non-asthmatic male (47.75%) and statistical significant difference was found to be ($P<0.05$). While asthmatic female were lower (45.53%) than non-asthmatic (52.2%) statistical significant difference was found ($P<0.05$).

Maternal asthma was higher among the asthmatic group (15%) than non-asthmatic (10.3%) a statistical significant difference was found between these two groups ($P<0.05$).

History of asthmatic father's (11.9%) had the strongest associations with childhood asthma than non-asthmatic (8.2%). and the difference was found to be ($P<0.05$).

Frequency of allergic rhinitis symptoms was significantly higher in asthmatic children (7.9%) than non asthmatic (1.8%) and their relationship was also significant difference ($P<0.05$). Indoor pollution (smoking)is a risk of development of bronchial asthma among the studied cases exhibited that father's smoking (56%) among the asthmatic and (37.9%) of non-asthmatic cases with a significant difference was found ($P<0.05$). The finding support the hypothesis that environmental risk factors, socio-demographic and family history may be had strong association factors of asthma.

The research had provided a good amount of quality data that can be of great advantage for school health programs as well as for research and development.

More comparative and community-based studies are needed to determine the differences among the various geographical areas of UAE and ethnic groups.

A detailed genetic study and a modified health education system are required in this region. Health care and school-health delivery system needs exhaustive assessment.

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LIST OF ABBREVIATIONS

AMP	Adenosine monophosphate
BCG	Attenuated bovine tuberculosis vaccine bacillus Calmette-Guérin
B-Eos	Eosinophil granulocyte in peripheral blood
BHR	Bronchial hyper-responsiveness
BMI	Body mass index
CI	Confidence interval
DDD	Defined daily dose
DNA	Deoxyribonucleic acid
ECP	Eosinophil cationic protein
ECRHS	European Community Respiratory Health Survey
ETS	Environmental tobacco smoke
FEV1	Forced expiratory flow in first second
FEV1%	Forced expiratory flow in first second as percentage of the predicted value
FVC	Forced vital capacity
GNP	Gross national product
IgE	Immunoglobuline E
ISAAC	International Study of Asthma and Allergy in Childhood
IUATLD	International Union Against Tuberculosis and Lung Disease
NO2	Nitrogen dioxide
OR	Odds ratio
PD20	Provocative dose causing 20%decline if FEV1
PGE2	Prostaglandin E2
RHINE	Respiratory Health in Northern Europe
RR	Relative Risk
SD	Standard deviation
SO2	Sulphur dioxide
SPT	Skin prick test
Thx	Lymphocyte class T helperx
VOC	Volatile organic compound vi
WHO	World Health Organization

SECTION (1):

INTRODUCTION

SECTION (1): INTRODUCTION

Bronchial Asthma is a substantial health problem among children and adults worldwide, with high and increasing prevalence rates in many countries (**Burney et al, 1990**), a substantial morbidity-reflected in hospital admission rates (**Halfon and Newacheck, 1986**), use of medical services (**Anderson HR, 1989**), and drug use (**Klaukka et al, 1991**)- and worrying trends in mortality rates in many countries (**Sears, 1992**).

The incidence of asthma varies by region and by age, but the burden of asthma can be approximated from measured prevalence rates (reflecting incidence, duration, persistence, and recurrence of disease), (**Sears, 1997**).

The determinants of asthma can be considered under the two general categories commonly used in epidemiology, namely environmental and host. Some determinants are modifiable, and have been the target for preventive measures; others such as age, race, and sex are not but are important because they modify the risk of developing asthma in response to environmental exposures in consistent ways and therefore affect the approach to diagnosis and management (**Becklake** and **Ernst, 1997**).

Von Mutius E (2000) clarified that many environmental factors have been proposed as risk factors for the development of childhood and adult-onset asthma. Yet in many instances results of different studies have yielded conflicting results or the precise relation between these risk factors and the disease has not clearly been established. Therefore, substantial uncertainty about identification of subjects at risk and effective prevention strategies remains.

However, carefully designed prevention studies might help to understand both the relative importance of different risk factors for the development of asthma and the benefit that can be gained by implementing these avoidance strategies.

Epidemiological studies are starting to provide information on the relevant environmental determinants and are for practical purposes the only way of identifying these determinants and the potential causes of asthma, and of evaluating the impact of preventive strategies, whether for those at high risk or at the population level. However, epidemiological studies are often limited by the need to use markers of asthma suitable for fieldwork, rather than the criteria necessary for clinical diagnosis (**Björksten, 1997** and **Burney, 1988**).

In spite of the efforts to improve asthma care that have taken place over the past decade, a majority of patients have not benefited from advances in asthma treatment and many lack even the rudiments of care. A challenge for the next several years is to work with primary health care providers and public health officials in various countries to design and evaluate asthma care programs to meet local Needs (**GINA, 1995**).

1.1. Definition of asthma

Many pulmonary diseases can be defined by their causative agent e.g., tuberculosis, or by defined pathology e.g., Squamous cell carcinoma, or by characteristic clinical presentation. For asthma, the primary cause remains unknown, pathology is rarely available at the time of diagnosis and the clinical presentation can be variable. Hence, to date, no universally acceptable definition has been formulated (**Sears, 1993**).

The term "asthma" encompasses a disparate group of disorders which produce similar clinical effects-that is, variable airflow obstruction -and this has formed the basis of the definition of asthma. Perhaps the most concise and useful description of asthma is: a clinical syndrome of "variable airflow obstruction."

Development of understanding the mechanisms of asthma, and desire to give a definition that would satisfy clinicians, physiologist, genetics, immunologists, molecular biologists, pharmacologists, epidemiologists as well as pathologists have added new components to the definition.

Current consensus definition of asthma has captured the history of development of understanding of the mechanisms of asthma in last decades and states:

Asthma is a chronic inflammatory disorder of the airways, in which many cells play a role, in particular mast cells, eosinophils, and T-lymphocytes. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, chest tightness, and cough, particularly at night and/or in the early morning. Theses symptoms are usually associated with widespread but variable airflow limitation that is at least partially reversible either spontaneously or with treatment. The inflammation also causes an associated increase in airway responsiveness to a variety of stimuli **(ICRDMA, 1992).**

This descriptive definition of asthma, however, cannot be translated into practical terms for epidemiological studies. Furthermore, despite the definition accepted for asthma, a diagnosis of asthma in clinical practice is made on the basis of combined information from history, physical examination, and respiratory function tests, often over a period of time. Time-consuming clinical testing is of little value in epidemiological setting because of low

response rates and possible non-comparability of methods across countries and regions. For these reasons, comparisons of asthma prevalence are increasingly being based on a simple comparison of symptom prevalence in a questionnaire survey of a large number of people, followed by more intensive testing of the underlying immunological and physiological components that often characterize clinical asthma, like bronchial hyper-responsiveness (BHR) and atopy, in a sub sample (**De Marco et al., 1998**).

1.2. Outcome variables in asthma epidemiology

Variable obstruction

Methods used in clinical diagnosing of asthma are difficult to apply in epidemiological settings, especially in cross sectional studies. The main reason for that is the variable nature of asthma. Verification of reversible airway obstruction, which is considered critical in establishing a clear clinical diagnosing of asthma (**ICRDMA, 1992** and **GINA, 1995**), would therefore underestimate the true prevalence, as reversible airway obstruction might not be revealed at time of testing.

Self reported asthma

Questionnaire based self reported asthma is often used in epidemiological studies. It has usually high specificity but low sensitivity to the actual disease, as all the cases of asthma are never diagnosed in the population (**De Marco et al, 1998**). This leads to an underestimation of the true prevalence of asthma.

Further problems arise, when different populations with differences in labeling of asthma and diagnostic practice are compared, as the differences in diagnostic practice may be as great in

magnitude as the real differences in asthma morbidity. A definition of high specificity is however important, when risk factors are estimated (**Pershagen, 1997**).

Symptoms of asthma

A number of symptoms, including wheezing, chest tightness, breathlessness, and coughing with or without sputum, are recognized by physicians as indicative of asthma. Symptom recording, based on standard postal and interview questionnaires, have also widely been used in epidemiological studies and can overcome some of the problems with diagnostic labeling. Symptom recording has, however, its own potential problems, arising from subjective symptoms recognition and recall, depending on variety of psychological, social and cultural characteristics, including healthcare practices, and also on the translation of the questionnaire (**Jögi, 2001**).

Those problems are evidently enhanced, when populations with different background regarding those factors are compared to give an example, the term "wheeze" that has been used most often in epidemiological studies for the identification of asthma, can not be easily translated into many languages, furthermore, the specificity of the symptom to asthma may differ in populations with different prevalence of other conditions that can cause wheeze, like chronic bronchitis or COPD. High international consistency has, however, been shown in answers to different questions in multilingual studies, indicating that international comparisons are not affected by errors due to cross-cultural variations in the reporting of symptoms (**Sunyer et al., 2000**).

To overcome cultural and language differences, video questionnaires have been introduced to asthma epidemiology studies. So far they have been used in children, and have shown good repeatability and slightly lower accuracy in detecting asthma (**Fuso et al, 2000**), but general good correlation with the results of the written questionnaires (**ISAAC, 1998**).

Bronchial hyper-responsiveness (BHR)

The main objective using BHR in asthma epidemiology studies is, to avoid problems of subjective symptom recall that may occur with symptom questionnaires. The results of BHR testing clearly depend on which agent or stimulus has been used for provocation (**Anto, 1998**).

The best-standardized methods are today available for direct stimuli, like histamine and methacholine that cause air-flow limitation by a direct action on the effectors cells involved in the air-glow limitation, such as airway smooth-muscle cells, bronchial vascular endothelial cells and mucus-producing cells (**Smith** and **McFadden, 1995**).

Comparisons between different delivery systems (**Chinn et al, 1993; Knox et al, 1991; Siersted et al, 2000**) and agents used (**Toelle et al, 1994**) have shown good correlation, so different methods using either histamine or methacholine, have been considered roughly equivalent (**Anto, 1998**).

As choosing a threshold is always arbitrary in defining if the subject is bronchially hyper-reactive or not, and to overcome the data loss in dichotomous analyses of the data, slope measures have been introduced that allow to get data for analyses from all participants in the study (**Chinn et al, 1993**).

Agents that act as specific constrictors in asthma through indirect stimuli via cellular activation or neural stimuli, such as inhaled adenosine 5'- monophosphate (AMP) are more closely associated with eosinophilic airway inflammation in asthma, but have so far been used sparsely in community studies (**Ludviksdottir et al, 2000** and **Van Den Berge et al, 2001**).

BHR testing was introduced to the asthma epidemiology studies at the beginning of the 1980s, based on early studies, where high sensitivity of BHR for diagnosed asthma was shown on selected groups of patients compared to healthy controls (**Townley et al, 1975** and **Toelle et al, 1992**).

Subsequent, population based validations, comparing both symptom questionnaires and BHR, with asthma defined on the basis of a clinical assessment by a physician, however, did not confirm those results (**De Marco et al, 1998; Jenkins et al, 1996**; **Pekkanen et al, 1999**). Thus, while the sensitivity of questionnaires for physician diagnosis of asthma was 80%in adults and 61% in children, and the specificity 97% and 94%, respectively, the sensitivity of BHR for physician diagnosed asthma was only 39% for adults and 54%for children (**Jenkins et al, 1996**).

The problems of the validity of BHR testing in asthma prevalence studies cannot be overcome by combining BHR with symptoms in defining asthma, which has also been suggested (**GINA, 1995** and **Toelle et al, 1992**).

A definition of asthma requiring both a positive questionnaire response and BHR was highly specific but not sensitive for adults (37%) or children (47%) (**Jenkins et al, 1996**).

The subject of validity of BHR testing in asthma prevalent studies has recently been summarized by **Pearce *et al* (2000)**, concluding that current evidence suggests that BHR testing has no greater validity (and may even have lesser) than symptom questionnaires for measuring the difference in asthma prevalence between populations with the same language and similar symptom recognition and reporting, but it may provide more comparable information when comparing populations which do not share these characteristics. BHR testing in epidemiological studies can, besides potentially lowering the response rate, introduce selection bias, as severe obstruction is usually considered a contraindication for provocation test and the most severe cases will therefore not be tested.

Thus, BHR testing cannot provide validation of the existence of differences in the prevalence of asthma between populations but can, however, be useful in terms of interpreting the findings of symptom prevalence questionnaire, showing to what extent the differences can be explained by BHR.

Other outcome variables

Although asthma and atopy are strongly associated, they also occur independently of each other (**Pearce et al, 1999**).

Thus, in asthma epidemiology studies, atopy is not a surrogate measure for asthma. It is both an associated condition (which is of interest in itself), and a risk factor for developing asthma. In the latter context, atopy can also be considered as an intermediate step in the causal pathways leading from allergen exposure to asthma, it may be an intermediate factor and a modifier of the effects of other exposures (**Pearce et al, 1998**).

Atopy is also strongly associated with BHR, but the two conditions often occur independently of each other. The number of eosinophil granulocytes in peripheral blood (B-Eos) is traditionally the most common method in assessing inflammation in epidemiological research (**Kauffmann et al, 1988; Annema et al, 1995; Burrows et al, 1991; Mensinga et al, 1990; Ulrik, 1998**).

More recently, measurements of eosinophil degranulation products, such as eosinophil cationic protein (ECP) in peripheral blood has been introduced as a method to assess and monitor inflammation in asthmatic patients (**Bousquet et al, 1998; Venge, 1995; Dahl, 1993**).

There is, however, limited experience in using these kinds of inflammatory markers in asthma epidemiological research (**Björnsson et al, 1994 and 1996**).

1.3. Increase in asthma prevalence

Data from epidemiological studies conducted in several countries worldwide have revealed that the prevalence of allergic conditions, including allergic rhinitis, asthma and eczema, have increased from the 1940s/50s to the 1990s (**Burr et al, 1989; Aberg, 1989; Robertson et al, 1991; Burney, 1990**).

Although the increase of asthma prevalence may, in part, be explained by altered diagnostic criteria and increased awareness of the disease, both in the general population and among physicians, these studies together indicate that the increased prevalence of asthma during the last decades is real. Trends of increasing prevalence of asthma have also been shown in many developed countries (**Lundback, 1993** and **Hansen et al, 2000**).

1.4. Regional differences in asthma prevalence

International and regional asthma prevalence comparisons are required as a key step in ascertaining the causes of asthma (**Beasley et al, 2000**).

The key issue in prevalence comparisons is, that the information is obtained in a comparable manner across all participating centers and countries, and that problems of translation of questionnaires and other problems of non-comparability of information are minimized. Two large scale epidemiological studies that allow comparison across several countries to fulfill these criteria: The European Community Health Survey (**ECRHS, 1996** and **Burney et al, 1994**) among adults and the International Study of Asthma and Allergy in Childhood (ISAAC), (**Asher et al , 1995**) .

The database of the ECRHS includes information from approximately 140,000 individuals from 22 countries. Based on analyses of the whole ECRHS data set, geographical variations in different outcome variables, i.e. diagnosed asthma, respiratory symptoms, atopic sensitization and bronchial responsiveness have been described (**ECRHS, 1996; Chinn et al, 1997; Burney et al, 1997; Janson et al, 1997**).

An eight-fold variation in the prevalence of wheeze, a four-fold variation in the prevalence of nasal allergy (**ECRHS, 1996**) and a six-fold variation in the prevalence of current asthma (**Chinn et al, 1997**) was found and the prevalence of all symptoms varied widely (**Janson et al, 1997**).

Although these were generally lower in northern, central and southern Europe and higher in the British Isles, New Zealand, Australia and the United States, there were wide variations even within some countries (**Burney et al, 1996**).

Analyses of bronchial responsiveness showed an eight-fold variation in BHR (PD20 <1 mg). The geographical distribution of bronchial responsiveness (ECRHS slope) and BHR fitted well with that for symptoms and asthma (**Chinn et al, 1997**).

A wide geographical variation was also found when investigating the prevalence of atopic sensitization (specific IgE), (**Burney et al, 1997**). A high prevalence was found in Australia, New Zealand, United Kingdom and United States, while the prevalence was low in Iceland, Greece, Norway, Italy and parts of Spain.

A five-fold.7 variation was found in the geometric mean of total IgE but there was no correlation between total IgE and atopic sensitization (**Burney et al, 1997**).

The ISAAC design comprises three phases. Phase One used simple core written questionnaires for two age groups, and was completed in 156 collaborating centers in 56 countries and a total of 721.601 children participated. In the 13-14 years age group 155 centers from 56 countries participated, of which 99 centers also completed a video questionnaire. For the 6-7 years age group there were 91 collaborating centers in 38 countries. The study has demonstrated a large variation in the prevalence of asthma symptoms in children throughout the world (**Asher et al, 1995**).

The prevalence of wheeze in the last 12 months ranged from 2-32% in the older age group and from 4-32% in the younger age group and was particularly high in English speaking countries and Latin America. A video questionnaire, completed in the older age group in 99 centers (42 countries), showed a similar pattern (**ISAAC, 1998** and **Asher, 1998**).

The prevalence data from the ISAAC study were compared to the data from the ECRHS (**Pearce et al, 2000**).The prevalence estimates in the ECRHS were consistently lower than the prevalence rates in the 13-14 years age group in the ISAAC study.

There was, however, generally a good correlation between the prevalence of wheeze and asthma in the ECRHS and the corresponding prevalence in the ISAAC study. Within country comparisons of urban and rural populations have shown lower prevalence of asthma in the latter, both in western (**Bråbäck et al, 1994**) and in developing countries (**Van Niekerk et al, 1979**).

In some cases the differences have been striking (**Yemaneberhan et al, 1997**)). Furthermore, comparing the lifetime prevalence of physician diagnosed asthma in Northern Europe first-year university students aged 18-24 according to childhood residence, revealed a significantly lower prevalence in those students who grew up in farm environments compared to those who grew up in urban or rural areas but not in a farm. No difference in asthma prevalence was found when students from urban and from rural non-farm environment were compared (**Kilpelainen et al, 2000**).

Prevalence studies have revealed three distinctive patterns: first, asthma is more prevalent in English-speaking countries; second, asthma is more common in Western countries than in developing countries and the difference is larger in later birth cohorts; third, the prevalence of asthma is lower in rural than in urban regions, especially among those, who have grown up in farm environment. These results also indicate that the current recognized risk factors for the development of asthma probably cannot fully account neither for the

worldwide increase in prevalence nor for the international variations in asthma prevalence (**Jögi, 2001**).

East-West difference in asthma prevalence

Western lifestyle has been accused for the increase in the prevalence of asthma and allergies. Prosperity in West European countries with market economy might have changed the environment to the extent, where the spread of possible disease modifiers have reached to the extent where it is not possible to evaluate their impact without comparing them with reference populations. At Germany comparison was carried out shortly after the reunification of Germany among 10-year old schoolchildren living in Leipzig, former East, and Munich, former West Germany (**von Mutius et al, 1992**). The study found lower prevalence of hay fever in the eastern part of Germany. The prevalence of asthma, defined as either doctor diagnosed asthma ever or recurrent wheezy bronchitis did not differ significantly between the two populations, while the doctor diagnosed chronic bronchitis was about twice as high in Leipzig.

The prevalence of wheeze was similar and there was no difference in the prevalence of BHR measured by cold air challenge. In a subsequent study, the same population in Munich was compared to a bigger random sample from Leipzig and Dresden. Atopic sensitization was estimated by skin prick testing, current asthma, and life long incidence of asthma was reported. In this study the prevalence of asthma, hay fever, atopic sensitization and BHR was lower in Leipzig and Dresden, while the prevalence of respiratory symptoms and chronic bronchitis was higher in Leipzig and Dresden than in Munich (**Von Mutius et al, 1994**).

Other prevalence comparisons in the beginning of the 1990s between previously socialist countries in Eastern Europe and industrialized countries have constantly shown lower prevalence of atopic sensitization and hay fever, both among children (**Riikjärv et al, 1995; Bråbäck et al, 1995; Schafer et al, 1996; Duhme et al, 1998**) and adults (**Nowak et al, 1996** and **Heinrich et al, 1998**).

The difference in sensitization between the centers has been found to decrease with increasing age (**Nicolai et al, 1997** and **Heinrich et al, 1998**), indicating a cohort effect, i.e. the causative environmental factors would be operating mainly in early childhood.

There is at least one presented exception, where no east-west difference in the prevalence of atopic sensitization was revealed in the study conducted in the year 1991.In this study five-to seven-year-old children from Eastern and Western Germany were tested by a multi-puncture device (**Schafer et al, 1996**).

The reasons for the East-West differences in atopy and related respiratory diseases have remained mostly un-explained. In most cases neither personal nor environmental risk factors could provide satisfactory explanations for the area differences in symptoms, BHR (**Nowak et al, 1996** and **Vasar et al, 1996**) or atopic sensitization (**von Mutius et al, 1994** and **Nicolai et al, 1997**), as the differences in prevalence have remained statistically significant after allowing for confounding factors.

In some reports the observed prevalence differences in atopy and respiratory allergies have partly been related to factors like "wood or coal heating" (**Duhme et al, 1998** and **Nowak, et al 1996**) or "reported family history of atopy" (**Duhme et al, 1998**), that can also serve as surrogate measures for some environmental factors not measured.

The published data do not support the view that the differences in sensitization are caused by differences in the exposure to specific allergens (**Hirsch, 1999**).

The east-west difference in the prevalence of respiratory symptoms and chronic bronchitis has been explained by higher prevalence of smoking and ambient air pollution with particulate matter and SO2 (**von Mutius et al, 1992** and **Kramer et al, 1999**).

Recent studies have shown some converging in the described differences. Thus, the prevalence of atopic sensitization and hay fever had increased in Leipzig after four years of unification (**von Mutius et al, 1998**) and no difference in the prevalence of atopic sensitization and hay fever among 9-11 year old children could be revealed between Leipzig and Munich (**Weiland et al, 1999**).

Similar trends have shown among adults. By the ECRHS study group in Germany a second stage questionnaire was sent out to a new random population sample 3-4 years after the previous one. The result of this study showed that the prevalence rates of wheezing, asthma attacks, asthma medication and allergic rhinitis were stable in Hamburg but increasing in Erfurt and approaching those of Hamburg (**Heinrich et al, 1998**). In a sub-sequent follow up the 2nd stage sample the prevalence of BHR was found to have increased in subjects living in Erfurt, while BHR remained unchanged in the group of subjects from Hamburg (**Richter et al, 2000**).

In Estonia two cross-sectional studies, the first in 1992-1993 (**Riikjärv et al, 1995**) and the second in 1996-1997 (**Riikjärv et al, 2000**), among 10-year-old children did not show significant increase in 12-month prevalence of wheeze, self reported asthma or atopic sensitization.

1.5. Factors that influence asthma

The prime consideration in asthma epidemiology studies is usually to obtain exposure information of similar accuracy for the groups being compared. Strictly speaking, "exposure" refers to the presence of a substance in the environment, whereas "dose" refers to the amount of substance that reaches susceptibility targets within the body. As personal dose measurements can often not be obtained, the term exposure has been used in the very general sense, including other attributes or agents that may be risk factors for asthma, like demographic and genetic factors (**Pearce et al, 1998** and **Jögi, 2001**).

Due to inherent variable nature of asthma, it is also usually not possible to distinguish, especially in cross sectional studies, if the factor studied is attributable to development of or exacerbation of asthma. However, major problems of morbidity, occurring through exacerbation and prolongation of asthma symptoms, can best be addressed by studying prevalence rather than incidence. Thus, if factors are found to be associated with asthma prevalence, then this is of major interest in itself, irrespective of whether the etiologic mechanism involves increase in asthma incidence or increase in duration (**Jögi, 2001**).

Age and Sex

The prevalence of wheeze and reported asthma is negatively related to age among adults (**Jarvis et al, 1994; Neukirch et al, 1995; Björnsson et al, 1994; Abramson et al, 1996**).

BHR declines with age in atopic non-smokers and increases with age in smokers (**Burney et al, 1987 & Chinn** and **Sunyer, 2000**).

Furthermore, many cases of adult-onset asthma can actually be the return of asthma symptoms in subjects who had asthma in childhood. In childhood males have a higher incidence of asthma than females, but in adolescence this reverses and between the ages 10-50 years females have a higher incidence than males. In later life the incidence possibly reverses once more (**Yunginger et al, 1992**).

This has also been reflected in prevalence studies (**Papageorgiou et al, 1997; Sunyer et al, 1997; de Marco et al, 2000**). In males, higher BHR has been observed in childhood-adolescence age groups and at older ages, while in females a higher level of BHR has been observed during adulthood (**Paoletti et al, 1995** and **Norrman et al, 1998**).

Those differences can be partly attributable to sex differences in lung growth and airway geometry (**de Marco et al, 2000 and Wassmer et al, 1997**), but may also be connected to the sex-related differences in the immune system.

Facilitation of ovule implantation and tolerance to the foetus (a semi allograft) are required for a successful pregnancy. A decrease in the rejection of potential allogens is particularly marked during pregnancy, but also occurs in general in women, during the childbearing age, and more particularly during the periovular phase of the menstrual cycle (**Chinn** and **Sunyer, 2000 & Kauffmann** and **Becklake, 2000**).

A positive correlation has been found in asthma incidence and hormone replacement therapy in postmenopausal women (**Troisi et al, 1995**).

Also, premenstrual worsening of asthma symptoms with increase in airway resistance has been reported (**Agarwal** and **Shah, 1997 & Chandler et al, 1997**).

Gender differences in the effects of environmental factors may be related to personal habits, like tobacco and alcohol consumption, the occupational environment, and the home environment. Women have been found to be more susceptible than men to environmental factors (**Leynaer et al, 1997; Jarvis et al, 1996; Jarvis et al, 1998; Jarvis, 1999**).

As it is, in general, socially acceptable for women to be breathless of effort and for men to bring up phlegm and snore, this can introduce recall bias and challenge the interpretation of results (**Kauffmann and Becklake, 2000**).

Genetic factors

Asthma appears to be multi-factorial in origin and influenced by multiple genes and environmental factors. A particular genetic factor may increase susceptibility to the effects of an environmental exposure and may thereby affect one or more aspects of the complex etiological process involved in asthma, but whether this genetic potential is expressed will depend on whether sufficient exposure to the environmental factor occurs.

Asthma genetic studies are further complicated by the difficulties in defining the asthma "phenotype" and by the fact that this phenotypic expression may vary with age. Despite the fact that genetic susceptibility to changing environmental exposures may play an important role in the changes in asthma prevalence, the considerable increase in asthma prevalence during the last decades indicates that genetic factors alone are unlikely to account for a substantial proportion of asthma cases. Major susceptibility genes for asthma and atopy have not been determined to date (**Jögi, 2001**).

Family concordance

People with family history of asthma are more likely to develop asthma themselves, and parental asthma is a stronger predictor of asthma in the offspring than parental atopy (**von Mutius and Nicolai, 1996**). This association, however, is not necessarily due to genetic factors, and could merely reflect similar life-styles and exposures in family members.

Twin concordance

Twin studies have shown the considerable genetic component of asthma. This component most likely consists of genes of additive effect (**Koppleman et al, 1999**). The probandwise concordance for asthma in monozygotic twins has been 38-52% and even this may in part be due to similar environmental exposures, including common intrauterine environment. Twin studies have also indicated that not only the shared, but also the individual specific environmental factors may be important as well (**Skadhauge et al, 1999**).

Most of the environmental risk factors are, however, shared by nature. Furthermore, two presumptions of the genetic studies using twin-designs, first, that twins are representative of the general population and second, that the environment for both monozygous and dizygous twins is similar, may not be totally valid. Thus, the shared intrauterine environment may have an adverse effect on the growth and organ maturation of the foetus and higher similarity in environment for monozygous compared to dizygous twins is likely (**Koppleman and Postma, 1999**).

Segregation and linkage analysis

Segregation analysis tests explicit models of inheritance in families –for example, by observing the frequency of the condition in offspring and siblings and comparing it to the distribution expected on the basis of various models of inheritance. Linkage analysis uses

DNA marker data in order to follow the transmission of genetic information between generations in order to determine if a genetic marker is linked to the gene involved in a particular disease. Attention has been particularly focused on chromosomes 5 and 11, both of which may contain genes relevant to asthma and atopy (**Jögi, 2001**).

Atopic sensitization

"Atopy" has previously been used as a poorly defined term to refer to allergic conditions that tend to cluster in families, including hay fever, asthma, atopic eczema, and other specific and non-specific allergic states. More recently, atopy has been characterized by the production of circulating IgE in response to common environmental allergens. Although atopy has sometimes been defined as "a genetic disposition" for this IgE response, most definitions focus on the production of IgE irrespective of the mechanism (genetic or environmental) by which it is produced (**Pearce et al, 1998**).

The commonly accepted hypothesis of the relationship between atopy and asthma is, that exposure of genetically susceptible individuals to allergen leads to the development of sensitization, and continued exposure leads to clinical asthma through the development of airway inflammation, BHR and reversible airway obstruction (**Pearce et al, 1999**).

The nature of the relationship between allergic sensitization to specific allergens and asthma can either be a direct causative one, with allergen exposure causing asthma in susceptible individuals, or an indirect one, where the genetically determined atopic diathesis causes both asthma and expression of sensitization (**Duffy et al, 1998**).

The association between atopy and asthma depends on the population studied. There is a considerable amount of studies showing an association between atopy and asthma

(Nowak et al, 1996; Sunyer et al, 1997; Chowgule et al, 1998; Wieringa et al, 1997; Abramson et al, 1996).

In a Finnish twin cohort, 262 twin pairs discordant for incident asthma were analyzed. The atopic twin had an increased risk of asthma compared with the non-atopic co-twin (RR 2.91,95%CI 1.81 to 4.68) **(Huovinen et al, 2001).**

There are, however, negative reports **(Peat et al, 1995)**, and inverse association between asthma and atopy has been reported **(Yemaneberhan et al, 1997).**

Reviewing the available epidemiological evidence on the association of asthma and atopy, Pearce et al (1999 and 2000) showed that the proportion of asthmatic and non-asthmatic subjects who are skin prick test positive vary considerably between different studies. The population attributable risk varied from 25% to 63% in children and from 8% to 55% in adults. The results were similar if specific or total IgE was used as an outcome. Furthermore, increase of the prevalence of atopic sensitization without increase in the prevalence of asthma 69, as well as increase in the prevalence of asthma with only minor changes in the prevalence of atopy 104 has been reported.

Allergens differ in their potency to cause asthma symptoms, thus pets are more potent than pollen **(Plaschke et al, 1999)**. The association between allergic sensitization and bronchial responsiveness has been studied on a combined ECRHS data set **(Chinn et al, 1999).**

Sensitization to mite, cat and timothy grass explained between 1.4 to 12.7% of the total variation in bronchial responsiveness in the different centers. The variation of bronchial responsiveness was better explained by taking account of all the individual allergens than by

classifying the subjects as atopic or non-atopic. Mite sensitization was the most important allergen in 15 centers, cat in 8, cat and mite equally in 1, timothy in 8 and Cla-dosporium in 2 centers. Total IgE has been showed to associate with BHR independently of specific IgE **(Chinn et al, 1999; ECRHS-Italy, 1998; Sunyer et al, 1996)**.

The attributable risk of atopic sensitization for BHR was found to be 19% in Sweden and 21% in Spain **(Plaschke et al, 1999)**.

Possibility has been proposed that asthmatics become sensitized disproportionately to those allergens that (because of particle size) are deposited in the inflamed/primed lower airway (so asthma causes sensitization). Bronchial hyper-responsiveness can in turn prevent the deposition of larger particles in the lower airways, thus prevent sensitization **((Jögi, 2001)**.

Furry pets

Positive correlation has been reported between the community prevalence of cat ownership and sensitization to cat, as well as respiratory symptoms and physician diagnosed asthma in young adults **(Roost et al, 1999)**, stressing the importance of cat allergens in developing and exacerbating asthma **(Plaschke et al, 1999; Gelber et al, 1993)**.

Children exposed to cat during the first year of life were, however, less often SPT positive to cat at 12-13 years and children exposed to pets during the first year of life had a lower frequency of allergic rhinitis at 7-9 years of age and of asthma at 12-13 years **(Hesselmar et al, 1999)**.

Analyses of the combined ECRHS data have shown a positive association between current cat ownership and specific sensitization in subjects reporting no respiratory symptoms associated with pet exposure. Subjects with symptoms associated with exposure to

pets were deliberately excluded from the analyses to avoid the selection bias in cat ownership. Having a cat in childhood was, however, negatively associated with cat sensitization in the same analysis in subjects with a family history of atopy, while this association was not found in subjects without such a family history. Based on the analyses of the same database, Svanes *et al.* reported that atopy was negatively associated with having a dog in childhood, but that there was no significant association with having a cat as a child, and atopy (**Svanes et al, 1999**).

The possibility, that higher allergen exposure had induced tolerance in those having a dog in childhood is unlikely, as having a dog in childhood was protective also against sensitization to other allergens. The finding may reflect pet avoidance by allergic families, however, there is no reason why dogs, but not cats, should be selectively avoided, as cats are often more potent to cause symptoms (**Plaschke et al, 1999**). It is therefore more likely, that some lifestyle factors confound the favourable effect of dog keeping, that are not present among cat keepers, or that more potent cat allergen overwhelms the potential protective effect.

Smoking

Smoking is a well-recognized and prevalent risk factor for respiratory symptoms (**Björnsson et al, 1994; Wieringa et al, 1997; Lindstrom et al, 2001; Vesterinen et al, 1988**) as well as BHR (**Burney et al, 1987; Paoletti et al, 1995; Norrman et al, 1998; Peat et al, 1992**).

The association between current smoking and asthma is less clear. Association between smoking and asthma has been found in cross-sectional (**Abramson et al, 1996**), also an

association between adult onset asthma (**Toren et al, 1999** and **Ronmark et al, 1997**) and severity of asthma (**Althuis et al, 1999**) has been found.

Many cross-sectional (**Björnsson et al, 1994; Gulsvik; 1979; Higgins et al, 1977**) and some longitudinal studies (**Vesterinen et al, 1988**) have, however, not found a clear association between smoking and asthma.

Furthermore, in a case control study of subjects from 16 countries, the risk of asthma with an onset within the last 3 years before the survey was found to be 43%lower in current smokers than in never smokers (**De Marco et al, 2000**).

However, in a three-year follow up study (**Plaschke et al,2000**) found that onset of asthma was more common in current smokers than in non-smokers, but this effect of smoking was mainly found in non-atopic subjects.

Similarly, (**Sunyer et al, 1997**) found, that smoking was associated with bronchial responsiveness only in subjects without atopy.

In the analysis of the combined ECRHS data set (**Jarvis et al, 1995**) reported a higher risk of sensitization to mite in smokers than in never smokers.

The risk of sensitization to grass and cat was lower in current and ex-smokers than in never smokers. Smoking has been found to be positively associated with total IgE 124-126 though less than 1%of the variation in total IgE could be explained by smoking (**Jarvis et al, 1995**).

Every risk factor that is voluntary, is selection biased to factors like social status, personal habits and awareness. Smoking in this sense is an extreme example. Unhealthy nutrition patterns have been found consistently higher in smokers than in non-smokers. Smokers consume more saturated fat, more alcohol, more cholesterol, less fruit, and fewer

vegetables, more fried foods, less fiber, less antioxidant vitamins, more salt. Smokers have less knowledge on how to improve risk and lower intention to change (**Slama, 2000**).

Thus, part of the association or lack of association found between respiratory symptoms, asthma and smoking, can be attributed to other factors than smoking itself ((**Jögi, 2001**).

Environmental tobacco smoke (ETS)

Passive smoking is widespread and passive smokers are exposed to both side-stream and mainstream tobacco smoke, which contains many potent respiratory irritants. A considerable amount of studies have evaluated the effects of ETS on asthma in childhood (**Jaakkola, 2000**).

There is strong evidence for a causal role of ETS in the development of asthma in children and parental smoking is related to more severe prognosis of asthma at least until school age (**Cook and Strachan, 1999**).

Much less is, however, known on the relationship between ETS and asthma in adulthood. Six studies, one longitudinal (**Greer et al, 1993**), four cross-sectional (**Hu et al, 1997; Janson et al, 2001; Leuenberger et al, 1994; Ng et al, 1993**) and one nested case-reference study (**Thorn et al, 2001**) have addressed the role of ETS in induction of asthma in adults, and shown the excess risk of asthma from 10 to over 200% in young adults and from 40 to 60% in older age groups due to ETS exposure. ETS has also shown to worsen the existing asthma (**Jindal et al, 1994**), and impair the lung function (**Coultas, 1998**).

A recent paper, based on the analysis of the whole ECRHS data set showed that passive smoking in the workplace, but not at home, was significantly related to current asthma as well as all respiratory symptoms except attacks of breathlessness at rest (**Janson et**

al, 2001). A significant dose-related association to passive smoking was found for respiratory symptoms. ETS was also related to increase bronchial hyper-responsiveness.

Indoor environment

Importance of indoor environment is stressed by the fact that in developed countries people spend the major part of the time indoors. The data on the indoor environmental risk factors for asthma in adults is, however, limited. Physical characteristics of the indoor environment include temperature and humidity. The latter increases when ventilation is inadequate. Home dampness, reflected as damp stains or visible mould was shown to associate with cough and asthma in a population based study in adults in the Netherlands (**Brunekreef, 1992**).

The association between asthma, and both dampness and visible mould, was strong in crude analysis but was not statistically significant when two markers were combined and adjusted for active and passive smoking, indoor NO2 sources and educational level in multiple regression analyses. Self-reported mould growth at home has been associated with asthma in young adults in a selected population attending the smoking cessation programs (**Hu et al, 1997**), first year university students (**Kilpelainen et al, 2001**) and in a case control study of physician diagnosed asthma patients (**Williamson et al. 1997**). In the latter study the severity of asthma correlated with actual measures of total dampness in the dwelling.

In Sweden, current asthma was more prevalent in subjects living in damp dwellings and particularly so in those living in dwellings with dampness in the floor construction (**Norback et al, 1999**).

Dampness in the home during childhood has been associated with increased airway responsiveness in adolescence (**Nicolai et al, 1998**).

In a recent population based case-references study of adult onset asthma, (**Thorn et al, 2001**) reported increased risk to out-come in subjects who reported visible mould growth (OR 1.4-3.5), visible dampness and mould growth (OR 1.1-3.1) or who had a wood stove in their dwelling.

Homes abound in chemical air pollutants. Sources include unvented combustion appliances, smoking, building materials, carpets etc. In 1996 Jarvis and co-workers reported that in the UK, women who used gas cookers had an increased risk of wheeze and other asthma symptoms as well as lower lung function (FEV1 and FEV1/FVC) than women not using gas cookers (**Jarvis, 1999**).

No such association was found in men. In a subsequent analysis of the combined data set, Jarvis *et al.* reported that there was an overall association between the use of gas cookers and respiratory symptoms in women, but that this association varied considerably between the centers (**Von Mutius** and **Nicolai, 1996**).

In general there was a positive association between gas cooking and symptoms in most European centers but a negative association in Australia, New Zealand and the United States. The reason for this across country heterogeneity was not explained but could possibly be related to differences in the nature of the gas in different countries (**Jögi, 2001**).

Building materials can act as sources of formaldehyde and other volatile organic compounds (VOC). Nocturnal breathlessness has been shown to associate with higher levels of CO2, formaldehyde and total concentration of VOC (**Norback et al, 1995**).

An association between respiratory symptoms and living or working in a newly painted dwelling, and BHR and living in a dwelling with newly painted wood details or kitchen painting has been reported (**Wieslander et al, 1997**).

Björnsson *et al* (1995) have re-ported higher levels of house dust mite and airborne bacteria in houses of subjects with asthma related symptoms. This association was, however, not related to mite allergy as none of the subjects who lived in houses with measurable house dust mite levels were sensitized to house dust mite.

Outdoor environment

Air pollution is convincingly associated with many signs of asthma aggravation. The adverse effects of SO2, ozone, and particulate matter to asthma patients have been proved in many experimental studies (**Koenig, 1999**).

There is less evidence of effects of NO2 from experimental studies. However, a slight increase in daily admissions to hospital for asthma in adults has been seen in relation to increasing ambient levels of nitrogen dioxide (NO2) (**Sunyer et al, 1997**).

In adults no consistent effects of long-term exposure to ambient ozone concentrations on the development of asthma and atopy have been demonstrated (**von Mutius, 2000**).

If air pollution influences the prevalence of asthma, it has clearly not been the main driving influence over the last decades, as the main increase in asthma prevalence in Western countries has come at a time when air pollution has been considerably decreased, and Eastern Europe that had particularly bad air pollution had a markedly low prevalence of asthma. That, however, concerns mainly particulate matter and SO2. It has been argued that NO2,

diesel particles of other products associated with traffic pollution may be responsible for the West-East difference in the prevalence of asthma (**Jögi, 2001**).

Occupational exposures

Asthma is the most common occupational respiratory disorder in industrialized countries. Occupational asthma can be caused by several high and low molecular weight sensitizes, of which more than 250 have been identified (**Heederik, 2000**).

The determination of how many cases of asthma may be caused or worsened by occupational exposures is highly dependent on how asthma is defined, what constitutes work-relatedness, and what specific methodology is employed. The proportion of asthma attributed to occupational exposure has been estimated to be 5–10% (**Blanc** and **Toren, 1999; Kogevinas et al, 1999; Kogevinas et al, 1996; Fishwick et al, 1997**).

Asthma has been associated with high dose exposure to biological, and mineral dust as well as exposure to gases and fumes (**Kogevinas et al, 1999**).

A higher risk for asthma has been found among laboratory technicians, painters, plastic workers, cleaners and agricultural workers (**Kogevinas et al, 1996 and Fishwick et al, 1997**).

Socio-economic status

A high prevalence of asthma has been found in affluent Western countries. On the community level a higher prevalence of asthma has been shown in more affluent regions in developing countries (**Von Mutius, 2000** and **Heinrich et al, 1998**).

Higher paternal social class was also associated with increased asthma prevalence in a British 1970 birth cohort studied at 26 years of age 155, and in children who had wheezed by five

years of age, the persistence of symptoms at 16 years of age was independently related to high social status of the mother (**Lewis et al, 1995**).

Higher educational level was also a risk factor for clinical allergies among adults in East Germany (**Nicolai et al, 1997**). However, the more educated twin of 262 twin pairs discordant for incident asthma, had a decreased risk of asthma compared to twin sibling with less education 102, and asthma has shown to be more severe in patients from poorer background (**Mielck et al, 1996**).

Number of siblings, day-care attendance and infections

One temporal association with the increase in atopy in developed countries has been the use of antibiotics, widespread vaccination of children and the fall in exposure to helminthes as Tapeworms. In his recent review, (**Weiss, 2000**) concludes that there is no conclusive evidence that parasitic infections protect against asthma development.

The association with antibiotic use and asthma is difficult to study as the use of antibiotics is widespread and the predominant reason for prescription is prolonged respiratory symptoms after a viral infection, where a bacterial involvement in the disease is suspected. Misclassification with asthma in this situation is likely, leading to a non-casual positive association between asthma and antibiotic use (**Jögi, 2001**).

There are social groups where conventional treatments, including antibiotics, are deliberately rejected. One study, carried out on a selected group of children at Rudolf Steiner schools, showed 400% increase in asthma prevalence associated with use of antibiotics (**Wickens et al, 1999**).

There are, however, many other aspects of the anthroposophy lifestyle, which may be relevant and antibiotic use in this context, Nevertheless, the effects of antimicrobial therapy on bacterial colonization of the infant gut should also be regarded. Several studies have found negative associations of family size, and particularly with the number of older siblings (**Strachan, 2000, and Matricardi, 1998**) with atopy (**Bråbäck et al, 1995; Nowak et al, 1996; Matricardi et al, 1998; von Mutius et al, 1994**), hay fever (**Kilpelainen et al, 2000; Hesselmar et al, 1999**), asthma (**Sunyer et al, 1997; Hesselmar et al, 1999; Shaheen et al, 1999; Jarvis et al, 1997**) and BHR (**Chinn et al 1998**).

In the analysis of the whole ECRHS data set, atopy was negatively associated with family size, partly attributable to an independent protective effect of a greater number of brothers, but not older siblings. Bedroom sharing was associated with a lower prevalence of atopy, particularly to cat allergen (**Svanes et al, 1999**).

However, a protective effect of family size and bedroom sharing could only be detected in subjects reporting no parental allergy, indicating that in subjects with a strong genetic predisposition, environmental factors in childhood are possibly of less importance (**Jögi, 2001**).

Similarly, day-care attendance has been associated with decreased risk of atopy, but that only in children from families with up to three people. The association was higher among children who attended day-care earlier (**Kramer et al, 1999**) but an opposite association also has been reported (**Kilpelainen et al, 2000**), where day-care attendance at age 0-2 years was a weak risk factor for allergic rhinitis but not for asthma.

Furthermore, children who attend day-care have shown to increase risk of asthma with early infections as a probable mediator of risk (**Wickens et al, 1999**).

In Swedish young adults an increased risk of atopic sensitization was found in subjects who had attended a day-care center before the age of five years (**Plaschke et al, 1996**), but this association was not found to be significant when analyzing the combined ECRHS data set (**Svanes et al, 1999**). No association of adult asthma and day-care attendance has been reported.

Strachan, who first found the negative association between hay fever and number of older children in the household, suggested that small family size could reduce infectious diseases "transmitted by unhygienic contact with older siblings", but not respiratory viral infections in infancy, and that this could in turn increase the risk of atopic disease at older ages (**Strachan, 1989**).

This hypothesis has been developed further. The immunological bases of the hypothesis stand on T helper (Th) lymphocyte differentiation in maturation of the immune system. Th2-like cytokines (interleukin (IL) 4,IL-13, and IL-5) produced in the uterine environment are important to maintain the pregnancy. The continuation of foetal allergen-specific Th2 responses during infancy is associated with decreased capacity for production of the Th1 cytokine interferon ã by atopic neonates (**Prescott et al, 1999**).

According to the hygiene hypothesis, T-cell responses to the microbial and viral infections generate Th1-like cytokines such as IL-12 and interferon-ã that down-regulate Th2 responses, helping T-cell immune responses to mature into a balanced phenotype that would be less likely to favor allergen sensitization (**Holt et al, 1997**)

There is also more direct evidence that childhood exposure to infections might prevent atopic diseases. Less atopy (**Shaheen et al, 1996**) and asthma (**Bodner et al, 2000**) in those with history of measles and less atopy in those with hepatitis A (**Matricardi et al, 1997**) has been reported.

Furthermore, in a study of Italian male military students the prevalence of serum markers of microbes transmitted through the oral rout was higher in the non-atopic than in the atopic cadets, while presence of serum markers of the viruses transmitted by other routes was not associated with atopy (**Matricardi et al, 2000**).

Strongly positive tuberculin responses in early life were associated significantly with less asthma in later childhood in Japanese children (**Shirakawa et al, 1997**).

The positive tuberculin responses in early life were also associated with a dominance of lymphocyte class T helper (Th1) over Th2 in the blood cytokine profiles at 12 years of age. However, differences in immune responses and natural resistance in different subjects result in differences in handling both infections and potential allergens, and thus differences in the clinical expression of a disease. This difference in host characteristics is reflected also in differences in cytokine levels (**Jögi, 2001**).

The intensity of tuberculin responses in young adults receiving BCG at 14 years of age did not correlate with total serum IgE in the Norwegian population, nor did the positive tuberculin test distinguish the atopics from non atopics (**Omenaas et al, 2000**).

These two papers together indicates, that it is not the host characteristics in the Th lymphocyte balance, but rather the sub-clinical exposure to *M .Tuberculosis* or immunization

earlier in life that can result in decrease in atopy. The latter hypothesis has, however, been rejected by two Swedish studies on children (**Strannegard et al, 1998 and Alm et al, 1997**).

As a proxy of differences in natural exposure to *M. Tuberculosis*, WHO derived tuberculosis notification rates were matched to the prevalence of respiratory symptoms within the ISAAC study in ecological analyses. The results showed that increase in notification was negatively correlated with the prevalence of wheeze and asthma in 85 centers from 23 countries studied (**Von Mutius et al, 2000**).

A Finnish registry based study has shown reduced occurrence of subsequent asthma in women *with M. Tuberculosis* infection in child-hood, the relation was, however, inverse among men (**Von Hertzen, 1999**).

Thus, BCG vaccination once, either in infancy or adolescence, seems not to shift the Th1-Th2 balance. There is also no conclusive evidence that treated active tuberculosis before the age of 20 could do that. However, repeated contacts with mycobacteria at a certain age might have an influence on the immune system. This is supported by the results of **Von Mutius *et al* (1994, 1998, and 2000).**

Other possibility is that other aspects of lifestyle can confound these relations. While some kinds of infections may have a role in the maturation of the immune system in early childhood with consequences that can last to adulthood and determine the subsequent development of respiratory diseases (**von Mutius, 2000** and **Björkstén, 1997**), viral upper respiratory infections are a common trigger for episodes of bronchospasm and have been associated with 80%of all asthma exacerbations (**Nicholson et al, 1993 and Micillo et al, 2000**).

Prospective studies in children, where viral markers have been measured at clinical respiratory infection episodes in early childhood, have shown variable, but declining with age, association between respiratory virus infections in infancy, and frequent wheeze up to at least 13 years of age (**Sigurs et al, 2000 and Stein et al, 1999**).

The extent of the association depends on the severity of the disease episode. The strongest association has been found between respiratory syncytial virus (RSV) bronchiolitis in infancy, severe enough to cause hospitalization (**Sigurs et al, 2000**). A positive association with respiratory syncytial virus infection and BHR has been shown among adults (**Björnsson et al, 1996**). As no population based studies with serological proof and adult asthma as an outcome-are available, the causal role of viral infections in asthma has remained unclear. The role of bacterial infections in exacerbating asthma is more controversial A positive association with serological signs of a current or recent Chlamydia pneumonia infection and reported wheeze has been reported (**Björnsson et al, 1996**) but not confirmed by others 187(**Ferrari et al, 2000**).

Diet

There are different ways in which food could affect lung health. First, relative deficiencies of certain foods may alter the lung's defense mechanisms, making it more liable to develop disease. Second, food sensitivity may act through immunological or non-immunological mechanisms to cause bronchoconstriction (**Seaton et al, 2000**).

Third, changes from locally produced food, like fresh and fermented vegetables, and non-sterilized diary products to industrially prepared and processed foods and use of microwave ovens, can change microbial load and influence the gut micro flora. One of the first

observations on the association of general diet habits and asthma, and table salt were related to asthma morbidity statistics in Britain. This gave rise to the suggestion that salt intake is relevant for the risk of asthma (**Bureny1987**).

Later studies have not proved this and it has been suggested that salt intake can be a marker of generally poor diet (**Seaton et al, 2000 and Burney, 1987**).

Another early observation was, that low prevalence of asthma in Eskimos could be associated with their diet, rich in fish and marine mammals or, with a genetic deviation in fat metabolism in Eskimos, or both (**Horrobin, 1987**).

The increase of asthma prevalence has been subsequent to the fall in the consumption of animal fat and oily fish with high content of saturated fat and increase in the amount of margarine and vegetable oils, containing polyunsaturated fat, in the diet. It has been suggested, that an increase in the intake of fatty acids, such as linoleic acid, and a decrease in the intake of -3 fatty acids, such as eicosapentaenoic acid, may have led to an increase in allergic sensitization, which in turn may account for the increase in the prevalence of asthma. The reason, why a decreased consumption of -3 fatty acids may lead to increased prevalence of allergy may be the ability to inhibit the synthesis of arachidonic acid from linoleic acid and prostaglandin E2 (PGE2) from arachidonic acid. The ability of PGE2 to inhibit the productions of interferon would in turn lead to the immune modulation towards Th2 inflammatory reaction to allergens (**Black** and **Sharpe, 1997**).

Association between the intake of polyunsaturated fat and atopy has been shown in children (**Bolte et al, 2001 and Dunder et al, 2001**) and adults (**Heinrich et al, 2001**).

Epidemiological evidence to show the importance of these mechanisms in asthma is scarce. The association with asthma and dietary fat has been revealed in some studies in children (**Peat et al, 1992 and Hodge et al, 1996**), but not others (**Bolte et al, 2001**) and not among adults.

Thus, among young Norwegian adults, with an overall high fish intake, fish consumption was not a significant predictor of current respiratory symptoms or asthma (**Fluge et al, 1998**), similarly, no relation between the intake of various fats and asthma was found in the Nurses' Health Study (**Troisi et al, 1995**). However, improvement of asthma in children using fish oil capsules vs. capsules with olive oil in a double blind clinical trial in a strictly controlled environment has been shown (**Nagakura et al, 2000**). It has been suggested that intake of a mild anti-inflammatory agent, like fish oil, might matter more in children, than in adults (**Schwartz, 2000**). Acting as antioxidants, or through influences on immune function, vitamins C and E and beta-carotene in the diet may reduce airway inflammation, thereby decreasing the severity of asthma or preventing the expression of asthma in susceptible individuals. Vitamin E intake from diet was inversely associated with adult onset asthma in the prospective Nurses' Health Study. Thus, women in the highest quintile of daily vitamin E intake from diet alone had a 47% lower risk of asthma than women in the lowest quintile (**Troisi et al, 1995**). The effect of processed food consumption instead of eating locally produced food and food products and potential effect of the detail changes on gut micro-flora and the subsequent development of atopy and asthma has been less studied. However, studies comparing infants in Sweden and Estonia have shown marked differences in the types of faecal bacteria in unselected infants in the two countries, which broadly match the

differences seen between atopic and non-atopic infants in each country (**Björkstén et al, 1999** and **Sepp et al, 1997**). The differences included a more intensive colonization with lactobacilli and eubacteria in Estonian children, whereas the Swedish infants had increased numbers of clostridia, particularly Clostridium difficile, as well as bacteroides and other anaerobes (**Sepp et al, 1997**). In Britain and in the USA the rise in asthma has been accompanied by an epidemic increase in the prevalence of obesity, a characteristic related to diet, and often unbalanced diet. An independent association between increase of body mass index (BMI) and prevalence of current wheeze, chest tightness and breathlessness at night, after controlling for age, sex, occupational exposure, smoking and dietary fish consumption, has been relieved in young Norwegian adults (**Fluge et al, 1998**). The prevalence of asthma increased with BMI also among British adults studied at 26 years of age. The association between fatness and asthma was stronger among women (**Shaheen et al, 1999**).

In an Australian study among adults, it was found that severe obesity was associated with a higher prevalence of wheeze, diagnosed asthma and medication use. Despite the fact that FEV1 and FVC were significantly reduced in severely obese subjects, there was no increase in airway responsiveness to histamine among them. A low level of physical activity, clearly associated with higher BMI, can be a result of asthmatic individuals avoiding physical exercise. However, asthmatic children have been shown to be physically as active as non-asthmatic children (**Chen et al, 2001**).

Furthermore, it has been shown, that weight reduction has both short time and long-time favourable effects on lung function, and symptoms (**Stenius-Aarniala et al, 2000**).

SECTION (2):

AIMS AND OBJECTIVES

SECTION (2): AIMS AND OBJECTIVES

2.1. General Objective:

The study aimed to describe the current epidemiological pattern of bronchial asthma among the school-aged children in Abu Dhabi, UAE, in order to provide some essentially needed information to the authority bodies (MOH) and school health decision makers for their utilization in the development of an efficient school health program based on quality field information.

2.2. Specific Objectives

The specific objectives of the study were to:

i. Determine the prevalence of bronchial asthma among the school-aged children in Abu Dhabi area of the United Arab of Emirates.

ii. Identify the major environmental risk factor for the school-aged bronchial asthma.

iii. Estimate the degree of association between bronchial asthma and environmental risk factors.

iv. Measure the relationship between bronchial asthma and the other pertinent allergic and hereditary diseases e.g. allergic trinities and eczema.

v. Find out the effect of indoor pollution on the severity of bronchial asthma with particular emphasizing on passive smoking.

vi. Compare the findings with other relevant studies conducted in the Arab Gulf area and other countries with similar socio-economic and environmental risk indication .

SECTION (3):

MATERIAL AND METHODS

SECTION (3): MATERIAL AND METHODS

3.1. Research Strategy:

The cross-sectional analytical research design was chosen as a suitable method to investigate the current research issue. It has the advantage of studying the exposure variables (environmental risk factors) and existing bronchial asthma (prevalence) simultaneously in a representative sample. The subjects of the study were the Primary Schoolchildren aged 7-12 years in Abu Dhabi City, UAE.

As a prerequisite for the proper research design a survey of literature and exploratory study will be carried out during the preparatory research phase.

The research process included the specification of the main research issue, defining the scope and measuring the study variables, specification of variables and indicators, the sampling procedures, and the procedures for quantification and combining the data into scores and indices.

The quality of the collected data was ensured through a variety of mechanisms. The collected data were computerized and sufficient descriptive and analytic statistical methods were made.

3.2. Research Setting:

The study was conducted in Abu Dhabi Emirate (**Figure, 1-A**), the capital of United Arab of Emirates (**Figure, 2-A**) at the school health delivery level. The following table (**Table, 1 A**) shows the main demographic, health and socio-economic indicators of UAE and some relevant indicator to the current study.

Table (1-A): Demographic and Health Indicators of United Arab of Emirates

ASPECT	Year	Source	Value
A. Population Indicators			
- Total Population (Millions)	2001	UNDP, 2003	2.9
- Crude Birth Rate - Crude Death Rate - Population Annual Growth Rate (%)		MOH, 2002	18.46 3.01 2.40
B. Health Indicators			
- Life Expectancy (Years) - Children Under Weight - Total fertility Rate - Access to Improved Sanitation (%)	2001 2001 2000 2001	UNDP, 2003	74.40 14 2.80 100
C. Educational Indicators			
- Adult Literacy Rate (%) - Combined 1ry, 2ry & 3ry School enrolment ratio - Education Index	2001	UNDP, 2003	76.60 67 0.73
D. Economic Indicators			
- GDP Per Capita (USS) - Long Term Un-employed (%) - Public Health Expenditure (% GDP)	2001	UNDP, 2003	20,530 00.00 2.50
E. Human Development Index (Rank)	2002	UNDP, 2003	48

Figure (1-A): United Arab of Emirates-Map

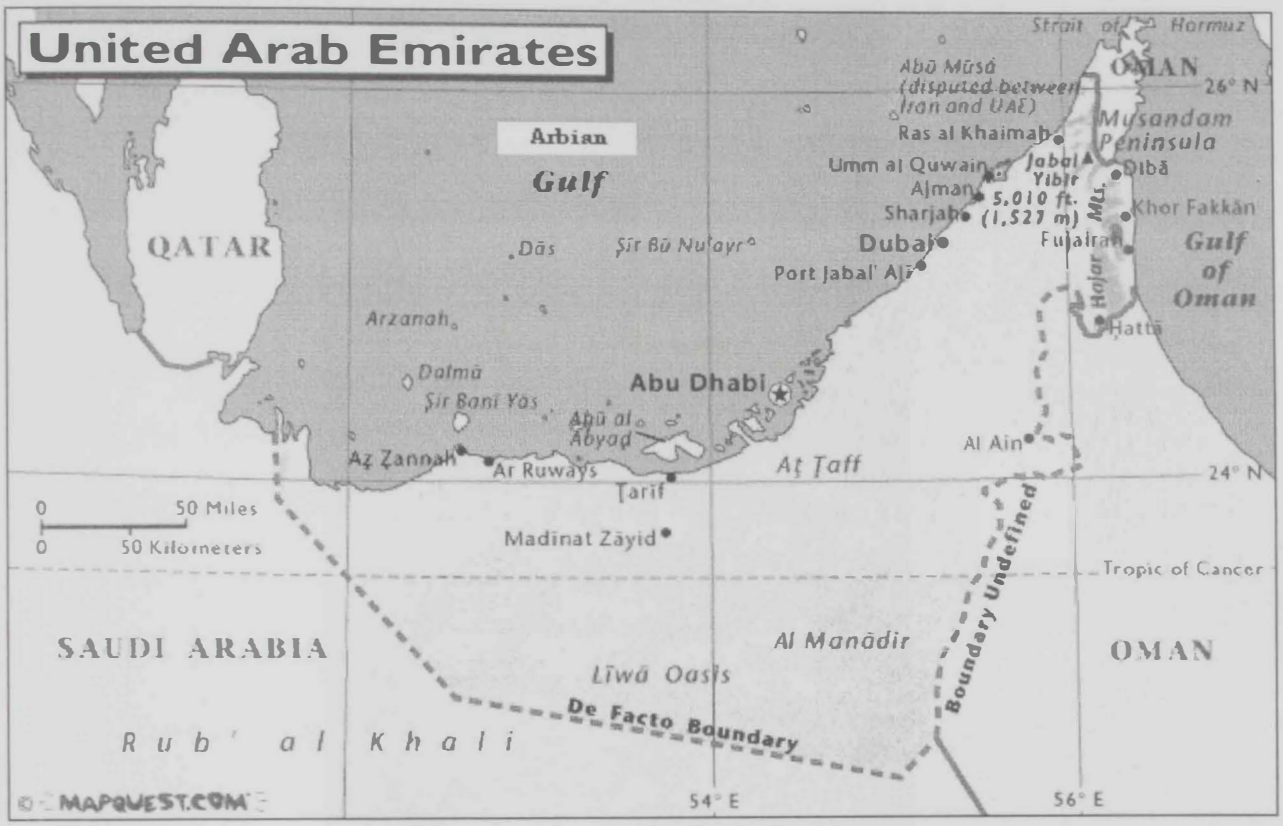
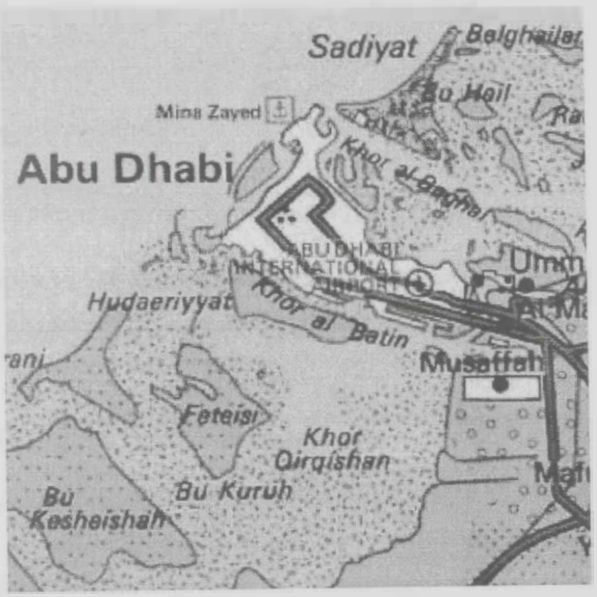


Figure (2): Abu Dhabi Emirate-Map



3.3. Literature Survey

A survey of literature was carried out during the preparatory research phase. The objectives of this part of the study were the followings:

i. To help in the proper understanding and clarification of the research problem.

ii. To review the different methodological approaches that might be used in the study.

The literature survey covered the following topics:

i. School and Chronic Diseases Health Services

ii. Bronchial Asthma.

iii. Different Methodological Approaches to investigate the present research issue.

iv. Health Care System in UAE.

The following resources were adequately reviewed:

i. Statistics collected at the national and functional that obtained from Ministry of Health (Central Authority) and School Health levels.

ii. Cards catalogues of books and journals in medical libraries.

iii. Bibliographies prepared for selective topics.

iv. Computer based literature search.

v. Responses from agencies and researchers interested in the present research topic e.g. WHO.

An important approach to identifying the literature and collecting information on the instruments and methods used, included Internet based search.

Search for online information, using the web browsers (Google, Netscape, etc..) was conducted for combinations of the key words: bronchial asthma, epidemiology, chronic

diseases, school health, cross-sectional studies, research methodology, survey, environment, risk factors.

Web sites of the respective environmental risks and health agencies, statistical offices, as well as research institutions were browsed as well.

Publications on methodological aspects of the health surveys and specific environmental health issues assessed were identified by the Medline based search (using the above key words) and from the lists of references available for some surveys concerning: survey design and instruments, availability of questionnaires, availability of sampling and measurement protocols, relevant publications

3.4. Research Design

The Analytical Cross-Sectional Design (ACSD) was chosen as a suitable method for the current research issue as it can investigate the environmental risk exposure factors and bronchial asthma simultaneously in a representative sample of the school-aged students.

Therefore, by taking a representative sample, it is possible to generalize the results obtained in the sample for the school-aged population as a whole in UAE.

Also, ACSD can measure the association between the exposure variables (environmental risk factors) and existing bronchial asthma (prevalence), unlike cohort studies, which measure the rate of developing disease (incidence). A common fact recognized the cross-sectional studies as a widely used tool for surveillance and research methods of collecting information not available through monitoring and routine statistics (**WHO, 2001**).

So, it's enabling the specific research problems to be identified and implementation of the relevant policies to be monitored. If planned accordingly, they may also detect and monitor the health status of the school-aged children.

The present design gave a special emphasis on the study questionnaire as a surveillance tool for further adaptation and utilization by MOH and relevant health organization in UAE.

The following are the main advantages of the chosen research design:

i. It has the great advantage over case-control studies of starting with a reference population from which the cases (bronchial asthma) and controls were drawn.

ii. It has s short-term in conduction, and therefore less costly than prospective studies.

iii. It is the starting point in prospective cohort studies for screening out already existing conditions.

iv. It will provide a good amount of quality data that can be of great use in school health program research and development.

v. It will allow a risk statement to be made, although this is not precise.

But, the current design will be limited as regards the following disadvantages:

i. It provides no direct estimate of risk.

ii. It is prone to bias from selective survival.

iii. Since the environmental exposure and bronchial asthma are measured at the same point in time, it is not possible to establish temporality (i.e. whether the exposure or presence of a characteristic preceded the development of bronchial asthma).

3.5. Exploratory Study

An exploratory study have been carried out during the preparatory research phase (1st. 2 months) in order to:

i. Formulate the problem of the study for more precise investigation.

ii. Reformulate research questions and hypothesis.

iii. Refine the study variables.

iv. Examine the delivery of school health services at the actual study sites.

v. Make the researcher more familiar with the study population under the investigation.

vi. Examine the delivery of School Health Services at the local level.

vii. Identify the locally available resources that might be used in the study.

3.6. Sample Design:

3.6.1. Target Population and Sample Size

The target population of the current study was the school children aged 7-12 years of Abu-Dhabi primary schools, UAE. The total number of schools in Abu Dhabi City is 60 primary schools, 30 boys and 30 girls with total student 17472=8568 girls and 8904 boys. The determination of the sample size in the current research was actually coincided with its objectives; the suitable method for determination sample size was the one sample situation that based on estimating a population proportion.

Thereby, the criteria for sample size determination were:

1. The total population of the final research sample sites.

2. The anticipated population proportion

3. The confidence level: A confidence level of 95% is the least acceptable one, and this level was adopted in the current research.

4. The standard error of the mean: The worst acceptable standard error of the mean is in the range of 5-10 %

According to the previous criteria and the available data, it was expected that a

sample size of less than 500 schoolchildren with bronchial asthma was sufficient to determine the prevalence rate. Also, according to the exploratory study, it was found that the prevalence of bronchial asthma might be around 12%.

The minimal sample size was calculated according to an estimated prevalence of bronchial asthma, 12%, with the application of **Lwanga and Tye formula (1999)** for sample size calculation:

n= [(Z)² x P (1-P)/ d².] where **n**= sample size, **Z**= The standard normal deviate, and usually set at 1.96 (or more simply at 2.0), **P** = the proportion, and **d** = the absolute sample error that can be corrected, usually set at .5. n =169.

3.6.2. Sampling procedure:

The Multi-Stage Random Sample Technique was used to recruit the required sample. The stratification of the target population was based on the geographical distribution of the primary schools in Abu Dhabi city. The participant samples were 3521 children attending the primary school at Abu-Dhabi City.

3.7. Pretest Study

A pretest study was carried out during the first two months to help in the finalization of the research instruments and form as well as the finalization of the study design.

It was guided by the following tasks:

i. Testing the form design, content and language at the study sites.

ii. Measuring the time and resources needed for the fieldwork.

iii. Determining the categories and codes for open-ended questions.

iv. Examining the internal validity and reliability of the study forms.

3.8. Study Form and Data Collection Techniques:

An interviewing questionnaire has been developed and covered all the above-mentioned variables and comprehensive clinical chest examination questionnaires were also done.

The following variables were included:

3.8.1. Child Data: Name, age, sex, birth order, number of siblings and feeding history.

3.8.2. Parent Data: Age, educational level, presence of job and smoking.

3.8.3. Family History: Asthma, hay fever and eczema.

3.8.4. Environmental condition of the House: Crowding index, pets/livestock, and indoor pollution.

3.9. Ethical Considerations:

Although the current study has not any experiment aspect but the researcher has reviewed several international conventions (e.g. the Helsinki declaration) and once the potential subjects have been determined, the researcher obtained their oral 'informed consent' before they are subjected to the study tools and instruments.

3.10. Data Management and Analysis Plan

3.10.A. Quality Control

The accuracy of the collected data was ensured through the following measures:

i. The average daily of collected forms was not exceeded 6 schools/day

ii. Daily revision of all forms was made by the researcher with some of his academic colleagues to ensure good quality for the collected data.

iii. Discussion of the identified constraints with the relevant school health administrators and an appropriate action was taken.

iv. The quality of data was checked to assure accuracy, uniformity and comparability.

v. Some field visits were repeated to see whether the collected data for certain target population obtained similar results or not.

vi. Other techniques for quality control were developed according to the actual situation.

3.10.B. Data Processing

The data processing phase of this research was started as early as possible to deal with the collected data with great accuracy. This research phase can classify into:

3.10.B.1. Data processing planning stage:

The following tasks were adequately reviewed:

i. Specification what is the research output to be with great precision.

ii. Determining the best way to produce this output.

iii. Allocation of the required resources to achieve this processing speedily and accurately.

The researcher was guided by the following:

i. The draft contents of the intended final thesis.

ii. The research questions, hypothesis to be tested and the relationship to be explored.

iii. The selection of variables for comparative purposes.

iv. A draft set of tabulations (Dummy Tables), in which the variables to be tabulated were clearly determined and the size groupings for frequency distribution is precisely allocated.

3.10.B.2. Data preparation phase:

This phase included the following tasks:

i. Editing and coding.

ii. Initial examination of a sample of the data.

iii. Data entry.

The researcher determined the following missions for the editing process:

i. To detect data that incorrect owing to misunderstanding by respondent and return the study form to the field for correction.

ii. To detect sets of forms containing data superficially acceptable, but on closer examination raise suspicion of possible inaccuracy.

iii. To correct obvious improper-recording that was mere slips of the pen.

For the proper extraction of data from the original study forms, the researcher used the following guidelines:

i. Most of codes were being numeric.

ii. The identification code was identified uniquely not only the respondent but also the location.

iii. A distinction was made between a response with the value zero and a non-response.

iv. Total that can be calculated by the computer from the individual component value was entered only if a check with a computed value is to be used in validation.

A validation program was designed to detect erroneous data and including the following checks:

i. The number of items for each variable on the file with specified totals.

ii. All codes are within the specified range.

iii. Specified consistency checks.

iv. The recorded numeric values that lie outside a specified range were specified with precision.

After the researcher achieved a clean file the data were protected to avoid any accidents that may happen to the computer disc.

3.10.C. Statistical Analysis:

The collected data were statistically managed as follows:

i. **Descriptive statistics**: The mean and median will be used as measures of central tendency. The standard deviation was used as a measure of dispersion.

ii. **Analytic statistics**: The chi-square, T and Z tests were used to test for the presence or absence of a statistically significant difference among the studied variables. Logistic regression analysis (Forward stepwise/FSTEP method) of different risk factors related to bronchial asthma. The accepted level of significance is < 0.05.

3.11. Implementation Plan: -

The research was implemented in randomly selected primary schools in Abu Dhabi, UAE. The research process proceeded as follows (**Figure, 3-A**):

3.11.A. Planning Phase:

Duration: 6 Months

This phase started early after getting the administrative and technical approvals on the proposal. It included the review of literature and exploratory study in order to finalize the following:

1. Detailed formulation of the research objectives, research hypothesis, and research variables.

2. Planning of the research procedures, methods of data collection, and sample design.

3. Selection of final research sites and development of research instruments and tools.

4. Planning schedules for data collection and specification of the research inputs such as manpower, facilities, supplies and operating costs.

5. Pre-test (pilot study). , first testing the questioners by selecting three school and call for parent meeting , in different times, selecting 120 parent of equal educational level 40 from each school , the out come result and points of complains was important to modification the 2ed questioners .(Appendix a)

3.11.B. Implementation phase:

Duration: 8 Months

The activities of this phase included the following:

1. Collection of research data.

2. Data Management and analysis.

3.11.C. Reporting Phase:

Duration: 8 Months

A preliminary final thesis was prepared and presented for open discussion in which due modifications were made and planning for dissemination of the research findings (workshop and publication in scientific journal) were agreed upon.

Figure (3-A): Implementation Plan-Chronogram

Phase/Activities	Month																					
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th	11th	12th	13th	14th	15th	16th	17th	18th	19th	20th	21st	22nd
A. Research Planning: -:																						
A.1. Topic Selection	X	X																				
A.2. Protocol Development	X	X																				
A.3. Exploratory Study			X	X																		
A.4. Literature Survey	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X		
A.5. Questionnaire Development				X																		
A.6. Sample Design				X																		
A.7. Sample Sites Selection					X																	
A.8. Administrative Approval				X	X																	
A.9. Data Collection Schedule						X																
B, Research Implementation: -																						
B.1. Pre-Test Study							X															
B.2. Data Collection							X	X	X	X	X	X	X	X								
B.3. Data Management								X	X	X	X	X	X	X								
B.3.1. Quality Control							X	X	X	X	X	X	X	X								
B.3.2. Data Entry												X	X	X								
B.3.3. Data Analysis													X	X								
C. Research Reporting: -																						
C.1. Progress Achievements															X	X	X	X	X	X		
C.2. Thesis Development															X	X	X	X	X	X		
C.3. Preliminary Thesis																				X	X	
C.4. Final Draft For Discussion																						X
D. Dissemination of Results: -																						X

- =May 2002
- =March 2004

SECTION (4):

RESULTS

SECTION (4): RESULTS

4.1. General characteristics of the studied sample:

The study was carried out in Abu Dhabi Emirates at the primary school health delivery level. The target population was 3521 school children aged 7-12 years of Abu Dhabi primary schools, UAE. The prevalence of bronchial asthma among the studied children was (14.3%), 503children as noticed in **(Figure 1-B).**

The mean age of the asthmatic and non-asthmatic children were nearly the same (8.78 ± 1.51& 8.77 ± 1.52) years respectively. No statistical significant difference was found (P > 0.05), in **(Table 1-B & Figure 2-B).**

As regard the sex distribution, male children were more among the asthmatic children (54.7%),with odds ratio (1.31) while females were more among the non-asthmatic group (52.2%) with odds ratio(0.76). A statistical significant difference was found between the two groups (P< 0.05) in **(Table2-B & Figure 3-B).**

Meanwhile, the order of birth most of the children in the asthmatic and non-asthmatic groups were coming the second in the order of birth (44.1%&44.9%) respectively. A statistical significant difference was found between the two groups (P< 0.05).in **(Table 3-B & Figure 4-B).** Most of the children were having the nationality of the UAE (90.8% among asthmatic & 85.2% among non-asthmatic). The 2nd. Nationality noticed was the Omani children (5.2% among asthmatic & 7.7% among non-asthmatic). A statistical significant difference was found between the two groups (P < 0.05) in **(Table 4-B & Figure 5-B).**

The majority of the studied children were born in the UAE (96.82% among asthmatic & 96.92% among non-asthmatic). No statistical significant difference was found (P> 0.05) in**(Table 5-B).**

Concerning the number of brothers most of the children in the asthmatic group were having two brothers while most of the non-asthmatic group were having one brother (24.6% & 26.3%) respectively .The mean number of brothers were (2.36 ±1.52& 2.23 ± 1.43) of the asthmatic and non-asthmatic groups respectively. No statistical significant difference was found between the two groups ($P > 0.05$). And $\chi 2 = 8.44$ in (**Table 6-B**).

As regard the number of sisters most of the children in the asthmatic and non-asthmatic groups were having two sisters (42.6% & 43.8%) respectively .The mean number of sisters were (2.31 ±1.34 & 2.17 ± 1.26) of the asthmatic and non-asthmatic groups respectively. No statistical significant difference was found between the two groups ($P > 0.05$). in (**Table 7-B**).

And in **Table (8-B)** shows the father's job, 12.7% of the asthmatic's fathers and 9.5% of the non-asthmatic fathers were have no job. A statistical significant difference was found between the two groups ($P < 0.05$).

As regard the mother's job 80.5% of the asthmatic's mothers and 86.4% of the non-asthmatic's mothers were having no job. A statistical significant difference was found between the two groups ($P < 0.05$), in **(Table 9-B).**

The results illustrated that most of the father's educational level were preparatory education (35.8% of the asthmatic & 31.7% of the non-asthmatic children). Uneducated fathers were the minority (4.60% of the asthmatic & 4.20% of the non-asthmatic children). A statistical significant difference was found between the two groups ($P < 0.05$), in **(Table 10-B & Figure 6-B).**

The results illustrated that most of the mother's educational level were preparatory education (30.6%) among the asthmatic children & secondary education among non-

asthmatics (32.8%). Uneducated mothers were the minority (8.4% of the asthmatic & 6.0% of the non-asthmatic children). A statistical significant difference was found between the two groups ($P < 0.05$), in **(Table 11-B & Figure 7-B).**

The results in **(Table 12-B)** is represent the multiple logistic regression analysis of the effect of the different socio-demographic variables on the development of bronchial asthma. It showed that the child's age, sex, order of birth and original nationality were significant risk factors for the development of bronchial asthma ($p < 0.05$). Also, mother's job and education were significant risk factors ($p < 0.05$).

4.2. Family history of asthma, hay fever & eczema and risk of development of bronchial asthma among the studied sample:

The relationship between a mother's history of asthma, hay fever, and eczema and the presence of asthma in children is presented in **(Table 13-B & Figure 8-B).**
Maternal asthma had the strongest associations with childhood asthma than hay fever and eczema. Maternal asthma was higher among the asthmatic group (15%) than the non-asthmatic group (10.3%) and a statistical significant difference was found between the two groups ($P < 0.05$).

The relationship between a Father's history of asthma, hay fever, and eczema and the presence of asthma in children is presented in **(Table 14-B and figure 9-B).** Father's asthma had the strongest associations with childhood asthma than hay fever and eczema. Father's asthma was higher among the asthmatic group (11.9%) than the non-asthmatic group (8.2%) and a statistical significant difference was found between the two groups ($P < 0.05$).

The relationship between a sibling's history of asthma, hay fever, and eczema and the presence of asthma in children is presented in **(Table 15-B).** Sibling's asthma had the

strongest associations with childhood asthma than hay fever and eczema. Sibling's asthma was higher among the asthmatic group (18.7%) than the non-asthmatic group (12.9%) and a statistical significant difference was found between the two groups ($P < 0.05$).

The relationship between a relative's history of asthma, hay fever, and eczema and the presence of asthma in children is presented in (**Table 16-B**). Relative's asthma had the strongest associations with childhood asthma than hay fever and eczema. Relative's asthma was higher among the asthmatic group (21.4%) than the non-asthmatic group (13.7%) and a statistical significant difference was found between the two groups ($P < 0.05$).

In addition, in the **Table (17-B)** shows a comparative analysis between Relatives' asthma among asthmatic & non-asthmatic children. Among relatives with asthmatic group, grandfathers constitute the majority among the asthmatic group (8%) while Aunts (father side) constitute the minority (1%). Among relatives of the non-asthmatic group, uncles (father-side) constitute the majority (8.3%) while uncles (mother- side) cousin constitute the minority (7%).

The results in (**Table 18-B**) is represent the multiple logistic regression analysis of the effect of family history of asthma on the development of bronchial asthma among children. It illustrated that the mother's, father's and grandmother's asthma were significant risk factors for the development of bronchial asthma ($p < 0.05$).

5.3. Indoor pollution (Smoking at present of asthmatic children) and risk of development of bronchial asthma among the studied sample:

Table (19-B) shows the relationship between family members smoking and the development of asthma among asthmatic & non-asthmatic children. The prevalence of father's smoking was higher in both groups than the other members. Father's smoking was

(56%) and (37.9%) of the asthmatic and non-asthmatic groups respectively. a statistical significant difference was found, (P <0.05). Brother's smoking was (15%) and (11.3%) among the asthmatic and non-asthmatic groups respectively. a statistical significant difference was found, (P <0.05). Mother's & sister's smoking were haven't a statistical significant difference, (P >0.05).

Table (20-B) shows the relationship between children bedroom status (Crowding Index =C.I.) and the development of asthma among asthmatic & non-asthmatic children. Only (6.8 %) of the asthmatic children has their own bedroom (C.I.=1) compared to (25.7%) of non-asthmatic children and (27.2 %) of the asthmatic children shared their bed room with three persons (C.I.=1/4) compared to (21.3%) of non-asthmatic children. A statistical significant difference was found between the two groups (P < 0.05).

Table (21-B) shows the relationship between child Passive Smoking status and the development of asthma among asthmatic & non-asthmatic children. Only (6.6%) of the asthmatic children exposed to passive smoking compared to (3.8%) of non-asthmatic children. A statistical significant difference was found between the two groups (P < 0.05) And odds ratio was(=1.655).

Table (22-B) shows the relationship between the pattern of feeding in the 1st. three months of life and the development of asthma among asthmatic & non-asthmatic children. It was noticed that (53.3 %) of the asthmatic children were practicing breast feeding only in the 1st. three months of life compared to (70%) of non-asthmatic children. a statistical significant difference was found between the two groups (P < 0.05).

Table (23-B) shows the relationship between the children history of Tonsillitis & Tonsillectomy and the development of asthma among asthmatic & non-asthmatic children. It

was noticed that (54.3%) of the asthmatic children has had 2 attacks of tonsillectomy in the last 12 months compared to (57.7%) of non-asthmatic children. No statistical significant difference was found between the two groups ($P > 0.05$). Tonsillectomy operation was recorded more among the non-asthmatic group (16.3%) compared to (6.4%) among the asthmatic group, a statistical significant difference was found between the two groups ($P < 0.05$).

Table (24-B) shows the relationship between Allergy to Food & Medicine and the development of asthma among asthmatic & non-asthmatic children. Allergy to food and medicine were higher among the asthmatic group (16.7% & 8.0% - respectively) than the non-asthmatic group (10.4% & 5.6% - respectively). A statistical significant difference was found between the two groups ($P < 0.05$).

Table (25-B) shows the relationship between Past History of some diseases and the development of asthma among asthmatic & non-asthmatic children. Pneumonia and/or pleurisy, bronchiolitis and Tuberculosis were higher among the asthmatic group (37%, 11.5% & 1.6% - respectively) than among the non-asthmatic group (24%, 8.5% & 0.4% - respectively). A statistical significant difference was found between the two groups ($P < 0.05$) And with odds ratio was (1.79,2.10 & 4.42 respectively).

Table (26-B) shows the relationship between presence and absence of Pets / livestock and the development of asthma among asthmatic & non-asthmatic children. Asthmatic children were more exposed to chicken and other pets (8.9%), while non-asthmatic children showed more exposure to dogs (7.7%). a statistical significant difference was found between the two groups ($P < 0.05$).

Table (27-B) shows the relationship between dealing with Pets/livestock and the development of asthma among asthmatic & non-asthmatic children. Asthmatic & non-asthmatic children were showed occasionally contact (25.6% & 10.2% - respectively). A statistical significant difference was found between the two groups ($P < 0.05$).

The result in (**Table 28-B**) shows the relationship between child's associated allergic conditions and the development of asthma among asthmatic & non-asthmatic children. The most frequent associated allergic condition among the asthmatic & non-asthmatic children was Hay fever, Rhinitis and Eczema (4.57% , 8.35% & 10.73% - respectively). A statistical significant difference was found between the three groups ($P < 0.05$). The results in (**Table 29-B**) is represent the multiple logistic regression analysis of the indoor pollution on the development of bronchial asthma among children. It illustrated that the father's smoking and presence of pets at home were significant risk factors for the development of bronchial asthma ($p < 0.05$).

4.4. Some Clinical characteristics of the asthmatic children:

The results in (**table 30-B**) is show that the majority of asthmatic children developed symptoms during the first two years of life (42.0% under one year & 20.5% between 1-2 years). Table (31-B) showed the multivariate analysis for Factors associated with asthma. Only age of onset below 48 months was associated with severe asthma (OR 2.13, 95% CI, 1.00-4.54). Exclusive breastfeeding for >4 months was found to be protective factor for development of asthma (OR 0.25, 95% CI 0.08-0.70). Associated allergy (OR 7.5, 95% CI 1.64-34.4) and past history of tuberculosis and bronchiolitis have the some ratio(OR 5.26, 95% CI 1.70-16.20) were associated with development of asthma.

Table (1-B): Distribution of the studied sample according to their age

Age (years)	Asthmatic Children		Non-asthmatic Children	
	No.	%	No.	%
7-8	139	27.63	866	28.70
8-9	105	20.87	587	19.45
9-10	81	16.10	487	16.14
10-11	102	20.30	616	20.41
11-12	58	11.53	353	11.69
12+	18	3.58	109	3.61
Total	503	100.00	3018	100.00
Mean ± SD	8.78 ± 1.51		8.77 ± 1.52	
Significance	t = 0.575, P > 0.05			

Table (2-B): Distribution of the studied sample according to their sex

Sex	Asthmatic Children		Non-Asthmatic Children		Odds Ratio
	No.	%	No.	%	Odds Ratio
Male	274	54.67	1441	47.75	1.31
Female	229	45.53	1577	52.25	0.76
Total	503	100.00	3018	100.00	
Significance					$P < 0.05$

Table (3-B): Distribution of the studied sample according to their order of birth

Birth Order	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
First	132	26.24	893	29.56
Second	222	44.14	1356	44.93
Third	19	3.77	131	4.34
Fourth	86	17.10	437	14.50
Fifth & more	44	8.75	201	6.67
Total	503	100.00	3018	100.00
Significance	$\chi 2 = 6.73$, $P > 0.05$			

Table (4-B): Distribution of the studied sample according to their original nationality

Original Nationality	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
U.A.E.	457	90.85	2572	85.22
Oman	26	5.17	231	7.65
Saudi	4	0.80	98	3.25
Yemeni	9	1.79	77	2.55
Others	7	1.39	40	1.33
Total	503	100.00	3018	100.00
Significance	$\chi 2 = 15.23$, $P < 0.05$			

Table (5-B): Distribution of the studied sample according to their place of birth

Place of Birth	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
U.A.E.	487	96.82	2925	96.92
Others	16	3.18	93	3.08
Total	503	100.00	3018	100.00
Significance	$\chi 2 = 0.01$, $P > 0.05$			

Table (6-B): Distribution of the studied sample according to number of brothers

Brothers (No.)	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
0	50	9.94	277	9.18
1	118	23.46	794	26.31
2	124	24.65	784	25.98
3	92	18.29	578	19.15
4	52	10.34	281	9.31
5+	67	13.32	304	10.07
Total	503	100.0	3018	100.0
Mean ± SD	2.36 ±1.52		2.23 ± 1.43	
Significance	$\chi 2 = 8.44$, $P > 0.05$			

Table (7-B): Distribution of the studied sample according to number of sisters

Sisters (No,)	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
0	19	3.78	98	3.25
1	124	24.65	847	28.06
2	214	42.54	1323	43.84
3	18	3.58	124	4.11
4	84	16.70	429	14.21
5+	44	8.75	197	6.53
Total	503	100.0	3018	100.0
Mean ± S.D.	2.31 ± 1.34		2.17 ± 1.26	
Significance	t = 7.66, P > 0.05			

Table (8-B): Distribution of the studied sample according to presence of a father's Job

Presence of a father's job	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Yes	439	87.28	2732	90.52
No	64	12.72	286	9.48
Total	503	100.0	3018	100.0
Significance	$\chi 2 = 5.08$, $P < 0.05$			

Table (9-B): Distribution of the studied sample according to presence of a mother's job

Presence of a mother's job	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Yes	98	19.48	410	13.59
No	405	80.52	2608	86.41
Total	503	100.0	3018	100.0
Significance	$\chi^2 = 12.15$, $P < 0.05$			

Table (10-B): Distribution of the studied sample according to father's educat-ional level

Father's Educational Level	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Uneducated	23	4.57	128	4.24
Primary level	44	8.75	184	6.10
Preparatory level	180	35.79	957	31.71
Secondary level	117	23.26	852	28.23
University level	139	27.63	897	29.72
Total	503	100.0	3018	100.0
Significance	$\chi 2 = 11.51$, $P < 0.05$			

Table (11-B): Distribution of the studied sample according to mother's educational level

Mother's Educational Level	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Uneducated	42	8.35	179	5.93
Primary level	75	14.91	373	12.36
Preparatory level	154	30.62	781	25.88
Secondary level	144	28.63	990	32.80
University level	88	17.49	695	23.03
Total	503	100.0	3018	100.0
Significance	$\chi^2 = 18.14, P < 0.05$			

Table (12-B): Socio-demographic factors associated with the risk of bronchial asthma development/logistic regression analysis

Variable	Odds ratio	Standard Error (S.E.)	D.F.	Significance
Child's Age	46.322	0.269	1	0.000
Child's Sex	7.5199	0.0969	1	0.006
Birth Order	82.68	0.167	1	0.000
Original Nationality	41.85	0.279	1	0.000
Number of Brothers	0.840	0.202	1	0.357
Number of Sisters	0.252	1.148	1	0.616
Father's Job	4.674	0.245	1	0.031
Mother's Job	12.466	0.125	1	0.000
Father's Education	.452	0.245	1	0.502
Mother's Education	8.975	0.205	1	0.003

Table (13-B): Distribution of the studied sample according to mother's history of asthma, hay fever and eczema

Mother's Disease		Asthmatic Children (N=503)		Non-Asthmatic Children (N=3018)	
		No.	%	No.	%
Asthma only	Yes	76	15.11	310	10.27
	No	427	84.89	2708	89.73
	Significance	$\chi2 = 10.34$, $P < 0.05$			
Hay fever only	Yes	14	2.78	59	1.95
	No	489	97.22	2959	98.05
	Significance	$\chi2 = 1.46$, $P > 0.05$			
Eczema only	Yes	26	5.17	119	3.94
	No	477	94.83	2899	96.06
	Significance	$\chi2 = 1.64$, $P > 0.05$			

Table (14-B): Distribution of the studied sample according to father's history of asthma, hay fever and eczema

Father's Diseases		Asthmatic Children (N=503)		Non-Asthmatic Children (N= 3018)	
		No.	%	No.	%
Asthma only	Yes	60	11.93	248	8.22
	No	443	88.07	2770	91.78
	Significance	$\chi2 = 7.44$, P <0.05			
Hay fever only	Yes	9	1.79	46	1.52
	No	494	98.21	2972	98.48
	Significance	$\chi2 = 0.20$, P >0.05			
Eczema only	Yes	30	5.96	133	4.41
	No	473	94.04	2885	95.59
	Significance	$\chi2 = 2.37$, P >0.05			

Table (15-B): Distribution of the studied sample according to his/her sibling's history of asthma, hay fever and eczema

Sibling's Diseases		Asthmatic Children (N=503)		Non-Asthmatic Children (N=3018)	
		No.	%	No.	%
Asthma only	Yes	94	18.69	390	12.92
	No	409	81.31	2628	87.08
	Significance	$\chi 2 = 12.09$, P <0.00			
Hay fever only	Yes	19	3.78	89	2.95
	No	484	9.62	2929	97.05
	Significance	$\chi 2 = 1.00$, P >0.05			
Eczema only	Yes	50	9.94	228	7.55
	No	453	90.06	2790	92.45
	Significance	$\chi 2 = 3.37$, P >0.05			

Table (16-B): Distribution of the studied sample according to his/her relative's history of asthma, hay fever and eczema

Relative's Diseases		Asthmatic Children (N=503)		Non-Asthmatic Children (N=3018)	
		No.	%	No.	%
Asthma only	Yes	108	21.47	413	13.68
	No	395	78.53	2605	8.63
	Significance	$\chi2 = 20.73$, $P < 0.00$			
Hay fever only	Yes	19	3.78	145	4.80
	No	484	96.22	2873	95.20
	Significance	$\chi2 = 1.02$, $P > 0.05$			
Eczema only	Yes	45	8.95	299	9.91
	No	458	91.05	2719	90.09
	Significance	$\chi2 = 0.45$, $P > 0.05$			

Table (17-B): A comparative analysis between relatives' asthma among asthmatic and non-asthmatic children

Relatives	Relatives diseased among asthmatic children (n = 169)		Relatives diseased among non-asthmatic children (n = 849)		Significance	
	No.	%	No.	%	X2	P
Grandfather	40	8.00	85	2.80	24.38	< 0.05
Grandmother	28	5.60	181	6.00	1.59	> 0.05
Uncle (father side) > him-self	16	3.20	250	8.30	29.12	< 0.05
Uncle (father side) > cousin	17	3.40	124	4.10	2.44	> 0.05
Uncle (mother side) > himself	19	3.80	24	8.0	24.65	< 0.05
Uncle (mother side) > cousin	16	3.20	22	7.0	18.53	< 0.05
Aunt (father side) > her-self	5	1.00	46	1.50	1.79	> 0.05
Aunt (father side) > cousin	12	2.40	26	9.0	6.39	< 0.05
Aunt (mother side) her-self	7	1.40	30	1.00	0.15	> 0.05
Aunt (mother side) > cousin	9	1.80	61	2.00	0.76	> 0.05

Table (18-B) Family history associated with the risk of Bronchial asthma development/logistic regression analysis

Variable	Odds Ratio	Standard Error (S.E.)	D.F.	Significance
Mother's asthma	4.612	0.645	1	0.032
Father's asthma	1.98	0.261	1	0.561
Brother's asthma	0.3597	0.0926	1	0.548
Sister's asthma	0.0217	0.0908	1	0.882
Grandmother's asthma	25.2765	0.2981	1	0.0000
Grandfather's asthma	0.03654	0.0917	1	0.7458

Table (19-B): Distribution of the studied sample according to family member's smoking

Family Member's Smoking		Asthmatic Children (N=503)		Non-Asthmatic Children (N=3018)	
		No.	%	No.	%
Father's smoking	Yes	282	56.06	1144	37.90
	No	221	43.94	1874	62.10
	Significance	$\chi2 = 58.99$, P <0.05			
Mother's smoking	Yes	165	32.80	789	26.14
	No	338	67.20	2229	73.86
	Significance	$\chi2 = 9.68$, P > 0.05			
Brother's smoking	Yes	76	15.11	341	11.30
	No	427	84.89	2677	88.70
	Significance	$\chi2 = 6.00$, P < 0.05			
Sisters' smoking	Yes	44	8.75	230	7.62
	No	459	91.25	2788	92.38
	Significance	$\chi2 = 0.76$, P > 0.05			

Table (20-B): Distribution of the studied sample according to child bedroom status

Child Bedroom Status	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Has own bedroom	34	6.80	774	25.65
Share with one other	182	36.0	839	27.80
Share with two others	151	30.0	761	25.22
Share with > two others	136	27.2	644	21.33
Total	503	100.0	3018	100.0
Significance	$\chi 2 = 87.63$, $P < 0.05$			

Table (21-B): Distribution of the studied sample according to child passive smoking status

Passive Smoking Status	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Yes	33	6.56	115	3.81
No	470	93.44	2903	96.19
Total	503	100.0	3018	100.0
Odds Ratio	= 1.655		P < 0.05	

Table (22-B): Distribution of the studied sample according to pattern of feeding in the 1st. three months of life

Pattern of Feeding	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Only breast fed	268	53.28	2113	70.01
Only bottle fed	76	15.11	157	5.20
Breast fed + Bottle fed	159	31.61	748	24.79
Total	503	100.0	3018	100.0
Significance	$\chi 2 = 89.60$, $P < 0.05$			

Table (23-B): Distribution of the studied sample according children history of tonsillitis and tonsillectomy

Child History of Tonsillitis and Tonsillectomy		Asthmatic children (N=503)		Non-Asthmatic Children (N=3018)	
		No.	%	No.	%
Two attacks of tonsillitis (Past 12 months)	Yes	273	54.27	1742	57.72
	No	230	45.73	1276	24.28
	Significance	$\chi 2 = 209$, $P > 0.05$			
Tonsillectomy	Yes	32	6.36	492	16.30
	No	471	93.64	2526	83.70
	Significance	$\chi 2 = 33.63$, $P < 0.05$			

Table (24-B): Distribution of the studied sample according to allergy to food and medicine

Allergy to Food and Medicine	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Allergy to Food	84	16.70	314	10.40
Allergy to Medicine	40	7.95	168	5.57
No Allergy to both	379	75.35	2536	84.03
Total	503	100.0	3018	100.0
Significance	$\chi 2 = 23.20$, P 0.05			

Table (25-B): Distribution of the studied sample according past history of some diseases

Past History (Multiple Responses)	Asthmatic Children (n=510)		Non-Asthmatic Children (n=3188)		
	No.	%	No.	%	Odds Ratio
Pneumonia & or Pleurisy	188	36.86	734	23.32	1.79
Bronchiolitis	58	11.37	176	5.52	2.10
Tuberculosis	8	1.57	11	0.35	4.42
None	256	50.20	2267	71.11	0.43

(n =number of responses)

Table (26-B): Distribution of the studied sample according to presence or absence of direct contacts with pets/livestock

Pets Contact	Asthmatic Children (n=517)		Non-Asthmatic Children (n=3073)	
	No.	%	No.	%
Cats	16	3.09	188	6.12
Camels	36	6.96	64	2.08
Dogs	33	6.38	237	7.71
Goats	15	2.90	32	1.04
Birds	8	1.55	119	3.88
Chickens and others	46	8.90	49	1.59
No direct contacts	363	70.22	2384	77.58
Significance	$\chi 2 = 156.79, P < 0.05$			

(n =number of responses)

Table (27-B): Distribution of the studied sample according to dealing with pets/ livestock

Pets contact	Asthmatic Children		Non-Asthmatic Children	
	No.	%	No.	%
Daily contact	11	2.19	327	10.83
Occasional contact	129	25.64	307	10.17
Never	363	72.17	2384	79.00
Total	503	100.00	3018	100.00
Significance	$\chi 2 = 119.53$, $P < 0.05$			

Table (28-B): Distribution of the studied sample according to child's associated allergic conditions and development of bronchial asthma

Associated Allergy	Asthmatic Children (n=503)		Non-Asthmatic Children (n=3018)		Odds Ratio
	No.	%	No.	%	OR
Hay fever	23	4.57	13	0.43	13.42
Rhinitis	42	8.35	57	1.89	5.57
Eczema	54	10.73	38	1.25	10.8
Not associated with asthma	384	76.35	2910	96.42	0.8
Significance	P < 0.05				

(n =number of responses)

Table (29-B): Indoor pollution and risk of bronchial asthma development/logistic regression analysis

Variable	Odds Ratio (95%. C.I.)	Standard Error (S.E.)	D.F.	Significance
Father's smoking	52.5509	0.107	1	0.0000
Mother's smoking	0.5179	0.140	1	0.479
Brother's smoking	1.8271	0.264	1	0.485
Sister's smoking	0.2019	0.223	1	0.145
Child has his/her own bed room	1.53881	0.046	1	0.00
Separate Kitchen	0.3027	0.266	1	0.432
Pets at home	19.9804	0.113	1	0.0000

Table (30-B): Age at which the attack of bronchial asthma began

Age at which the attack of bronchial asthma began	No.	%
Under one year	211	41.95
1-2 years	103	20.48
2-3 years	56	11.13
3-4 years	73	14.51
4-5 years	17	3.38
5-6 years	19	3.78
6-7 years	13	2.58
7+ year	11	2.19
Total	503	100.0

Table (31): Multivariate analysis for Factors associated with asthma

Factors	Odds Ratio	95% C.I
Age of onset of the disease <= 48 months	2.31	1-4.54
Age of onset of the disease <60 months	2.44	1.16-5.16
Exclusive breast feeding in the 1st. 4 months	0.25	0.08-0.70
Past history of tuberculosis with asthma	5.26	1.70-16.20
Past history of bronchiolitis with asthma	5.26	1.70-16.20
Presence of associated allergies	7.5	1.64-34.48

Figure (1-B): Prevalence of Bronchial asthma among the studied sample

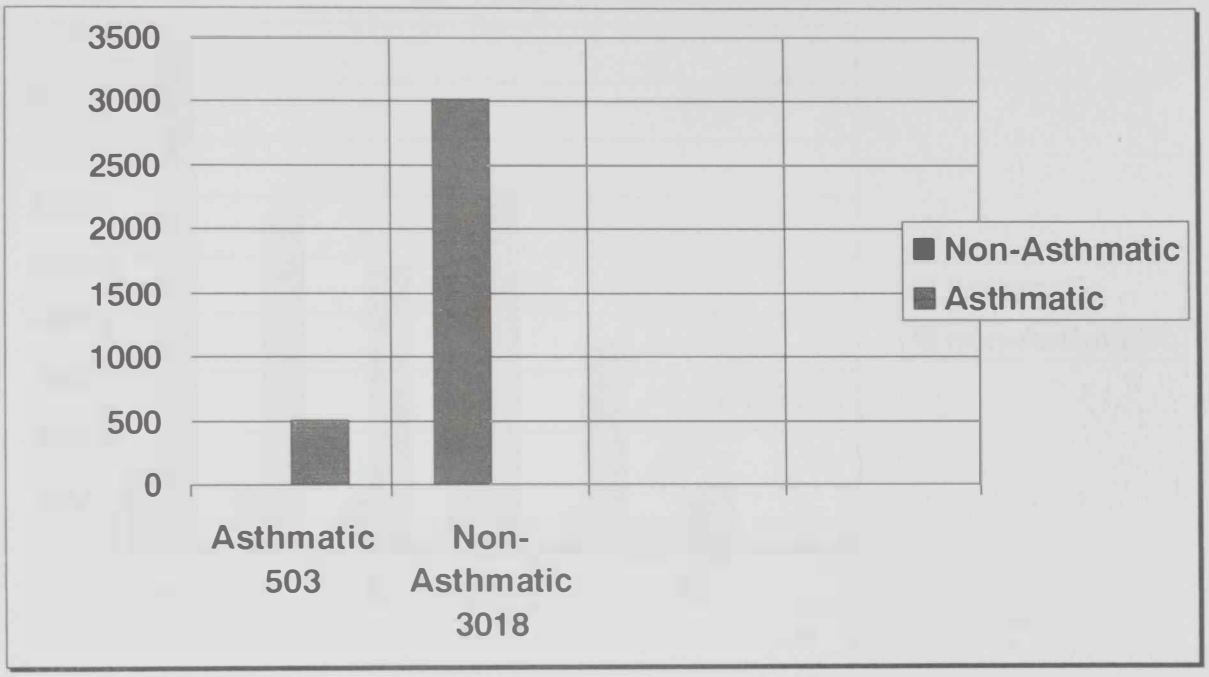


Figure (2-B): Distribution of the studied sample according to their age

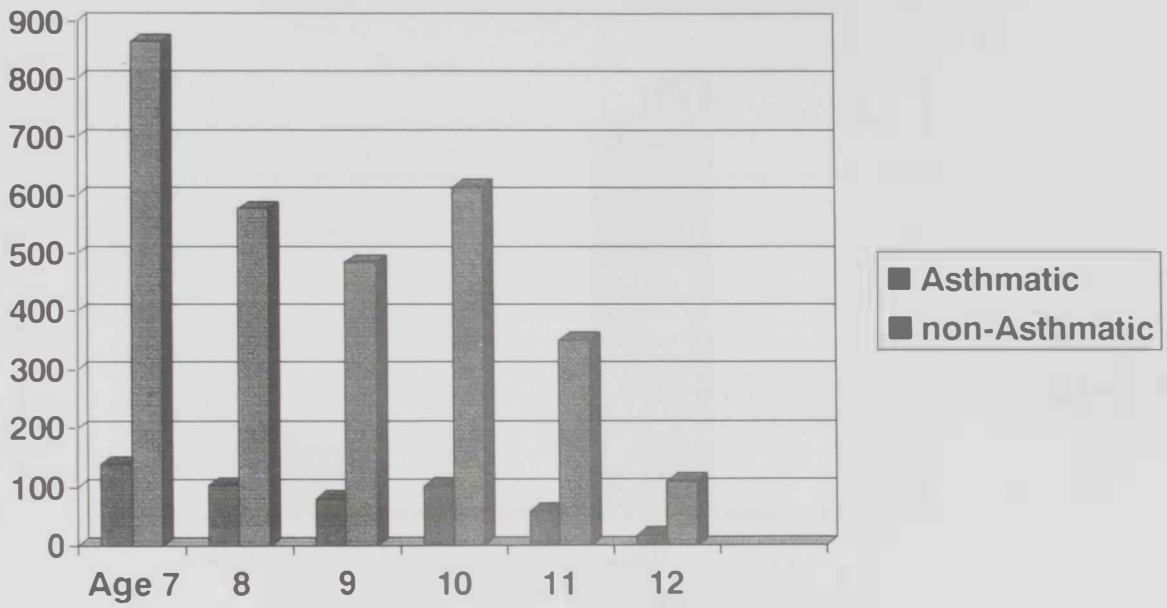


Figure (3-B): Distribution of the studied sample according to their sex.

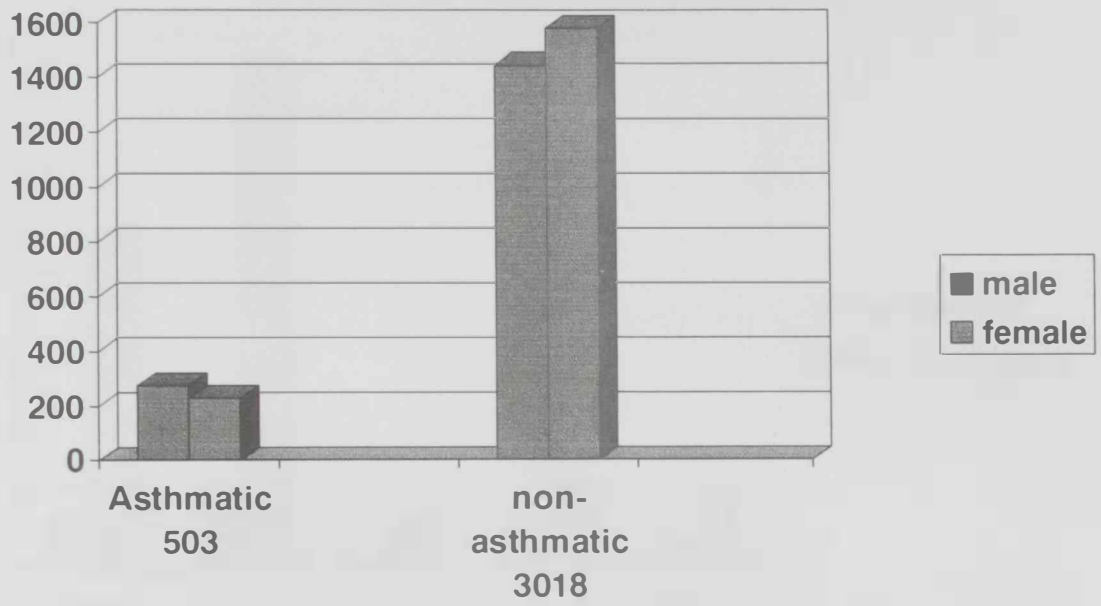


Figure (4-B): Distribution of the studied sample according to their order of birth

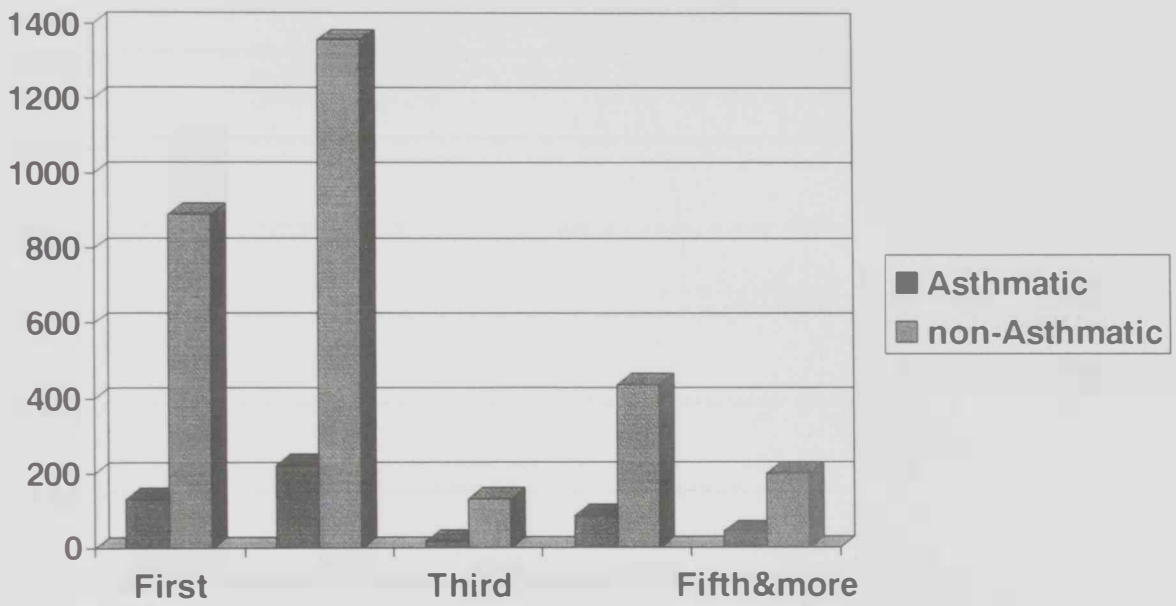


Figure (5-B): Distribution of the studied sample according to their original nationality

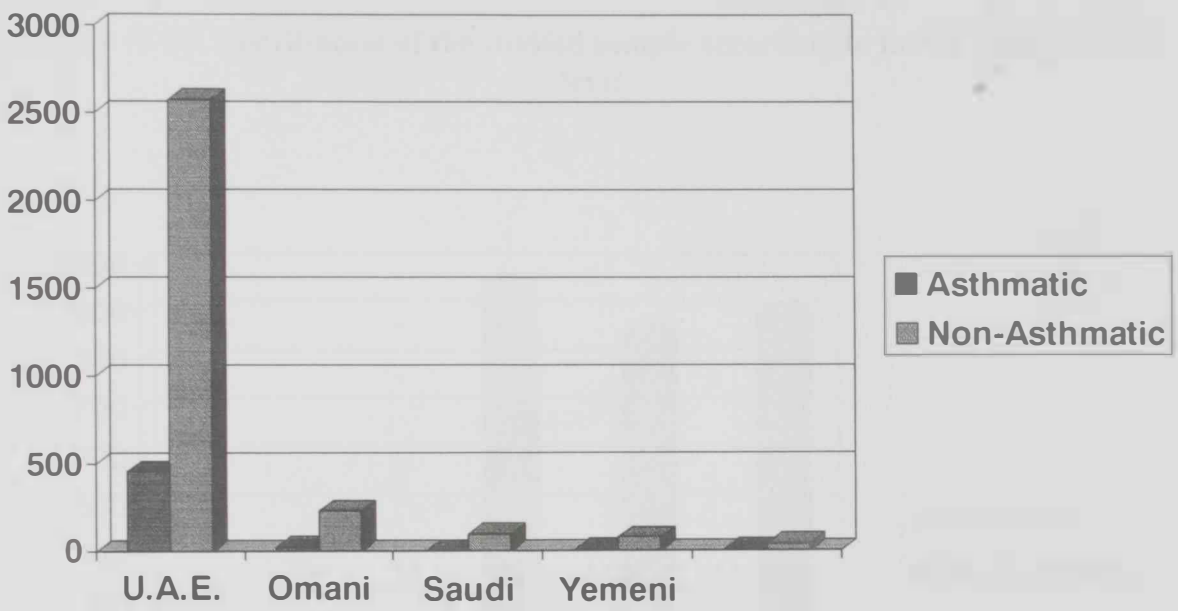


Figure (6-B): Distribution of the studied sample according to father's educational level

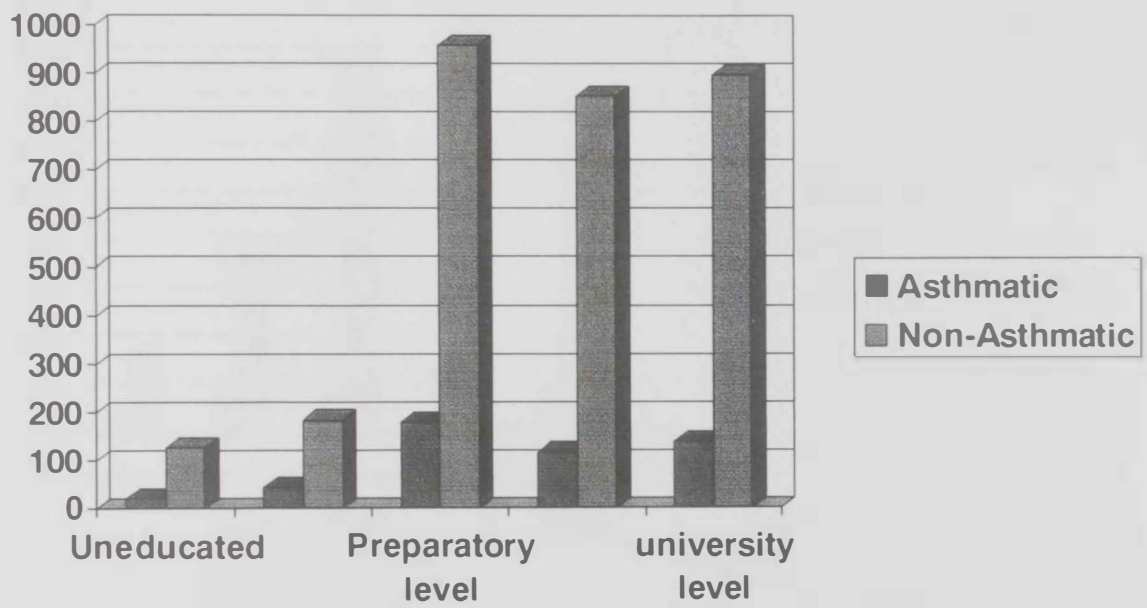


Figure (7-B): Distribution of the studied sample according to mother's educational level

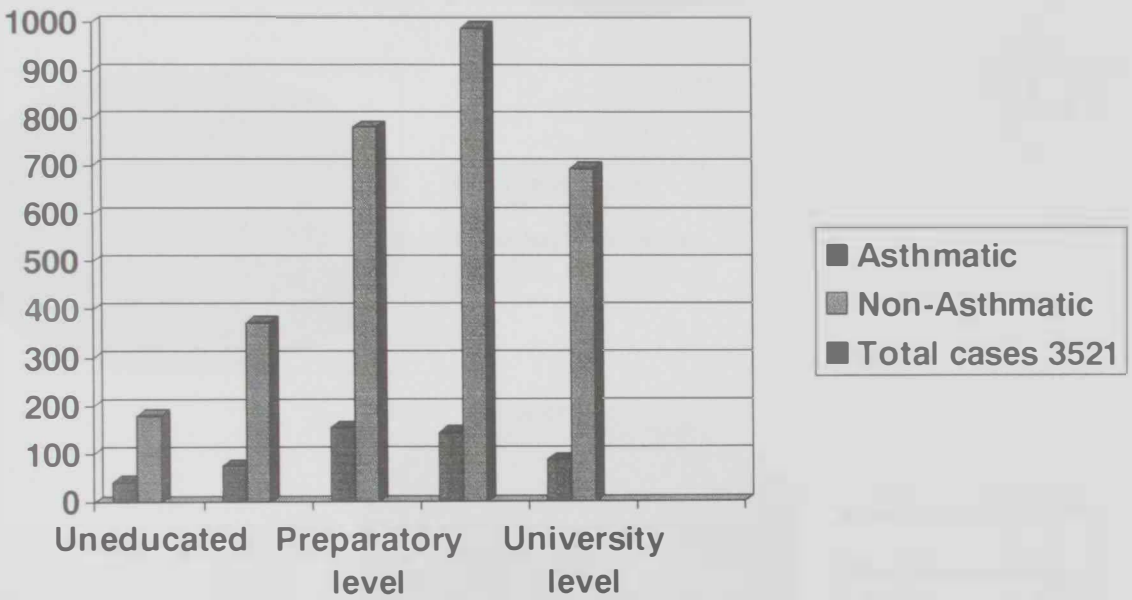


Figure (8-B): Distribution of the studied sample according to mother's history of asthma, hay fever and eczema

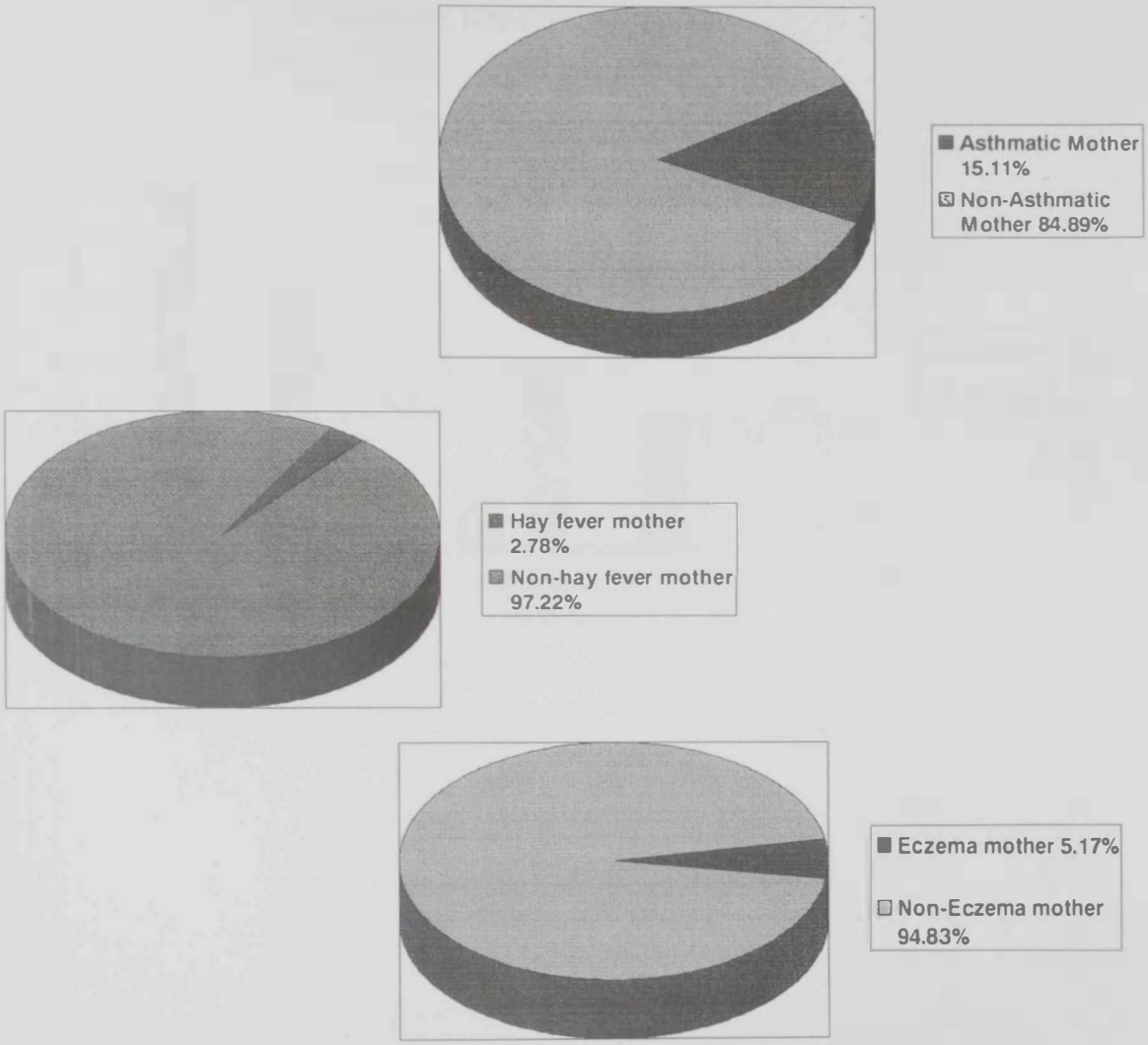
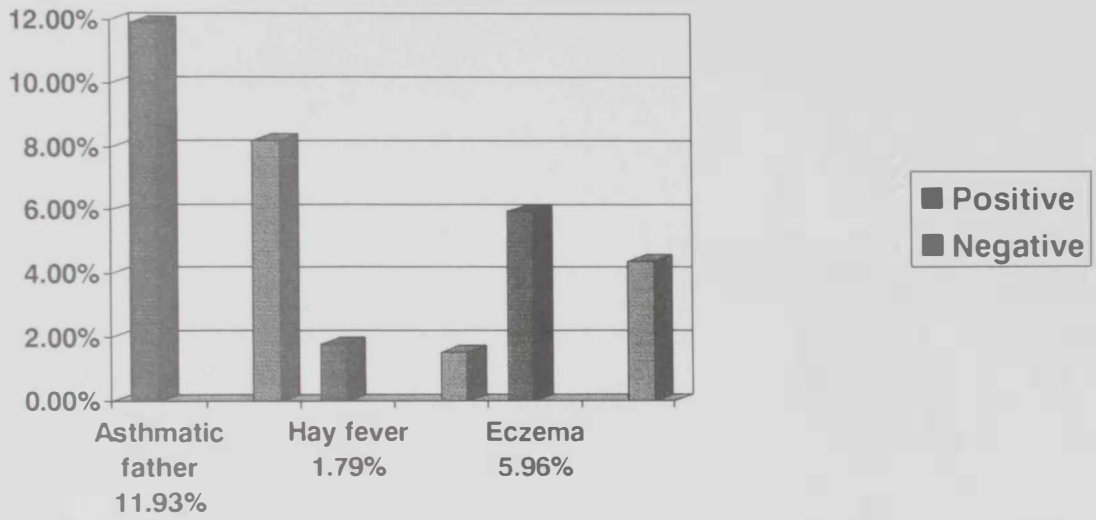


Figure (9-B): Distribution of the studied sample according to father's history of asthma, hay fever and eczema.



SECTION (5):

DISCUSSION

SECTION(5): DISCUSSION

Bronchial asthma is a reversible obstructive lung disease, caused by an increased reaction of the airways to various stimuli. It is a chronic condition with acute exacerbations. Asthma can be a life-threatening disease if not properly managed. It is estimated that 6.3 million children under 18 years of age have asthma; of which 4 million suffered from an asthma attack or episode in the past year **(WHO, 2003).**

Bronchial asthma is a common and life-threatening problem affecting school children and adolescents. Schoolteachers must be aware of this condition to educate their students, and to provide assistance to manage such attacks **(Al-Nasir, 2003).**

The first epidemiological studies of asthma date back to the first half of the XXth century. Since then a global increase in asthma prevalence has been revealed and there can be no doubt that asthma is now a major health problem worldwide. Meanwhile, asthma epidemiological studies are currently in the phase of that of cancer and coronary heart disease at the mid of the 20^{th} century when international comparisons of the prevalence of those diseases revealed the major risk factors and formed the bases of the further research **(Jögi, 2001).**

UAE realized the importance of the problem and initiated a National Control Program in 1990 **(Asthma Newsletter, 1991).**

This work was carried out on school children aged 7-12 years of Abu-Dhabi schools, UAE, 2003 to study the environmental risk factors of bronchial asthma. The Analytical

Cross-Sectional design (ACSD) was chosen as a suitable method for the current research issue as it can investigate the environmental risk exposure factors and bronchial asthma simultaneously in a representative sample of the school-aged students.

The present study revealed that the point prevalence (asthma at the time of the study survey) of bronchial asthma among the studied children was 503 out of 3521 (14.28%) (**Figure, 1-B**).

The prevalence of asthma has been reported to be increasing worldwide, but this apparent trend continues to be debated. Changes in prevalence can be confounded by factors such as: differing levels of awareness of the disease by health care providers and/or families; changes in access to medical care; and changes in medical diagnosis.

A similar study was done by (**Bener et al, 1993**), in Dammam and Riyadh, Saudi Arabia. The results showed that the prevalence was (11.86 %) in Riyadh and (6.54 %) in Dammam.

Our results are less than that found by (**Colin et al, 1998**), among the Australian primary school children. They reported that asthma prevalence was (24.6%). The prevalence rate of reported diagnoses of asthma among Omani primary school children was (10.5%) as reported by (**Bazdawi et al, 2003**).

However, the prevalence of asthma in our study among the school-age children is consistent with previous studies in many countries. (**Björkstén B, 1995**)

The increase in asthma prevalence in our study may be explained by altered diagnostic criteria, increase awareness of the disease, both among the general population and physicians. A diagnostic shift from COPD to asthma may explain part of the increase in asthma prevalence.

An American study found increasing trends of asthma prevalence to parallel increases in the broader category of chronic obstructive airway diseases (**Vollmer et al. 1998**).

A Danish study among young adults found no time trend in smoking habits or in mucus production prevalence of asthmatic patients, suggesting that misclassification might not be increasing (**Hansen et al. 2000**).

Sunyer and co-workers have recently reported a retrospective assessment of the incidence rates of asthma in Europe by using the ECRHS dataset (**Sunyer et al.2000**), they found a higher incidence of asthma in the younger cohorts.

As regard the sex differences, the result revealed that male children were suffering more from asthma than girls (**Table 2-B and Figure 3-B**). This difference can be partly attributable to sex differences in lung growth and airway geometry (**de Marco et al, 2000 and Wassmer et al, 1997**), but may also be connected to the sex-related differences in the immune system.

Also boys tend to have higher incidence of acute respiratory tract infection than girls (**Mont, 1974**). Male sex was found to be a risk factor compared to female sex in a similar study done by (**Horwood, 1985**) for children under 14. The prevalence of asthma among boys was found double that among females.

The study revealed that most of the asthmatic and non-asthmatic children came the second in the order of birth (**Table 3-B**), the analysis showed that the order of birth was not a significant factor in the occurrence of bronchial asthma.

Our findings are similar to that reported in literature. In a study by **Speight et al. (2000)**, it was observed that, no association between the order of birth and development of bronchial asthma.

The present study revealed negative association between the mean number of brothers and sisters on the development of bronchial asthma (**Tables, 6-B & 7-B**). Our results agree with that of (**Strachan, 2000, and Matricardi, 1998**) who reported negative association.

On the other hand, a significant number of studies conducted around the world have found an association between the mean number of brothers and sisters on the development of bronchial asthma.

Schwartz (1994) reported a positive association between the number of sibling and the occurrence of asthma. This association could result from genetic factors or a shared environment.

Among the socio-demographic variables studied was the father's job. From our study it is clear that father's job gives idea about the family income together with the mother's job (**Tables, 8-B & 9-B**).

Schwartz (1990) showed increase of the prevalence of asthma among children who's their father's has no job and inversely related to mother's job. It is likely that absent job of fathers is associated with poor educational level, excessive smoking and other conditions reflecting environmental pollution.

Our results suggest that father's and mother's educational level plays an important role in asthma prevalence (**Table, 10-B &11-B**). In Alexandria (Egypt), (**Sabry, 1998**) **and** (**Selim et al, 2000**) reported that paternal education is inversely proportional with bronchial asthma occurrence. In a study of 499 families living in the Boston metropolitan area, (**Litonjua et al. 1999**), found that low parental education (not higher than high school) was an independent predictor of childhood asthma.

The multiple logistic regression analysis of the effect of the different socio-demographic variables on the development of bronchial asthma (**Table 12-B**) showed that the child's age, sex, order of birth and original nationality were significant risk factors for the development of bronchial asthma ($p < 0.05$).

The work of (**Vendo et al. 2000**) in India revealed that child's age, sex, father's and mother's education were the significant risk factors.

The study revealed that (15%) of mothers and (11.9%) of fathers of children with bronchial asthma were asthmatic (**Tables, 13-B &14-B**). Sandford stated that Parental asthma has been shown in several studies to be a strong predictor of asthma in the child. (**Sandford et al. 1996**)

In a community-based sample of 770 children 5 to 9 yr of age from East Boston, Massachusetts, **Sherman and coworkers (1990)** showed that the relative risk for asthma in the index child was 1.95 (95% CI = 1.29 to 2.95) when at least one parent had asthma. The risk for childhood asthma was also increased (RR = 1.61, 95% CI = 1.03 to 2.50) when one of the parents had atopy (defined as either eczema or hay fever), but parental atopy was not as strong a predictor as was parental asthma. (Defined as either eczema or hay fever), but parental atopy was not as strong a predictor as was parental asthma.

In a study of 6,665 families of 9- to 11-years-old children in Munich and Southern Bavaria, **Dold and coworkers (1992),** similarly found that the risk for asthma in the child was increased when one parent had asthma (OR = 2.6, 95% CI = 1.7 to 4.0). However, the risk related to maternal asthma (OR = 1.5, 95% CI = 0.7 to 2.7) was not as great as that related to paternal asthma (OR = 4.4, 95% CI = 2.5 to 7.8).

The study revealed that 2.8% and 5 % of mothers of asthmatic children were having hay fever and eczema respectively (**Table13-B & Figure 8-B**), while 1.8% and 6.0% of fathers of the same children were complaining of hay fever and eczema respectively (**Table 14-B & Figure 9-B**).

In a study of 6-to 14-yrars-old schoolchildren in the United Arab Emirates (UAE) by **Abdulrazzaq et al. (1995)**, the risk for asthma conferred by a maternal history of asthma (RR = 2.67, 95% CI = 1.65 to 4.35) was about the same as that conferred by a paternal history of asthma (RR = 2.85, 95% CI = 1.81 to 4.49). In addition, both maternal allergic rhinitis and eczema also significantly increased the risk for childhood asthma, whereas a paternal history of these conditions was not related to childhood asthma.

In our study the risk of paternal asthma was also confirmed by doing logistic regression analysis (O.R. 12.153, p <0.0005, mother's asthma & O.R. 20.276, p <0.0000, father's asthma) as presented in (**Table 18-B**).

The children in our study were younger on average than those in the previously mentioned studies of German and UAE schoolchildren, and this age difference may explain why we found a stronger effect of maternal than of paternal asthma.

Our results are consistent with the hypothesis that there may be preferential inheritance of allergic diseases through the maternal line and suggest that this phenomenon is manifested early in the life of the child (**Ruiz et al. 1992**).

Our results, in conjunction with the results of the studies cited above, lead us to speculate that perhaps the maternal condition exerts a stronger effect early in the life of the child, whereas the paternal condition may be involved in the development of asthma in later life.

Cookson and colleagues (1992) suggested preferential inheritance of atopy through the maternal line by showing excess sharing of the maternal 11q13 allele among affected sibling pairs.

Other investigators, however, have been unable to demonstrate differences in the sharing of paternal and maternal alleles at 11q13 (**Coleman et al. 1993**).

Genetic mechanisms such as paternal genomic imprinting (**Doull et al. 1996**), and the Carter effect (**Happle et al., 1982**), which have been shown to operate in other diseases, have been proposed to explain the preferential inheritance through the maternal line, but thus far no evidence indicates that these mechanisms are operative in asthma or in other allergic diseases.

In addition to genetic inheritance, mechanisms for conferring early-life risk preferentially through the maternal line may include transplacental transfer of antigens, maternal antibodies, or maternally derived cytokines (**Doull et al. 1996**), leading to the reported association between maternal history of allergy and high neonatal serum IgE levels (**Hamada K. et al. 2003, Ruiz et al. 1992**).

Therefore, factors that modulate the maternal immune system during pregnancy are thought to be active in the development of allergic disease in the offspring (**Warner et al., 1997**). These mechanisms exert their greatest effect early in life and may lead to earlier onset of allergic disease.

Furthermore, inheritance by either genetic or environmental mechanisms may vary not only by age but also by type of atopic disease. Because of the cross-sectional design of this analysis, the lack of biologic markers, and the lack of information regarding maternal

exposures during pregnancy, we are unable to draw definite conclusions about the reasons for the differential effects of maternal and paternal asthma on childhood asthma.

In summary, although asthma and other allergic conditions often go together, children are more likely to develop atopic diseases similar to those of their parents. The explanation may be different inheritance patterns for asthma and the other atopic diseases. Furthermore, our study adds to the evidence for preferential inheritance of early-childhood asthma along maternal lines. Whether this effect is due to genetic factors, placental transfer of maternal factors, and/or exposure to environmental influences is not known and will need to be studied further.

The result of the study illustrated a significant association between fathers and brothers smoking and development of bronchial asthma among the studied children (chi square 58.99, $P < 0.05$), (**Table 19-B**). The link between tobacco smoke and asthma has been the subject of many epidemiological studies and research programs, worldwide and non-smokers exposed to passive smoking, (also known as environmental tobacco smoking or ETS) have been found to be adversely affected.

Recent research from Finland has shown that passive smoking plays a role in the development of childhood asthma. Researchers found that subjects exposed to tobacco smoke in the workplace were twice as likely to develop asthma as those who were not exposed, **(Jaakkola et al. 2001).**

Asthma is the commonest chronic illness of childhood, affecting between 10-15% of children. There is considerable evidence that passive smoking increases the frequency and severity of symptoms in children with asthma" **(Royal College of Physician, 1992)**.

The bronchial tubes of children are smaller and their immune systems are less developed, making them more likely to develop respiratory illness when exposed to environmental tobacco smoke. Because they have smaller airways, children breathe faster than adults and consequently breathe in more harmful chemicals per pound of their weight than an adult would in the same amount of time.

In an analysis of data on 4,000 children aged 0-5 years, it was found that father smoking of more than 10 cigarettes a day was associated with higher rates of asthma, an increased likelihood of using asthma medication, and an earlier onset of asthma than was observed in children of non-smoking fathers.

Research has also shown that when children have been hospitalized for acute asthma and return to a home where there is a smoker, their recovery is impaired. 82 per cent of children that went home to non-smoking households had less than 1 symptomatic day per week compared with smokers with only 27 per cent of the children who went home to households with smokers (**Lewis et al.1995**).

The **UK government** appointed Scientific Committee on Tobacco and Health (SCOTH) concluded in its 1998 report: "Smoking in the presence of infants and children is a cause of serious respiratory illness and asthma attacks".

The **World Health Organization (WHO)** convened an International Consultation on Environmental Tobacco Smoke (ETS) and Child Health in (**1999**). The Consultation brought together experts from developed and developing countries to examine the health effects of ETS on child health and to recommend interventions to reduce these harmful effects and eliminate children's exposure. The final conclusions of the Consultation state Both asthma and respiratory systems (wheeze, cough, phlegm) are increased among children whose

parents smoke, on the basis of over 60 studies of school-aged children. The pooled relative risks for either parent smoking range from 1.2 to 1.4."

In 1986 in the United States, a comprehensive review of the health effects of exposure to passive smoking was published by the US Surgeon General (**US Environmental protection Agency 1986**).

The report concluded that ETS could be causally associated with respiratory illnesses, including lung cancer, childhood asthma and lower respiratory tract infections. Following on from this study, in 1992 the US Environmental Protection Agency undertook a broad review of the major health effects associated with ETS. The findings of the review state: ETS exposure is causally associated with additional episodes and increased severity of symptoms in children with asthma. This reports estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to ETS."

As regard the child bedroom status (crowding index), it was noted that increase the number of persons/room is a risk factor for development of bronchial asthma (**Table 20-B**). This can be explained that increase of the C.I. enhances chest infection and this leads to increase of the bronchial hyper-responsiveness together with increase poor quality of air. **Vaughan-Williams E et al, (1989) and Anderson HR et al. (1994)** proved that smaller size home and increase of C.I. were all associated factors with increase rates of childhood asthma.

As regard the type of feeding in the 1st. three months of life (**Table 22-B**), it was observed that absence of breast-feeding is associated with development of bronchial asthma. Moreover, multivariate analysis for the factors associated with asthma (**Table 31-B**) evidenced that exclusive breast-feeding in the 1st. 4 months has a protective effect against

bronchial asthma development. **Zeiger et al (1986)**. Critically evaluated 16 studies out of which 9 prospective studies showed benefit and 7 showed lack of effect.

A recent study by **Wright et al. (1999)** has demonstrated that breast-feeding was most protective against wheezing lower respiratory tract illness early in life. Breastfeeding may decrease allergic sensitization by reducing both exposure and intestinal absorption of food allergens. The protective role of human breast milk immunoglobulin, especially serum IgA, in inhibiting absorption of antigenic substances has been documented in human neonate **Kerner et al. (2000)**.

As regard the presence of allergy to food and medicine (**Table 24-B**), it was observed that asthmatic children were having sensitivity to food and medicine than non-asthmatic children. This finding is in accordance with the result of **Lindfors et al. (1995)** who found that allergy to food such as cow milk and some medication was frequent among children with bronchial asthma.

As regard the presence of past history of some diseases (**Table 25-B**), it was observed that Pneumonia and/or pleurisy, bronchiolitis and Tuberculosis were higher among the asthmatic group than among the non-asthmatic group. In our study bronchiolitis was associated with development of asthma.

Numerous studies both retrospective and prospective have been performed evaluating the persistence of both clinical airway symptoms and the presence of functional airway abnormalities later in life (**Hogg, 1992 and Strachan & Carey, 1995**).

All these suggested a positive relation between bronchiolitis during infancy and development of recurrent wheezing in later life. There are no studies to link past infection with

Mycobacterium tuberculosis and asthma. In a study on 200 children presenting with acute wheeze, 20% of the patients were found to have active tuberculosis **(Pherwani et al., 1992).** Another study on bronchial challenge with Purified Protein Derivative (PPD) suggested presence of Type I Arthus type of IgE mediated allergic reaction to PPD in 59% in asthmatic and 12% in children with pulmonary tuberculosis. None of the children with extra pulmonary tuberculosis and controls had abnormal bronchial challenge to PPD. On the basis of positive bronchial challenge with PPD and detection of PPD specific IgE in serum of children with asthma and pulmonary tuberculosis, it was concluded that Arthus type IgE mediated reaction was responsible for hyper-reactivity of airways in some children with asthma **(Pherwani et al., 1992)**.

In a recent report, **Shirakawa et al. (2001),** observed an inverse association between tuberculin response and atopic disorders.

As regard the presence of pets **(Table, 26-B)** and dealing with pets **(Table, 27-B)** were found to be risk factor for the development of bronchial asthma and a statistically significant difference was found between the asthmatic and non-asthmatic group. **Strachan & Carey, (1995)** showed that pet ownership was associated with severe asthma.

As regard the presence of associated allergic conditions **(Table 28-B)**. The most frequent associated allergic condition among the asthmatic & non-asthmatic children was Rhinitis. And there is strong association between other histories of allergic disease such as recurrent rhinitis, atopic dermatitis and development of asthma were had been reported in other studies **(Anderson HR et al., 1986 & Foucard et al. 1984).**

As regard the age at which the asthma began **(Table, 30-B)**, our results suggest that earlier the onset of symptoms more frequency was the disease. In literature most studies

showed no correlation between age of onset of wheezing in children and persistence of symptoms into later childhood/adulthood or severity **(Samet et al., 1983)**. **Lebowitz et al. (1998)** have shown that children with the onset of symptoms later in childhood were at a higher risk for ongoing symptoms than those with onset in first two years of life.

SECTION (6):

CONCLUSIONS

SECTION (6): CONCLUSIONS

Asthma is one of the most common chronic diseases worldwide, and its prevalence is known to be increasing, particularly among children. The present study was conducted to study the effect of some environmental risk factors on the development of Bronchial Asthma among primary schools in Abu Dhabi city, UAE.

The result of the following study revealed the following conclusions:

I. The mean age of the asthmatic and non-asthmatic children were nearly the same (8.78 ± 1.51& 8.77 ± 1.52) years respectively.

II. The majority of asthmatic children developed the symptoms during the first two years of life (42.0% under one year & 20.5% between 1-2 years).

III. Bronchial asthma was more among male children (54.67%) than females (45.53%).

IV. Most of the children were having the nationality of the UAE (90.8% among asthmatic & 85.2% among non-asthmatic). The 2nd nationality noticed was the Omani children (5.2% among asthmatic & 7.7% among non-asthmatic).

V. The majority of the studied children were born in the UAE (96.82% among asthmatic & 96.92% among non-asthmatic).

VI. About 12.7% of the fathers of asthmatic children without job compared to and 9.5% of the non-asthmatic fathers.

VII. Most of the father's and mother's educational level were preparatory education.

VIII. The multiple logistic regression analysis of the effect of the different socio-demographic variables on the development of bronchial asthma showed that the child's age, sex, order of birth and original nationality were significant risk factors for the development of bronchial asthma.

IX. Paternal history of asthma was more among asthmatic children, 15% of their mother and 11.9 of their fathers were asthmatic

X. Father's smoking was (56%) and (37.9%) of the asthmatic and nun-asthmatic groups respectively.

XI. Crowding Index (C.I=1/3.) was .27.2% asthmatic and (C.I=1/4) of (21.3%) compared to of the nun-asthmatic children.

XII. About (6.65%) of the asthmatic children exposed to passive smoking compared to (3.83%) of non-asthmatic children.

XIII. Practice of exclusive breast-feeding in the 1st. 3 months of life was more among non-asthmatic children (69.87%) than asthmatic children (53.2%).

XIV. Pneumonia and/or pleurisy, bronchiolitis and Tuberculosis were higher among the asthmatic group (37.4%, 10.2% & 1.6% - respectively) than among the non-asthmatic group (24.4%, 5.9% & 0.4% - respectively).

XV. Asthmatic children were more exposed to chicken and other pets than non-asthmatic children.

XVI. The most frequent associated allergic condition among the asthmatic & non-asthmatic children was Rhinitis (7.2% & 3.4%) respectively.

XVII. Multiple logistic regression analysis of the indoor pollution on the development of bronchial asthma among children illustrated that father's smoking and presence of pets at home were significant risk factors for the development of bronchial asthma ($p < 0.05$).

SECTION (7):

RECOMMENDATIONS

SECTION (7): RECOMMENDATIONS:

Based on the above-mentioned conclusions, one can recommend the followings:

i. In medical management and planning of school health services measures to improve adjustment and quality of life of children with asthma, various aspects of asthma severity, notably the studied significant risk factors should be considered.

ii. Smoking cessation and pet avoidance should be advised for the unstable asthma.

iii. Asthma educational programs incorporate and validate "lived experiences" in the UAE schools, as well as skills that foster self-empowerment are important for the health promotion of children with bronchial asthma.

iv. Medical and environmental activists narrow the gap between their respective projects and work together to combat asthma Reinvigorating inner city neighborhoods to provide the community with open spaces which can help to foster positive perceptions of outdoor environments

v. Apart from asthma-specific features and given the fact that there are very few asthma cross-sectional studies have been conducted in UAE, this thesis with the others can contribute to the development of an evidence-based community approach for proper planning of health care of children with asthma.

vi. More comparative and community-based studies are needed to determine differences among the various seasons, geographical areas and ethnic groups in UAE. Also, trend analysis studies and health care delivery needs assessment are needed.

SECTION (8):

REFERENCES

SECTION (9): REFERENCES

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APPENDIX

UNITED ARAB EMIRATES
FACULTY OF MEDICINE AND HEALTH SCIENCE

Date:

Dear Parent,

This questionnaire is part of a survey, the aim of which is to find out the incidence of asthma and related diseases among schoolchildren in the U.A.E.

If your child is not asthmatic you may think there is no deed for you to complete the questionnaire. This is not so: only if every family surveyed responds can we get a true picture of the incidence of asthma in the population. Your co-operation is vital to the success of this study.

Please answer all questions as frankly and accurately as possible.

ALL INFORMATION OBTAINED IN THE STUDY WILL BE KEPT CONFIDENTIAL.

We will be visiting your child's school soon to do a simple breathing test on the children. This test requires the child to take a deep breath and blow out forcefully into a machine. It provides an indication of the size of his/her lungs and whether there is any narrowing of the bronchial tubes.

The test takes only a few minutes.

If you have returned a questionnaire, we will assume we have your approval to test your child, unless you inform the teacher otherwise.

Thank you for your assistance.

Dr. Fareed H. Saleh *MB.BS.*

Supervised by

DR. ABDULBARI BENER
DEPT. OF COMMUNITY MEDICINE
FACULTY OF MEDICINE AND HEALTH SCIENCES.

How to answer the questions:
Some questions require to answer YES or NO. by placing a cross in the appropriate square. Others give you a choice of several answers, and unless specifically told otherwise, you should mark only one square. Others require you to write an answer in the space provided.

Please answer all questions except where it is specifically stated that some should be omitted. If you leave out questions, we may not be able to best use that information you do give us.

We understand that the answers to some questions can only be approximate, and that one memory cannot always be perfectly accurate. Just answer each question as best you can.
If you wish to add more details for any question, please write them in the space provided for comments on the back page. Always place a cross in the most appropriate square, even if you qualify this with extra notes.

Child's on NO : _____
School : _____
Grade : _____
Date of questionnaire complet: _____

I.	Person completing questionnaire:		
	(A)	Mother	
	(B)	Father	
	(C)	Others : (please specify)	

II.	Origin Nationality:		
	(A)	U.A.E.	
	(B)	Omani	
	(C)	Saudi	
	(D)	Yemeni	
	(E)	Other	

III.	Sex of child		
	(A)	Male	

	(B)	Female	

IV.	What is the child's age now?		
	(A)	7 Years.	
	(B)	8 Years.	
	(C)	9 Years.	
	(D)	10 Years.	
	(E)	11 Years.	
	(F)	12 Years.	

V.	Child place of birth		
	(A)	U.A.E	
	(B)	Other.	

HEALTH & GENERAL INFORMATION

1.	Does the child share a bedroom ?		
	(A)	No, has own bedroom	
	(B)	Yes, shares with one other	
	(C)	Yes, shares with two others	
	(D)	Yes, shares with more than two others	

2.	If shared, does any one of the people sharing it smoke cigarettes?		
	(A)	Yes	
	(B)	No.	

3.	How was he/she fed in the first three months of life?		
	(A)	Breast only.	
	(B)	Bottle only.	
	(C)	Breast & bottle only	

4.	Has he/she had more than two sore throats attacks of tonsillitis in the past twelve months?		
	(A)	Yes	
	(B)	No	

5.	Has he/she had the tonsils removed?		
	(A)	Yes	

	(B)	No	

6.	Have you been told by a doctor that he/she is allergic to any foods or medicines?			
	(A)	Yes		
		1)	Food	
		2)	Medicine	
	(B)	No		

7.	Has he/she ever had eczema in the creases (bends) of elbows, wrists, or knees?		
	(A)	Yes	
	(B)	No	

8.	Have you been told by a doctor that he/she had pneumonia or pleurisy?		
	(A)	Yes	
	(B)	No	

9.	Has he/she at any time in his/her life suffered from attacks of asthma or of wheezy breathing?		
	(A)	Yes	
	(B)	No	

(Note: Please regard "asthma" and "wheezy breathing" as being much the same thing for this survey; we do not ask you to try to tell the difference.) The following questions relate to the details of these illnesses and need to be answered if the answer to question (9) was YES. If the answer to question (9) was NO, omit these questions and go on to question (22).

10.	At what age did these attacks begin?		
	(A)	Under 1 year.	
	(B)	Between 1 and 2 years.	
	(C)	Between 2 and 3 years.	
	(D)	Between 3 and 4 years.	
	(E)	Between 4 and 5 years.	
	(F)	Between 5 and 6 years.	
	(G)	Between 6 and 7 years.	
	(H)	Over 7 Years.	

11.	Since the attacks began, approximately how many has he/she had altogether?		
	(A)	One attack only	
	(B)	Two to five attacks	
	(C)	Five to ten attacks	
	(D)	More than ten attacks	
12.	On the average (as near as you can say) how often have these attacks tended to occur over the last two years or so?		
	(A)	About once in 24 hours	
	(B)	About once in a week	
	(C)	About once a fortnight	
	(D)	About once a month	
	(E)	About once every 3 months	
	(F)	About once every 6 months	
	(G)	About once a year (or less often)	
	(H)	No attacks in the last two years	

13.	How long ago did the last asthma occur?		
	(A)	less than 1 months	
	(B)	1-2 months ago	
	(C)	more than 12 months ago	

14.	Does he/she ever get attacks soon after he/she has played hard or exercised?		
	(A)	Yes	
	(B)	No	

15.	In this attack associated with these symptoms?		
	(A)	Wheezing	
	(B)	Breathlessness and tightness	
	(C)	Cough	
	(D)	Sputum	
	(E)	Sputum usually colored	
		1) White	
		2) Yellow	
		3) Green	
		4) with blood	
	(F)	Rhinitis	
	(G)	Tonsillitis	
	(H)	Sneezing	
	(I)	Red eye with itching and watery	
	(J)	Itchy skin	
	(K)	Palpitation	

16.	Is asthma symptoms (e.g. cough, sputum, breathlessness, wheezing increased at :	
	(A)	night
	(B)	day
	(C)	some over the day

17.	Which substances do you think cause the problem for your child?	
	(A)	food
	(B)	Outside dust
	(C)	home dust
	(D)	pollens
	(E)	cold weather
	(F)	Cold drinks or food
	(G)	smoking
	(H)	drug (medicine)
	(I)	animals product
	(J)	psychological stress (problem in the family)
	(K)	I don't know

	(L)	perfumes	
	(M)	exercise	

18.	Does the symptoms relieve by treatment?		
	(A)	Yes	
	(B)	No	

19.	If yes, what kind of treatment?		
	(A)	inhalation	
	(B)	syrup	
	(C)	tablets	

20.	On the average (as near as you can) how long do these attacks last (With usual treatment)?		
	(A)	Less than one hour	
	(B)	More than one hour & less than 12 hours	
	(C)	A day or so	
	(D)	A week or so	
	(E)	2 weeks	
	(F)	A month or so	
	(G)	Continuous (never free from asthma or wheezing)	
21.	Do attacks occur more frequently or more severely during any particular season? (You may mark more than one season).		
	(A)	Spring	
	(B)	Summer	
	(C)	Autumn	
	(D)	Winter	
	(E)	No seasonal difference	

22.	Don you have any pets or livestock in the house or the farm?		
	(A)	Yes	
	(B)	No.	

23.	Does the child have regular contact with pets or livestock?		
	(A)	Cats	
	(B)	Chickens	
	(C)	Camels	
	(D)	Others (please name)____	
	(E)	Dogs	

	(F)	Goats	
	(G)	Birds	

24.	Does your child deal with these pets?		
	(A)	Daily	
	(B)	Occasionally	
	(C)	Never	

25.	Has he/she ever had attacks of "hay fever" (that is, sneezing, runny or blocked nose, sometimes with itchy eyes or nose)?		
	(A)	Yes	
	(B)	No.	

26.	Do these hay fever attacks occur more frequently or more severely during any particular season?		
	(A)	Spring	
	(B)	Summer	
	(C)	Autumn	
	(D)	Winter	
	(E)	No seasonal difference	

27.	Is he/she prone to "colds in the head" (that is, more than two or three colds a year)?		
	(A)	Yes	
	(B)	No.	

28.	Do these colds occur more frequently or more severely during any particular season? (You may mark more than one season).		
	(A)	Spring	
	(B)	Summer	
	(C)	Autumn	
	(D)	Winter	
	(E)	No seasonal difference	

FAMILY MEDICAL HISTORY

It is thought that asthma and related illnesses tend to run in families. We would therefore like to have some information on the history of such illnesses in the child's immediate family. For this information to be useful, we need to know whether the answers to these questions refer to the child's natural parents or not.

29.	Is the male parent the child's natural father?		
	(A)	Yes	
	(B)	No.	

30.	Father			
	(A)	Has a job :		
		1)	Yes	
		2)	No	
	(B)	Educational level:		
		1)	Uneducated	
		2)	Primary level	
		3)	Preparatory level	
		4)	Secondary level	
		5)	University level	

31.	Is the female parent the child's natural mother?			
	(A)	Yes		
	(B)	No		
	(C)	Has a job :		
		1)	Yes	
		2)	No	
	(D)	Educational level:		
		1)	Uneducated	
		2)	Primary level	
		3)	Preparatory level	
		4)	Secondary level	
		5)	University level	

32.	How many brothers and sisters does the child have?			
	(A)	Brothers		
		1)	One	
		2)	Two	
		3)	Three	
		4)	Four	
		5)	More than four	
	(B)	Sisters		
		1)	One	
		2)	Two	
		3)	Three	
		4)	Four	
		5)	More than four	

33.	Has his / her father at any time in his/her life suffered from one of these diseases?			
	(A)	Asthma:		
		1)	Yes	
		2)	No	
	(B)	Hay fever:		
		1)	Yes	
		2)	No	
	(C)	Eczema:		
		1)	Yes	
		2)	No	

34.	Has his / her mother at any time in his/her life suffered from one of these diseases?			
	(A)	Asthma:		
		1)	Yes	
		2)	No	
	(B)	Hay fever:		
		1)	Yes	
		2)	No	
	(C)	Eczema:		
		1)	Yes	
		2)	No	

35.	Has his siblings suffered from one type of this diseases?				
	(A)	Asthma:			
		1)	Yes		
			a.	One	
			b.	Two	
			c.	Three	
			d.	Four	
			e.	More than four	
		2)	No		
	(B)	Hay fever:			
		1)	Yes		
			a.	One	
			b.	Two	
			c.	Three	
			d.	Four	
			e.	More than four	
		2)	No		
	(C)	Eczema:			
		1)	Yes		
			a.	One	
			b.	Two	
			c.	Three	
			d.	Four	
			e.	More than four	
		2)	No		

36.	Has any of his relative suffered from one of these diseases?			
	(A)	Asthma:		
		1)	Yes	
		2)	No	
	(B)	Hay fever:		
		1)	Yes	
		2)	No	
	(C)	Eczema:		
		1)	Yes	
		2)	No	

37.	If the answer is yes, choose one of his/ her relative had suffered from these diseases.			
	(A)	Grandfather		
	(B)	Grandmother		
	(C)	Uncle (father side)		
		1)	Him self	
		2)	Cousin	
	(D)	Aunt (father side)		
		1)	Her self	
		2)	Cousin	
	(E)	Uncle (Mother side)		
		1)	Him self	
		2)	Cousin	
	(F)	Aunt (Mother side)		
		1)	Her self	
		2)	Cousin	

38.	Do your parents smoke?			
	(A)	Father		
		1)	Yes	
		2)	No	
	(B)	Mother		
		1)	Yes	
		2)	No	

39.	How many cigarettes do they smoke per day?			
	(A)	Father		
		1)	less than 5 cigarettes	
		2)	Around 10 cigarettes	
		3)	Around 20 cigarettes	
		4)	Between 20-40 cigarettes	
		5)	More than 40 cigarettes	
	(B)	Mother		
		1)	less than 5 cigarettes	
		2)	Around 10 cigarettes	
		3)	Around 20 cigarettes	
		4)	Between 20-40 cigarettes	
		5)	More than 40 cigarettes	

40.	Does your brother/s or sister/s smoke?			
	(A)	Brothers		
		1)	Yes	
		2)	No	
	(B)	Sisters		
		1)	Yes	
		2)	No	

- END -

Thank you for your co-operation.

Dr. Fareed H. Saleh *MB.BS*.

For contact Tel: + 971 2 672 6613
: + 97150 622 1377
Fax: + 971 2 672 6673
E.Mail drfareed@runbox.com
P.o.box 72622 Abu Dhabi,
U.A.E

ARABIC ABSTRACT

البنـــية. و بعمـــل التحليل الأحصائى اللوجيستيكى المرجعى للعوامل الأجتماعية و الديموغرافية المختلفة و تأثيرها فى حدوث الربو الشعبى تبين ان أهمها كان عمر الطفل ، ترتيبة فى عدد الولادات و كذلك جنسية الطفل.

وأوضـــحت نتائج الدراسة أن (١٥%) من امهات و (١١,٩%) من اباء الأطفال المصابين بالربو الشعبى يعانوا مـــن الأصابةبالربو الشعبى . و بعمل التحليل الأحصائى اللوجيستيكى المرجعى لأصابة بعض افراد الأسرة بالربو الشـــعبى ، تبـــين ان أصابة كل من الأم و الأب و الجدة من اهم العوامل التى تؤدى إلى حدوث الربو الشعبى فى الأطفـــال موضـــع الدراســـة مـــع وجود دليل إحصائى. وأوضحت النتائج وجود علاقة بين التدخين عند الأباء و الأمهـــات و حـــدوث الربو الشعبى فى الأطفال موضع الدراسة مع وجود دليل على ان التدخين السلبى يؤدى إلى زيـــادة فى معدلات الأصابة و كثرة التعرض للازمات عند الأطفال. كما تبين ان متوسط عدد الأفراد داخل الغرفة الواحدة يعتبر من عوامل الخطورة لحدوث الربو الشعبى.

امـــا مـــن ناحية طبيعة التغذية فى الأشهر الثلاثة الأولى من العمر تبين أن غياب الرضاعة الطبيعية تعتبر من العـــوامل التـــي يســـاعد علـــى حدوث الربو الشعبي، و بالإضافة إلى ذلك تبين من التحليل الأحصائى المتعدد أن الرضـــاعة الطبيعـــية المطلقة فى أول أربعة شهور من العمر تعتبر من العوامل الوقائية ضد حدوث الربو الشعبي عـــند الأطفـــال موضـــع الدراسة. كما تم ملاحظة ان وجود حساسية ضد بعض الأطعمة و الأدوية كان اكثر فى الأطفال المصابين بالربو الشعبى عنة فى غير المصابين.

و بدراســـة وجـــود بعـــض الأمـــراض المصـــاحبة مثل الألتهاب الرئوى الحاد، التهاب الغشاء البلورى، التهاب الحويصـــلات الـــرئوية و الدرن تبين ان نسبة الأصابة اكثر فى الأطفال المصابون بالربو الشعبى . بالأضافة الى ذلـــك تبـــين ان الأصابة بحساسية الأنف هو اكثر انواع الحساسية شيوعا بين الأطفال المصابين بالربو الشعبى و الغيـــر المصـــابون . كما تبين أن وجود / او التعامل مع الحيوانات داخل المنزل يعتبر من العوامل التي تساعد على حدوث الربو الشعبي، مع وجود دليل إحصائى.

الملخص والتوصيات:

وقد خلصت الدراسة التى أجريت إلى ما يلى:

١ –العمـــل علـــى تحســـين نوعـــية الحياة عند الأطفال المصابين بالربو الشعبى مع الوضع فى الأعتبار عوامل الخطورة التى أدت الى ذلك.

٢ – التركيز على ايقاف التدخين داخل المنزل وكذلك تجنب وجود او التعامل مع الحيوانات .

٣ – وضع برنامج للتثقيف الصحى خاص بالربو الشعبى فى مدارس الإمارات العربية المتحدة.

٤ – الحاجـــة للمـــزيد من الدراسات المقارنة و المرتكزة على المجتمع لتحديد الاختلافات بين المناطق الجغرافية المتنوعة لدولة الامارات العربية المتحدة والمجموعات العرقية .

٥ – الـــبحث قد أعطى قدر جيد من البيانات النوعية التى يمكن أن تكون ذات استخدام واسع فى أبحاث و تطور برنامج الصحة المدرسية .

عوامل الخطورة البيئية للربو الشعبي في أطفال المدارس الابتدائية بمدينة ابوظبي – الإمارات العربية المتحدة

ملخص الرسالة

الربو الشعبي من أهم المشاكل الصحية التي تؤثر على صحة الطفل على مستوى العالم وهناك مؤشرات على ارتفاع نسب حدوث المرض وكذلك معدل الوفيات منة. وتعتبر العوامل البيئية من عوامل الخطورة المسببة للمرض على الرغم من تضارب النتائج الدالة على ذلك.

وتهدف هذه الدراسة إلى وصف الحالة الوبائية لمرض الربو الشعبي في أطفال المدارس الابتدائية بمدينة ابوظبي – الإمارات العربية المتحدة من اجل الحصول على معلومات تقدم للجهات المسؤلة في وزارة الصحة و صانعي القرار في الصحة المدرسية لاستخدامها في الارتقاء ببرنامج الصحة المدرسية اعتمادا على نتائج ذات جودة مشتقة من الحقل الميداني.

وقد تم تصميم الدراسة بالطريقة الوصفية المقطعية لدراسة عوامل الخطورة البيئية وكذلك معدل انتشار المرض في أطفال المدارس الابتدائية في مدينة ابوظبي – الإمارات العربية المتحدة. وكذلك تم عمل استطلاع ومراجعة للكتابات العلمية الخاصة بهذا الشأن بالإضافة إلى عمل دراسة ميدانية استطلاعية لتحديد كافة المتغيرات الواجب دراستها. وقد تمت الدراسة على ٣٥٢١ طفل ملتحقين بالمدارس الابتدائية في مدينة ابوظبي تتراوح أعمارهم بين السابعة و الثانية عشر من العمر. وقد تم اختيار الأطفال بالطريقة العشوائية المتعددة المراحل.

وأوضحت نتائج الدراسة أن نسبة الأصابة بالربو الشعبي بين اطفال المدارس الأبتدائية في مدينة ابوظبي – الإمارات العربية المتحدة كانت ١٤,٢٨% وأن متوسط أعمار الأطفال المصابين بالربو الشعبي كان (١,٥١± ٨,٧٨) سنة و (١,٥٢±٨,٧٧) سنة للأطفال الغير مصابين وكانت نسبة الذكور (٥٤,٧%) في الأطفال المصابين بالربو الشعبي و(٤٥,٥%) في الأطفال مع وجود دليل إحصائي.

وقد تبين أن عدم وجود وظيفة للأب يعتبر ضمن عوامل الخطورة لحدوث الربو الشعبي مع وجود دليل إحصائي على ذلك. وبالأضافة الى ذلك تبين ان المستوى التعليمي للأب و الأم يلعب دور هام في حدوث المرض ، كما تبين ان التعليم الإبتدائي هو المستوى العلمي الغالب لمعظم الأباء و الأمهات مما يتضح منة ان إنخفاض مستوى تعليم الأباء هو مؤشر لحدوث الربو الشعبي في الأطفال موضع الدراسة مع وجود دليل إحصائي.

وأوضحت النتائج عدم وجود علاقة بين عدد الأخوة و الأخوات وبين حدوث الربو الشعبي في الأطفال موضع الدراسة على عكس ما تبين في دزاسات اخرى ويمكن إرجاع ذلك إلى تباين العوامل الوراثية وإختلاف الظروف

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عنوان الرســـالة

عوامل الخطورة البيئية لظهور مرض الربو الشـــعبي بين طلاب المدارس الابتدائية في مدينة أبوظبي

رســـالة مقدمة من الطالب

فريد حـــسين صالح حـــسين جـــلبي
(M.B.B.S)

الي

جامعة الامارات العربيـــة المتحدة

استكمالاً لمتطلبات الحصول علي درجة الماجستير في علوم البيئة

أشراف:

دكتورة فاطيمه شاد كانيس استاذ مساعد جامعة الامارات العربيـــة المتحدة	بروفسير دكتورسيانا اودمان استشاري قسم الاطفال جامعة الامارات العربيـــة المتحدة

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