**Deanship of Graduate Studies**

**Al-Quds University**

# Risk Factors for Bronchial Asthma among adults in Gaza Strip: Case Control Study

**Wael Omar Matar**

**MPH Thesis**

**Jerusalem-Palestine**

**1439 / 2018**

# Risk Factors for Bronchial Asthma among adults in Gaza Strip : Case Control Study

Prepared By
**Wael Omar Mahmood Matar**

Bachelor of Medicine & Surgery (MB.BS) Sana'a University
Sana'a -The Republic of Yemen

**Supervisor : Dr. Walid Daoud, MBBCh, MSc, MD**
Assistant Professor of Chest Medicine, Al Azhar University-Gaza
Head of Chest Department, Shifa Hospital

Thesis Submitted in Partial Fulfillment of Requirements for the Degree of Master of Public Health/Epidemiology
School of Public Health - Al-Quds University

**1439/2018**

**Al-Quds University**

**Deanship of Graduate Studies**

**School of Public Health**

## Thesis Approval

# Risk Factors for Bronchial Asthma among adults in Gaza Strip: Case Control Study

Prepared By: Wael Omar Mahmood Matar
Registration No. : 21410033

Supervisor: Dr. Walid Daoud

Master thesis submitted and accepted. Date: / /
The names of signatures of the examining committee members as follows:

1. Head of committee: Dr. Walid Daoud Signature……………
2. Internal examiner: Dr. Yehia Abed Signature……………
3. External examiner: Dr. Rami Al-Abadlah Signature……………

**Jerusalem – Palestine**

**1439-2018**

## Dedication

Thanks to ALLAH for all donations in my life that brought me to this level of science.

Then to the greatest persons in the universe post ALLAH my parents.

To the most beautiful and fine heart, my wife

To my sons Omar, Abd-Al Rhman, Mohammad and Ahmad

To my daughter layan

To my brothers and sisters

To all those who encouraged, supported, and helped me all the way

I dedicate this research for all of them

Wael Omar Matar

**Declaration**

I certify that this thesis submitted for the degree of Master, is the result of my own research, except where otherwise acknowledged, and this study (or any part of the same) has not been submitted for a higher degree to any other university or institution.

**Signed:**

Wael Omar Mahmood Matar

Date18/4/2018

## Acknowledgment

First, I would like to thank (ALLAH).

Then I would like express my thanks to Dr. Walid Daoud (Assistant Professor of Chest Medicine, Al Azhar University-Gaza, Head of Chest Department, Shifa Hospital) for his continuous support and great efforts in follow me at time of my study.

Many and great thanks to all members in Al AL-Quds University- Faculty of Public Health specially Dr. Bassam Abo-Hamad, Dr. Yehia Abed and Dr. Khitam Abo-Hamad who supported me in all years at university.

Thanks to Dr.Ghada Al Jadba, (the chief health director in UNRWA health program) who facilitated my research at UNRWA clinics. To the area health officers and senior medical officers for their cooperation in data collection.

Great thanks for my wife (Dr.Wafa Al Amassi) who encouraged me all time of my study.

To my colleagues at UNRWA clinics who I worked with them at the time of data collection and special thanks for all my colleagues at Al-Buriej health center.

Thanks with all respect for the study participants and their families for their involvement in the study.

With respect

Wael Omar Mahmood Matar

# Abstract

Bronchial asthma remains one of common conditions, and potentially serious chronic disease that imposes a substantial burden on patients, their families and the community. It causes respiratory symptoms, limitation of activity, and flare-ups (attacks) that sometimes require urgent health care and may be fatal.

The overall aim of this study is to assess the main risk factors such as demographic, socioeconomic, environmental, psychological and other factors that contribute to bronchial asthma in Gaza Strip, which in turn will help in developing a base line data that can help in developing a preventive program aiming toward reducing the burden of bronchial asthma.

A clinical based case control study was carried out at UNRWA clinics. 214 cases and 214 controls were included in the study with matching the age, gender and address. Data was collected by special structured questionnaire.

The results of this study showed factors which considered as significantly risk factors for bronchial asthma in the Gaza Strip included low level of education increase the risk for bronchial asthma by 21% OR (CI) = 0.795 (0.674 -0.937). Exposure to tobacco smoking increase the risk for bronchial asthma by 66% as OR (CI) = 1.66 (1.007-2.773). Arrjila smoking increase the risk of bronchial asthma by 70% as OR (CI) = 1.742 (1.148-2.643). Also the researcher found that not using things for heating in cold weather is a risk factors for bronchial asthma by17.9% OR (CI) = 1.179 (1.007-2.773). Also not using things for cooling air in hot and humid weather increase the risk for bronchial asthma by 75.7% OR (CI) = 1.757 (1.109-2.785). According to food allergy the researcher found that food allergy increase the risk for bronchial asthma by threefold OR (CI) = 3.001(1.541-6.286). Also family history(genetic) increase the risk for bronchial asthma nearby fourfold OR(CI) = 3.7 (2.071-6.619). Drug allergy increase the risk for bronchial asthma by fivefold as OR (CI) = 5.299 (2.338-12.009). The researcher found the most risky season is summer more than fourfold as OR (CI) = 4.135 (2.743-6.231) then autumn less than 4 fold as OR (CI) = 3.946 (3.065-5.079) next spring three and half fold as OR (CI) = 3.5 (2.718-4.619) and lastly winter more than threefold OR (CI) = 3.271 (3.362-4.530). Exposure to perfume has risky on bronchial asthma by (82.4%) as OR (CI) =1.824 (1.379-2.413) also dust in home has risky on bronchial asthma by (37.6%) as OR (CI) = 1.824 (1.005-1.796). Stress increase the risk for bronchial asthma by 48% as OR (CI) = 1.480 (1.019-2150) and URTI increase the risk by two fold as OR (CI) = 2.029 (1.564-2.63).

The researcher in this study demonstrated positive relation between family size more than 8 member (P value = 0.012), low income less than1000NIS (P value =0.001), not working (P = 0.001), no exercises (P value =0.018), passive smoker (P value= 0.05), living nearby trash containers (P value = 0.001), use of mat to cover land (P value =0.004), painting home with polygad (P value= 0.033), home size less than 140m2 (P value = 0.003), house consist of 2 rooms or less (P value = 0.007), use of flagstones for house land (P value 0.005) also exposure for soil (P = value 0.001), cement (P value =0.001), exhausts fuels from vehicles (P value 0.001), exhausts burn of wood and plastic (P value = 0.001), derivatives of petroleum (P value= 0.001 ), chemical cleaning substance (P value = 0.001), frying of oil(P value= 0.001), entrance of

kitchen at time of cooking (P value = 0.001), mold inside home (P value 0.001), GERD (P value= 0.001), psychological condition (depression and grief) (P value= 0.001) and bronchial asthma.

The study demonstrated that there are relations between bronchial asthma and some factors as type of house that have roof either asbast or zinko, bed room size (2mx2m), cooking out site home, using wood as fuel for cooking, number of persons in bed room more than 4 members, residency nearby either corrals or crusher but not reach statistically significant level .

The study demonstrated that no relations between bronchial asthma and residency nearby electricity station, oven, main street, farms, BMI and manufacturing perfume.

The study is considered as a base line in Gaza Strip, contributes in highlighting the general picture of bronchial asthma risk factors, and provides hints for possible intervention strategies that could reduce the burden of bronchial asthma in Gaza Strip.

# Table of contents

## List of tables

## List of figures



## List of annexes

## List of abbreviation

| | |
|---|---|
| ACQ | Asthma Control Questionnaire |
| AD | Allergic Dermatitis |
| AED | Arab Emirate Dirham |
| AR | Allergic Rhinitis |
| ASA | Acetyl Salicylic Acid |
| BHR | Bronchial hyper responsiveness |
| BMI | Body Mass Index |
| CAD | Coronary artery disease |
| CDC | Centers for Disease Control and Prevention |
| CI | Confidence Interval |
| COPD | Chronic Obstructive Pulmonary diseases |
| ED | Emergency Department |
| EGEA | Epidemiological study on the Genetics and Environment of Asthma |
| EIA | Exercise Induced Asthma |
| ER | Emergency Rome |
| ETS | Environmental Tobacco Smoke |
| FA | Food allergy |
| FHT | Family health team |
| GBD | Global Burden of Disease |
| GDP | Gross Domestic Product |
| GERD | Gastro Esophageal Reflux Disease |
| GS | Gaza Strip |
| GHGs | Green house gases |
| GINA | Global initiative for asthma |
| GNP | Gross National Product per capita |
| ICS | Inhaled corticosteroid |
| ISAAC | International Study for asthma and allergy in children |
| LES | Lower esophageal sphincter |

| | |
|---|---|
| MOH | Ministry of health |
| NAEPP | National Asthma Education and Prevention Program |
| NAMCS | The National Ambulatory Medical Care Survey |
| NCD | Non Communicable Disease |
| NGO | Non Governmental Organization |
| NSAID | Non Steroidal Anti Inflammatory Drug |
| NIS | New Israel Shekel |
| OR | Odd Ratio |
| PHC | Primary Health care |
| SES | Socioeconomic status |
| SHS | Secondhand tobacco smoke |
| SPSS | Statistical Package for Social Science |
| UNRWA | United Nations Relief and Works Agency for Palestine Refugees in Near East |
| URTI | Upper respiratory tract infection |
| USA | United State of America |
| USD | United State Dollar |
| WHO | World Health Organization |
| WP | Water pipe |
| WPS | Water pipe smoke |

# Chapter 1

## Introduction

### 1.1 Back ground

Asthma is a serious global health problem affecting all age groups, it affects more than 300 million people worldwide. Its prevalence is increasing in many countries. Although some countries have seen a decline in hospitalizations and deaths from asthma,2 asthma still imposes an unacceptable burden on health care systems, and on society through loss of productivity in the workplace and disruption to the family. It is characterized by recurrent difficulties with breathing, including symptoms such as shortness of breath, wheezing, chest tightness, and coughing. Asthma symptoms vary over time, and also from individual to individual(Global Initiative for Asthma 2017).

Asthma is characterized by variable symptoms of wheeze, shortness of breath, chest tightness and/or cough, and by variable expiratory airflow limitation. Both symptoms and airflow limitation characteristically vary over time and in intensity. These variations are often triggers by factors such as exercise, allergen or irritant exposure, change in weather, or viral respiratory infections. Symptoms and airflow limitation may resolve spontaneously or in response to medication, and may sometimes be absent for weeks or months at a time. On the other hand, patients can experience episodic flare-ups (exacerbations) asthma that may be life threatening and carry a significant burden to patients and the community. Asthma is usually associated with airway hyper responsiveness to direct or indirect stimuli, and with chronic airway inflammation. These features usually persist, even when symptoms are absent or lung function is normal, but may normalize with treatment (Global Initiative for Asthma 2017).

Asthma is a common, chronic respiratory disease affecting 1–18% of the population in different countries (Global Initiative for Asthma 2017). Around 5%–20% of individuals in Europe suffer from bronchial asthma (Trzcinska H, 2013). Generally we found that the highest prevalence (>20%) was generally observed in Latin America and in English-speaking countries of Australasia, Europe and North America as well as South Africa (The Global Asthma Report, 2014).

Although asthma has many economic effects, the economic cost of asthma is considerable both in terms of direct medical costs (such as hospital admissions and cost of pharmaceuticals) and in terms of indirect medical costs (such as time lost from work and premature death).In 2007, according to data from the CDC National Asthma Control Program economic Burden of Asthma, in the United States, the total societal cost of asthma was estimated at $56 billion. Most of this total ($50.1 billion) was attributed to medical expenses. The economic impact of asthma is inflation-adjusted at 2013 has grown from over $10 billion in 1990 to over $60 billion in 2007 (CDC, 2007).

So, we want to study the triggering factors that may affect control of asthma as socioeconomic, socio demographic, environmental, and psychological factors, as asthma can be controlled with a plan that includes medical treatment and control of these trigger factors in order to reduce the burden of asthma as we can by determining the major factors that aggravate asthma and put recommendations to reduce them and support people with asthma and lead to give them healthy and active lives.

### 1.2 Problem Statement

Although wide variety and increase of prevalence , the rates of deaths due to asthma worldwide have reduced greatly over the past 25 years, but no available therapeutic regimens can cure asthma, and also the burden of asthma will continue to be driven by increasing prevalence(R Beasley, Semprrin, & Mitchell, 2015).

In recent decades, both asthma prevalence and incidence have been increasing worldwide, not only due to the genetic background, but also mainly to the effect of a wide number of environmental risk factors. However, the worldwide economic globalization could also have contributed to a significant increase in the asthma burden, even in developing countries that presented a low disease prevalence in the recent past (Nunes, Pereira, & Morais-Almeida, 2017).

Asthma is generally accepted as a costly illness, the total costs to society (direct, indirect and intangible asthma costs) are difficult to estimate, mainly due to different disease definitions and characterizations but also to the use of different methodologies to assess the asthma socio-economic impact in different societies. The asthma costs are very variables from country to country, however we can estimate that a mean cost per patient per year, including all asthmatics (intermittent, mild, moderate and severe asthma) in Europe is $USD 1,900, which seems lower than USA, estimated mean $USD 3,100 (Nunes et al., 2017).

Global asthma costs disease-related cost is usually classified into direct, in- direct and intangible costs. Direct cost include asthma management(e.g. visits to emergency services;

hospital admissions; medications, including all types of medications, such as over the counter and alternative medicines; outpatient visits, including all human resources involved, such as doctors, nurses, paramedics, psychologists), complementary investigations or treatments (e.g. imaging, skin and blood tests, lung function tests, pulmonary rehabilitation) and other costs (e.g. domestic or professional preventive measures, assistance in home care, transportation to medical visits).Indirect costs include work-related losses (e.g. temporary disability in terms of partial or total lost-days; early disability; permanent disability) and early mortality. Finally, intangible costs are those related with unquantifiable losses, such as the decrease in quality of life, increases in pain or suffering, limitation of physical activities and job changes. These costs, unfortunately, are not yet systematically referenced in the literature on asthma costs. A lot of studies reported data on asthma costs, either at the individual patient, or on the society (regional or country level), and it can be an average annual per asthmatic cost of about $USD 5,000, being much higher in severe asthmatics (Nunes et al., 2017).

Factors that affect asthma are present in the Gaza Strip which increase burden of disease on patient and government because this part of the world has special characteristics. For instance, 1,881,135 million people who are mostly (70%) refugees inhabit this area of 378 Sq Km. This population is highly fertile population and doubles every 15-20 years (PCBS, 2016). It also suffers from a collapse of economy, unemployment 30-40%, below poverty line (fluctuating) 40-85%, GDP per capita 750, GDP decrease, Uncertainty, War, chronic stress and Siege (PCBS, 2013).Therefore, it's necessary to explore the risk factors which commonly contribute to bronchial asthma. This will lead to improve the health status of patient and decrease the burden of asthma.

### 1.3 Justification of study

Asthma is a heterogeneous disease; Asthma is a common and potentially serious chronic disease that imposes a substantial burden on patients, their families and the community. It causes respiratory symptoms, limitation of activity, and flare-ups (attacks) that sometimes require urgent health care and may be fatal (GINA, 2017).

Prevalence of asthma in world is affected by wide variety of factors, but no one is a specific cause. Studies indicate the contribution of both genetic and non-genetic factors, all groups of factors may contribute to the severity and persistence of asthma (Global asthma network, 2014).

The study of risk of factors of bronchial asthma among adult people in Gaza Strip 2016 aims to support treatments strategies that may be considered relevant, to assist in improving symptom control and/or reducing future risk and burden of disease and to introduce recommendations to reduce the effect of these factors if they have correlation.

So, it is an essential subject for nation to study, to decrease the burden of asthma and support the policymakers to put rules to reduce exposure to risk triggers at home, work, and school environment that lead to exacerbation of asthma.

This study is needed to cover the gap of information globally and nationally because no study was done in Gaza Strip about risk factors among adults related to bronchial asthma, so it can be the first building block and base line for researchers, policymakers, students, medical staffs and all community.

A better understanding of the factors that provoke asthma is urgently needed, and this knowledge could be used to develop public health and pharmacological primary preventive measures that are effective in reducing the prevalence of asthma worldwide.

Finally, this study is essential and important for all health sectors in Gaza Strip and Palestine like MOH,UNRWA, NGO and private.

### 1.4 Study objectives

#### 1.4.1 General objective

The purpose of this study is to identify risk factors among adult patient with asthma, which in turn help in developing a base line data that can help in developing a preventive program aiming to reduce the burden of asthma, support people with asthma and lead
Them to a healthy and active life in Gaza Strip.

#### 1.4.2 Specific objectives

To identify the socioeconomic and demographic factors that contributes to bronchial asthma
To identify the environmental characteristics those contribute to bronchial asthma.
To identify psychological factors and others related to bronchial asthma.
To suggest recommendations to avoid or prevent the risk factors which in turn reduce burden of bronchial asthma and subsequently reduce the cost of asthma management in Gaza Strip.

### 1.5 Research questions

1-What are the main risk factors associated with bronchial asthma in Gaza Strip?

2-Is there an association between bronchial asthma and income?

3-Is there a relationship between bronchial asthma and level of education?

4-Is there a relationship between joblessness and bronchial asthma?

5- Does the family size have any effect on bronchial asthma?

6-Is there an association between bronchial asthma and living near the road?

7-Is there a relationship between type of building and bronchial asthma?

8-Does smoking affect bronchial asthma?

9- Is there an association between weather and bronchial asthma?

10-Do the anxiety or depression affect bronchial asthma?

11-Does family history have an effect on bronchial asthma?

### 1.6 Context of the study

#### 1.6.1 Demography and Socio- economic Context:

The GS is located in the Middle East on the eastern coast of the Mediterranean Sea, to the north of Egypt and the west southern edge of Palestine. It is approximately. The territory is 41 kilometers long, and from 6 to 12 kilometers wide, with a total area of 365 square kilometers .The Gaza Strip is one of the most densely populated places on earth(UNDP, 2014) and population of over around 1.8 million Palestinians on some 365 square kilometers (PCBS, 2016).

The total population of Palestine at first 2016 was about 4,816,503 million. The estimated population of GS totaled 1,881,135 million of which 894 thousand males and 866 thousand females. Data revealed that the population of Palestine is a young population; the percentage of individuals aged (0-14) constituted 39.2 % of the total population at first 2016 of which 36.9% in the West Bank and 42.8 % in GS. The elderly population aged (65 years and over) constituted 4.5% of the total population of which 5 % in the West Bank and 3.8 % in GS of 2016 (PCBS, 2016).

Gaza Strip is present at the south of Palestine at the coast of Mediterranean sea, that Egypt board it from the south and others side by Israel Occupation. This very narrow strip suffers for more than 100 years from British Mandate until 2016 though that period a lot of revolutions, intifada, wars and conflicts. All these factors affect the socioeconomic status in the Gaza strip, as poverty rate among Palestinian individuals is 38.8% in Gaza Strip (PCBS.2013).

PCBS.2013 found that population density in Gaza Strip is 4,661 persons/km2 at mid-2013, a very crowded area in the world (PCBS.2013). Also (GDP) per capital decrease from 1540 $ in 1998 to1484 in 2000 to 750 $ in 2013 that occurs specially post internal conflict 2006 and attack of military of Israel at 2008, 2012 and 2014 also we must not forget the siege since 2006. Also recurrent stop of electric supply every day more than 12 hours lead to increase stress and bad indoor pollution.

### 1.6.2 Political Context:

The West Bank and the GS have been under occupation by Israel since 1967. The Palestinian National Authority was established in 1994 following the Oslo agreement. However, there has been ongoing political turmoil and economic decline sparked, in particular, by the second intifada in September 2000 and in 2006 following the election of Hamas. Then ongoing Israeli blockade, imposed in June 2007 after that, these actions crippled the private sector, driving unprecedented numbers of Palestinian into unemployment and poverty. The situation in the GS was further exacerbated as a result of the Israeli military actions during the years 2008, 2012 and 2014.By these wars, vital infrastructure was damaged or destroyed, including manufacturing and commercial units, housing and other buildings, electricity, water and sanitation services. Access to health care for ordinary patients was severely restricted during the conflict and continued as a result of Israeli blockade.

### 1.6.3 Health status context:

Health in the Gaza Strip is deteriorated due to years of socioeconomic decline, conflict and closure have left the health sector across the Gaza Strip lacking adequate physical infrastructure and training opportunities. Facilities are overstretched, and service is frequently interrupted by power cuts. These challenges further threaten the health of the population, which is already at increasing risk (UNRWA, 2014).

Patient in Gaza Strip depends on health care services provided by the Ministry of Health; The Palestinian Ministry of Health has been faced with chronic shortages of drugs, payroll payments, fuel and training due to budget shortfalls, and rising debts due in part to the high expenditures for referral treatments representing 25% of the budget (WHO, 2015).

The Palestinian Ministry of Health and UNRWA together provide extensive geographic coverage of public health and non-profit health care services. However, the burden on households is high (39.8% of health expenditure comes from the general population) and two thirds of health expenditure concern curative care. The restrictions imposed on the movement of patients, health staff and goods have hindered the functioning and development of the health system. In recent years, this has reduced the Ministry of Health's ability to procure adequate stocks of essential drugs and medical disposables.

The main challenge to the health sector before and during the crisis was the availability of drugs and medical supplies; for example at 2014 more than 40% of the essential drug items in the essential drug list and more than 50% of medical consumables were out of stock before and during the crisis.

The Ministry reported that for 2013, an average of 29% of essential drugs and 52% of disposables were out of stock in the Gaza Strip. Although referrals increased by 10% compared with 2012 – in part owing to shortages of medicines – the financial crisis has also lead to an increase in debts to specialized hospitals for the care of patients referred within and outside the occupied Palestinian territory (PCBS, 2013).

### 1.7 Health services:

The Palestinian health care system is a complex mix of different providers operating at both primary and secondary levels. Medical services are provided by 5 sectors: MOH, UNRWA, non-governmental organizations (NGOs), Police medical services, and the private sector.

The Ministry of Health operates the largest network of facilities, with 72 PHC centers and 18 hospitals Gaza Strip. The Ministry of Health provides preventive health services through four primary health care levels. Private and nongovernmental organization hospitals make an important contribution to the provision of tertiary care services. Tertiary services are purchased by the Ministry of Health from the local private sector such as East Jerusalem Hospitals, and from hospitals in Israel, Jordan and Egypt for insured patients with a varying level of copayment. The majority of cases are referred by Ministry of Health medical committees to non- Ministry of Health facilities.

UNRWA operates mainly PHC services and serves those Palestinians, and their descendants, who were displaced in the war of 1948. Nongovernmental organizations operate 26.5% of all PHC centers and 31.1% of hospital (WHO, 2010).

No specific programs for follow up for bronchial asthma at primary and secondary health in Gaza Strip care except chest department at AL-Shifa hospital.

### 1.8 Primary Health Care Centers (PHCs):

Primary health care system (PHC) is a major component of Palestinian health care system; it has provided health care to all Palestinian people especially for children and other venerable groups. PHC centers in Palestine provide primary and secondary health care services as well as tertiary services. PHC centers classified from level 1 to level IV. They offer different health services according to clinic level, of these services.

Epidemiological surveillance and infectious disease management and control maternal and child health. Care of chronic diseases, daily care, family planning, dental, mental services and other services according to center level. The MOH is working with other health sectors in providing the PHC services mainly with UNRWA, and NGOs sector. 672 PHC centers are in Palestine. In the Gaza Strip, there are 54 MOH Primary health care centers (PHC) .UNRWA operates 53 PHC centers in Palestine, 21 PHC centers scattered in refugee camps in the Gaza Strip that have responsibility for helping over 1.8 million refugees in Gaza Strip . The NGO sector operates 178 PHC centers and general centers in Palestine, 57 of them in the Gaza Strip. The health services are distributed throughout Palestine. In addition, MOH provides a number of specific health programs such as health education community involvement community health, immunization, and school health programs (MOH, 2013).

### 1.9 UNRWA

UNRWA is the United Nations Relief and Works Agency for Palestine Refugees in the Near East established by the General Assembly in 1949 following the first 1948 Arab-Israeli War, and became operational in 1950. The UNRWA mandate is to provide assistance and protection to a population of over 5 million registered Palestine refugees. The UNRWA mission is to help Palestine refugees in Jordan, Lebanon, Syria, West Bank and the Gaza Strip to achieve their full potential in human development (UNRWA, 2015). UNRWA provides health, education and relief services that become the largest humanitarian operation in the occupied Palestinian territory. Originally, it intended to provide jobs on public works projects and direct relief for Palestinian Arabs who fled or were expelled by Israel gangs during the fighting that followed the end of the British mandate over Palestine. Today it provides education, health care, social services and emergency aid to some 5 million Palestinian refugees and it has been the main health care provider over 65 years that provides a comprehensive health care from maternal and child health care, infant care and immunizations, school health, oral health, consultations, diagnostic or laboratory services for management of non-communicable diseases (UNRWA, 2013).

UNRWA is the second primary health care provider in the GS that plays an important role in health services delivery, providing free of charge PHC and purchasing secondary and tertiary services for the registered Palestinian refugees through 137 PHC centers, 21 of them located in GS and 1 hospital in Qalgelia in WB. In 2014, UNRWA medical officers provide almost 9.4 million medical consultations totally and almost 4.2 million of them were in GS only, in addition to about 700.000 dental consultations and about 220000 dental screening sessions (UNRWA, 2015).

Moreover, UNRWA provides maternal and child health care through life cycle approach. Maternal health include, preconception care, antenatal care and family planning services. In GS during 2014 the total number of registered pregnant women was 39,546 with ANC coverage 87.3%, the percentage of high and alert pregnancy were 40.1%, Diabetes and hypertension during pregnancy were 2.7% and 10% respectively. Delivery care: number of total reported deliveries was 38096, Reported abortions 2662, percentage of C-Section among reported deliveries 17.4% and 10 maternal deaths.

UNRWA has around 11000 employees in GS, in which health department included around 1001 health workers.

In the recent years UNRWA health program faced many challenges, the main of them was its vertically-oriented health services through maternal and child care, non-communicable disease care and general clinical care such services are not integrated, this challenge faced the epidemiological transition among Palestinian people including refugees with a shift of diseases from communicable ones to non-communicable which appears in the higher rates of diseases including heart diseases, cancer, hypertension and cardiovascular diseases and diabetes mellitus which become the leading causes of death to general population including Palestinian refugees and account for 70 to 80% of causes of deaths (UNRWA ,2013). Addressing the above challenges UNRWA has begun the health reform in June 2011 by adopting the family health team (FHT) approach and e-health as the core strategy of the reform to strengthen primary health care. Family Health Team (FHT) introduced as a primary care package focused on providing holistic and comprehensive primary health care for the entire family, depending on long-term provider-patient/family relationships (person-centered approach). In order to improve effectiveness, efficiency and service delivery UNRWA adapted e-health, which composed of the electronic medical records developed by UNRWA to improve patients' data management and the improvement of the overall health services. The E.Health software contained four main modules: NCD, maternal health care, child health care, and outpatient, in addition to other support modules such as laboratory, pharmacy, dental and specialists (UNRWA, 2015).

# Chapter 2

## 2.Conceptual framework and literature review

This chapter summarizes the arguments, studies and claim pertaining to the main study concepts, which are risk factors of bronchial asthma

### 2.1Conceptual framework

The researcher drew the conceptual framework based on the literature review and personal experience, it shows what the researcher studied, and it consists of four domain demographic factors, socioeconomic, environmental, and psychological and others as shown in figure 2.1.

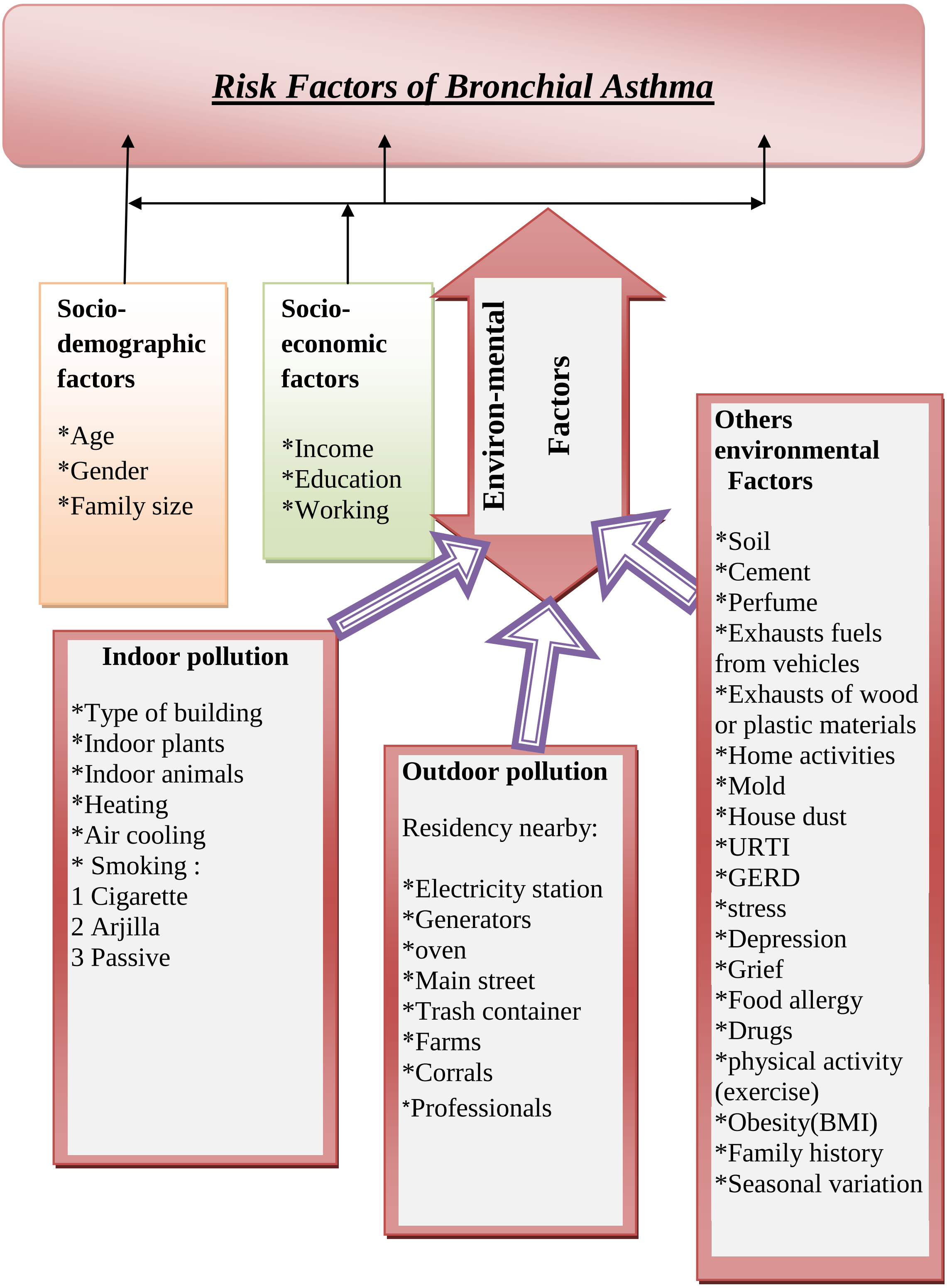


**Figure ( 2.1) Conceptual frame work**

## 2.2 Literature review

### 2.2.1 Definition and diagnosis of bronchial asthma

Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation (Global Initiative for Asthma 2017).

Asthma is characterized by variable symptoms of wheeze, shortness of breath, chest tightness and/or cough, and by variable expiratory airflow limitation. Both symptoms and airflow limitation characteristically vary over time and in intensity. These variations are often triggered by factors such as exercise, allergen or irritant exposure, change in weather, or viral respiratory infections.

Symptoms and airflow limitation may resolve spontaneously or in response to medication, and may sometimes be absent for weeks or months at a time. On the other hand, patients can experience episodic flare-ups (exacerbations) asthma that may be life threatening and carry a significant burden to patients and the community. Asthma is usually associated with airway hyper responsiveness to direct or indirect stimuli, and with chronic airway inflammation. These features usually persist, even when symptoms are absent or lung function is normal, but may normalize with treatment (Global Initiative for Asthma 2017).

**Many phenotypes have been identified, some of the most common include:**

Allergic asthma: this is the most easily recognized asthma phenotype, which often commences in childhood and is associated with a past and/or family history of allergic disease such as eczema, allergic rhinitis, or food or drug allergy. Examination of the induced sputum of these patients before treatment often reveals eosinophilic airway inflammation. Patients with this asthma phenotype usually respond well to inhaled corticosteroid (ICS) treatment.

Non-allergic asthma: some adults have asthma that is not associated with allergy. The cellular profile of the sputum of these patients may be neutrophilic, eosinophilic or contain only a few inflammatory cells (paucigranulocytic). Patients with non-allergic asthma often respond less well to inhaled corticosteroid (ICS).

Late-onset asthma: some adults, particularly women, present with asthma for the first time in adult life. These patients tend to be non-allergic, and often require higher doses of ICS or are relatively refractory to corticosteroid treatment.

Asthma with fixed airflow limitation: some patients with long-standing asthma develop fixed airflow limitation that is thought to be due to airway wall remodeling.

Asthma with obesity: some obese patients with asthma have prominent respiratory symptoms and little eosinophilic airway inflammation (Global Initiative for Asthma, 2017).

### 2.2.2 Burden of bronchial asthma

Asthma is one of the most common diseases in the world resulting in a substantial burden of disease. Although rates of deaths due to asthma worldwide have reduced greatly over the past 25 years, no available therapeutic regimens can cure asthma, and the burden of asthma will continue to be driven by increasing prevalence. The reasons for the increase in asthma prevalence have not been defined, which limits the opportunities to develop targeted primary prevention measures (Richard Beasley, Semprini, & Mitchell, 2015).

Although some countries have seen a decline in hospitalizations and deaths from asthma, asthma still imposes an unacceptable burden on health care systems, and on society through loss of productivity in the workplace and, especially for pediatric asthma, disruption to the family (Global Initiative for Asthma 2017)**.**

In people of all ages, wheeze is the most common symptom. The most recent revised global estimate of asthma suggests that as many as 334 million people have asthma and that the burden of disability is high (Lara, Akinbami, Cheryl & Fryar, 2016)**.**

According to known asthma expenditures for example a recent study estimated medical expenses associated with asthma were $3,259 per person per year during 2002—2007, and the total direct and indirect costs of asthma to society in the United States was $56 billion in 2007.While it is difficult to calculate the exact cost of asthma in Florida, it is important to note that in 2012 the combined charges for ED visits and hospitalizations with asthma listed as the primary diagnosis exceeded one billion dollars ($1,085 million). From 2008 to 2012, the number of asthma ED visits and hospitalizations covered by commercial insurance decreased by 11.1% and 25.8%, respectively, and the number covered by Medicaid increased by 72.5% and 30.4%, respectively. In 2012, Medicaid covered 55.9% of the total ED visits and hospitalizations due to asthma (Forrest & Dudley, 2013)

Another study that done in Arab Emirate in 2013, it found the total direct cost of asthma in Dubai was about 88 million Dirham's (AED 87,917,202). The maximum contribution to this was from expenditure on outpatient visits (37%-AED 32,217,143), followed by that on hospital stays (23% - AED 23,587,008). The cost on medication and ER visits represented 20% and 16% of the direct cost respectively. Absenteeism from school was reported by 50% of asthmatic children, asthmatics also reported an average loss of 4 days of work per year due to asthma. Therefore, there is a huge economic burden on the patients and healthcare services due to asthma. Taking into account the considerable expenditure on the outpatient visits, ER visits and hospital stay, efforts must be directed towards improved asthma control and patient education about their disease. So expenditure due to asthma is a substantial burden on the healthcare system. The cost of asthma for hospital stay, outpatient visits and Emergency visits is considerably higher than that of the medication cost. The efforts should hence be directed towards achieving better asthma control by means of patient education and disease awareness (Mahboub et al., 2013).

Although asthma cannot be cured, it can be treated and controlled. Good asthma control means no, or very minimal, symptoms, and a low risk of asthma attacks or other poor outcomes. A person whose asthma is under good control can go to work or school, exercise, and participate fully in life (Lara, Akinbami, Cheryl & Fryar, 2016)**.**

In people of all ages, wheeze is the most common symptom. The most recent revised global estimate of asthma suggests that as many as 334 million people have asthma and that the burden of disability is high. Overall current asthma prevalence among adults increased from 2001–2002 (7.1%) to 2013–2014 (9.2%) (Lara, Akinbami, Cheryl & Fryar, 2016)**.**

The increasing number of hospital admissions for asthma, which are most pronounced in young children, reflect an increase in severe asthma, poor disease management, and poverty. Worldwide, approximately 180,000 deaths annually are attributable to asthma, although overall mortality rates have fallen since the 1980s. Most asthma deaths occur in those > 45 years old and are largely preventable, frequently being related to inadequate long-term medical care or delays in obtaining medical help during the last attack. The financial burden on patients with asthma in different Western countries ranges from $300 to $1,300 per patient per year, disproportionately affecting those with the most severe disease. There are a number of significant barriers to reducing the burden of asthma, particularly in developing countries, where many patients have limited access to care and essential medications. The Global Initiative for Asthma has outlined a six-point patient management plan to address the effective handling of the increased number of patients in primary care. The plan focuses on patient education, written treatment plans, and ongoing communication and review with patients and their providers (Braman, 2006).

### 2.2.3 Bronchial Asthma prevalence

Asthma is a common, chronic respiratory disease affecting 1–18% of the population in different countries. Asthma is a serious global health problem affecting all age groups. Its prevalence is increasing in many countries, especially among children (Global Initiative for Asthma2017).

In people of all ages, wheeze is the most common symptom. The most recent revised global estimate of asthma suggests that as many as 334 million people have asthma. Overall current asthma prevalence among adults increased from 2001–2002 (7.1%) to 2013–2014 (9.2%) (Lara, Akinbami, Cheryl & Fryar, 2016).

Another study at 2002 demonstrate that the prevalence of bronchial asthma increase all over the word (D'Amato, Liccardi, D'Amato, & Cazzola, 2002).

Another study about Chronic respiratory diseases involve a heterogeneous group of diseases, including, chronic obstructive pulmonary disease (COPD), asthma, sleep apnea syndrome, pulmonary hypertension, and many occupational diseases. They affect more than one billion people worldwide. Their medical, social, and economic impacts are heavy, especially in developing countries such as Middle East and North Africa countries, where they represent a public health problem. They are essentially represented by COPD, asthma, and allergic diseases. Chronic respiratory diseases are increasing in frequency, morbidity, and mortality. In addition, their economic and social impact is increasing rapidly in this region (Ben Abdallah, Taktak, Chtourou, Mahouachi, & Kheder, 2011).

Another study that done in Iran for determines the prevalence of asthma and related symptoms in Middle East countries through review article was to determine the prevalence of asthma in Middle East countries. Data sources Search was carried out in Pub Med, EMBASE, Medline, Web of Science and Google Scholar to get articles which evaluated asthma prevalence in children, adolescents and adults in Middle East countries from 1991 to 2014.The result of this study was the highest asthma prevalence in children and adults was 35.4% in Tehran, Iran by ISAAC phase I and 15% in Kuwait by self-designed questionnaire and the lowest prevalence was 1% in Kermanshah, Iran by ISAAC and 2% in Tehran, Iran by ECRHS questionnaire, respectively. Most studies showed that the prevalence of this common disease declines with increasing age. The study also conclude that the data showed that the prevalence of asthma varies among different countries and even among different cities of the same country (Alavinezhad & Boskabady, 2017).

### 2.2.4 Bronchial Asthma in Palestine

There are some studies in Palestine, one of them case-control study for 400 children aged 3 to 15 years, were studied for environmental risk factors for asthma during 1986 to 1987. It found 98% of the asthmatic patients, exposure to house dust aggravated the symptoms.97% of the cases, the symptoms were more severe at home; in more than 50%, the symptoms persisted throughout the year. Significantly, more cigarettes were smoked in the homes of the allergic children than in the control homes. House dust samples from homes of 20 asthmatic and 20 non-asthmatic children were examined for the presence of house dust mites. Twelve species of mites were identified, of which Dermatophagoides pteronyssinus, the most prevalent (97%), was found in all samples examined. The average number of mites per gram dust was 447 in homes of children with asthma, and 399 in homes of controls. Although dust and mites seem to be the most important factors causing allergies in Gaza and most of the children are exposed to large numbers of mites in their homes, it appears that those who are genetically predisposed and who are exposed to environmental tobacco smoke are at greater risk for allergic symptoms (Abed et al., 1994).

Another study at emergency room of Alia Governmental hospital in Hebron district, south of West Bank, Palestine done to determine the factors associated with chronic asthma severity among asthma patients attending the emergency rooms in Palestine. By cross-sectional study using previously validated questionnaires. Among the 121 patients, 45.5% had moderate/severe asthma. Most days' regular intake of oral theophylline, and using 5 courses/year of oral steroids were more likely to be associated with moderate/severe asthmatics ($p<0.05$). Moderate/severe asthmatics compared with mild asthmatics were more likely to use inhaled short B2-agonists more frequently (most days, 50% vs. 17%; $p<0.05$) and in higher concentrations. They were also more likely to get regular treatment ($p<0.05$) and to report their inability to afford/obtain asthma medicines ($p>0.05$). Also, study concluded access to health services does not necessarily ensure a good quality of care for asthmatics. The effectiveness of oral theophyline in controlling the more severe asthma symptoms should be reconsidered. they recommend a training program for health professionals and an educational one on self-management for the asthma patients (Al Zabadi & El Sharif, 2009).

Another study that aim was to investigate the prevalence and severity of asthma and asthma symptoms in school- children in the Ramallah District in Palestine. In the autumn of 2000, 3,382 schoolchildren aged 6–12 yrs were surveyed in 12 schools, using the International Study for Asthma and Allergies in Childhood (ISAAC) - phase III, parents-administered translated questionnaire. The crude prevalence rates for " wheezing-ever", "wheezing in the previous12 months", and "physician-diagnosed asthma" were 17.1, 8.8 and 9.4% respectively, with urban areas having higher prevalence rates than rural areas. Within urban areas, refugee camps had higher prevalence rates than cities. Yet, within the rural areas, the 12-month prevalence was lower in the deprived villages than other residences. Place of residence remained significant for asthma and asthma symptoms, after adjusting for sex, age, and place of birth. To conclude, children from refugee camps appear to be at higher risk of asthma than children from neighboring villages or cities. The prevalence of asthma and asthma symptoms in Palestine appears to be close to that of Jordan, but it is much lower than Israel, and lower than some other countries in the region, such as Kuwait and Saudi Arabia, and more developed countries. This initial study is a baseline for a study on lifestyle and environmental determinants for asthma among Palestinian children (El-Sharif, Abdeen, Qasrawi, Moens, & Nemery, 2002).

Another study in An-Najah University, Palestine, that found statistically significant association between asthma and gender, smoking, chronic respiratory infections, with a male predominance (Minawi, 2005).

## 2.2.5 Socio-demographic

### 2.2.5.1 Age and gender

Asthma is a serious global health problem affecting all age groups. Its prevalence is increasing in many countries, especially among children (Global Initiative for Asthma 2017)**.**

Gender affects the development of asthma in a time-dependent manner. Until age 13–14 years, the incidence and prevalence of asthma are greater among boys than among girls.
Studies through puberty have shown a greater incidence of asthma among adolescent and young adult females and a greater proportion of males with remission of asthma. Before age 12, boys have more severe asthma than girls, with higher rates of admission to

hospital. In contrast, adult females have more severe asthma than males, with more hospital admissions slower improvement, longer hospital stays and higher rates of readmission. Most authors have attributed these changes in prevalence and severity to events of puberty, although mechanisms for differences between the sexes have not been established (Subbarao, Mandhane, & Sears, 2009).

There are clear-cut gender differences in the prevalence of asthma. Childhood asthma tends to be a predominantly male disease, with the relative male predominance being maximal at puberty. After age 20, the prevalence remains approximately equal until age 40, when the disease becomes more common in females. Reasons for gender differences are unclear and largely unexplored. Possible explanations are first, the greater prevalence of atopy (ie, evidence of IgE sensitization to allergens) in young boys. Second educed relative airway size in boys compared to girls . Smaller airway size may also contribute to the increased risk of wheezing after viral respiratory infections in young boys compared to girls (Litonjua & Weiss, 2016).

In Jordan there is a study that showed the prevalence of asthma is higher in adult male and female than that in both male and female children ($p<0.05$). Also there is no significant difference between adult male and adult female, while there is a significant difference between male children and female children ($p<0.05$). The highest prevalence of asthma was in May and December of each year while the lowest level was in August. So Bronchial asthma is a significant health problem among children and adults in Ma’an province (Hroob, Nawafleh, Altaif, & Elfaqir, 2015).

### 2.2.5.2 Family size

The inverse association between family size and childhood asthma is unique to high-income countries, possibly because of the greater contribution of atopic mechanisms to the pathogenesis of asthma in these populations. However, there is a greater severity of asthma symptoms in larger families, which suggests that risk factors need to be considered in the context of the prevalence of both asthma and severe asthma (Beasley et al. 2015).

Changes in family size over the last 30 years between 1961 and 1991 in England/Wales do not appear to explain much of the reported increase in asthma or hay fever prevalence. The contribution that other risk factors have made to these increases could be assessed with use of a similar approach (Strachan, 2000).

But another study objective that objective to review the “protective” effects of having a higher number of siblings for the risk of atopic eczema, asthma wheezing, hay fever, and allergic sensitization by review of the literature for 53 different studies were identified. The researcher found that the main result as following eczema, 9 of 11 studies reported an inverse relation with number of siblings; for asthma and wheezing, 21 of 31 reported the inverse association, for hay fever, all 17 studies showed the effect; for allergic sensitisation or immunoglobulin E reactivity 14 of 16 studies supported the "protective" effect of a higher number of siblings. The studies emphasise a "theory" that is based exclusively on epidemiological associations. The Conclusions of research have not yet answered the question of which causal factors explain the sibling effect. Causal factors must meet two criteria; they must vary with sibling size and they must protect against atopic manifestations. The prevailing "hygiene hypothesis" failed to explain the findings adequately. Alternative explanations include in utero programming or endocrine explanatory models. The epidemiology research into siblings and atopic disorders has entered an intellectually challenging phase. Possessing sufficient knowledge about the causal factors might prevent at least 30% of all cases of asthma, eczema, and hay fever (Karmaus & Botezan, 2002).

### 2.2.6 Socio economic status (income, working and education)

Socioeconomic status (SES) is defined as an individual's social or economic standing, and is a measure of an individual's or family's social or economic position or rank in a social group. It is a composite of several measures including income, education, and occupation, location of residence or housing. Studies have that found a lower SES has been linked to disproportionate access to health care in many diseases. There is emerging data in pulmonary diseases such as COPD, asthma, cystic fibrosis, pulmonary hypertension and other chronic respiratory conditions that allude to a similar observation noted in other chronic diseases. In the setting of COPD, SES has an inverse relationship with COPD prevalence, mortality, health utilization costs. Asthma and cystic fibrosis show an increased severity and hospitalizations in relationship to a lower SES. Similar observations were seen in sarcoidosis, pulmonary hypertension and obstructive sleep apnea. There remains a limited data on non-CF bronchiectasis and interstitial lung diseases. Population SES may be gauged by various measures such as education, occupation, marital status but no value is more indicative than income (Sahni, Khanijo, Talwar, & Talwar, 2017).

The historical view of asthma being a disease of high-income countries no longer holds: most people affected are in low- and middle-income countries, and its prevalence is estimated to be increasing fastest in those countries (GlobalAsthmaNetwork, 2014).

The prevalence of asthma varies widely around the world, probably because of gene-by-environment interactions (Subbarao, Mandhane, & Sears, 2009).

There is currently no known cure for asthma, but effective strategies to prevent and manage asthma symptoms exist. Individuals with asthma can achieve optimal health outcomes and live normal, active lives with proper clinical treatment, medication adherence, self-management education, environmental control, and trigger avoidance (Forrest & Dudley, 2013).

In 2012, approximately 1.9 million Florida adults (12.6%) reported lifetime asthma. As seen with the national data, the highest prevalence of lifetime asthma in Florida occurred among females, households with lower-income levels, and younger adults (Forrest & Dudley, 2013).

The burden of asthma, measured by disability and premature death, is greatest in children approaching adolescence (ages 10-14) and the elderly (ages 75-79). The lowest impact is borne by those aged 30-34. The burden is similar in males and females at ages below 30-34 years but at older ages, the burden is higher in males. This sex difference increases with increasing age. GBD's measure of health loss attributable to specific diseases, for asthma. The GBD used mortality statistics and health survey data, where available, to estimate, for many countries of the world, two components of disease burden: years of life lost due to premature death, and years of life lived with disability. The latter quantifies both the extent of disability and its duration. The years of life prematurely lost, and the years of life lived with disability are added together and expressed as disability adjusted life years (DALYs), which is the measure of burden of disease. Among people aged less than 45 years, most of the burden of disease is disability. The GBD estimated that asthma was the 14th most important disorder in terms of global years lived with disability. However, for people in older age groups, premature death due to asthma contributes more to the burden of disease. Asthma has a global distribution with a relatively higher burden of disease in Australia and New Zealand, some countries in Africa, the Middle East and South America, and North-Western Europe (Global Asthma, 2014).

The steepest increase in asthma and allergic rhinitis occurred in conscripts with a low socioeconomic status (Hjern, Rasmussen, & Bra, 2005).

Households with the lowest income levels per year had the highest prevalence of lifetime and current asthma (Forrest & Dudley, 2013).

Asthma prevalence in the United States has been reported to be higher in minority groups such as Blacks and Hispanics. Because a disproportionate number of individuals from such minority groups are of low socioeconomic status (SES), it is unclear, how much of the racial/ethnic differences in asthma prevalence is related to low SES. They investigated the effect of SES on the relationship between race/ethnicity and asthma prevalence in a cohort of families with a history of asthma or allergies from the Boston, Massachusetts area. From 499 families, a cohort of 998 parents and 307 children were identified. They used total yearly family income (<$50,000 vs.50, 000), highest level of education (high school vs. College), and residence in high-poverty areas vs. low-poverty areas as measures of SES. Yearly family income <$50,000, high school education, and residence in high poverty areas were all associated with increased risks for asthma in both cohorts. In the parental cohort, Blacks and Hispanics (OR = 2.1, 95% CI = 1.5, 2.8; and OR = 2.2, 95% CI = 1.5, 3.2, respectively) were at greater risk for asthma than Whites. In the cohort of children, Black and Hispanic children (OR = 2.9, 95% CI = 1.0, 8.0; and OR = 5.3, 95% CI = 1.6, 17.5, respectively) were also at increased risk for asthma. When the three measures of SES were included in the multivariable models, the risks associated with Blacks and Hispanics decreased in both cohorts: OR = 1.4, 95% CI = 0.9, 2.0; and OR = 1.6, 95% CI = 1.0, 2.6, respectively, for the parents; and OR = 0.8, 95% CI = 0.2, 3.0; and 2.5, 95% CI = 0.5, 11.7, respectively, for the children. So the study concluded that a large proportion of the racial/ethnic differences in asthma prevalence in our study is explained by factors related to income, area of residence, and level of education (Litonjua, Carey, Weiss, & Gold, 1999).

Another study that to assess whether the association with social class differed between allergic rhinitis and asthma and whether these associations have changed over time. The Swedish Military Service Conscription Register was linked to two other national registers for 1,247,038 male conscripts in successive cohorts born between 1952 and 1977. The percentage of asthma cases associated with allergic rhinitis was 15% in the oldest cohort and 44% in the youngest cohort. Low socio-economic status (SES) was associated with an increased risk (assessed as odds ratio) of asthma without allergic rhinitis (1.14, 95% confidence interval (CI) 1.11–1.17) but a slightly reduced risk of asthma with allergic rhinitis (0.96, 95% CI 0.93–1.00). The risk of allergic rhinitis was 0.84, 95% CI 0.82–0.85. A positive interaction between SES and year of birth occurred in all three conditions. Low SES was related to a reduced risk of asthma with allergic rhinitis in the earliest cohort (0.72, 95% CI 0.53–0.82) but a slightly increased risk in the most recent cohort (1.07, 95% CI 1.01–1.14). In conclusion, the role of social class has changed over time. The steepest increase in asthma and allergic rhinitis occurred in conscripts with a low socio-economic status (Hjern et al., 2005).

National Asthma Education and Prevention Program (NAEPP) guidelines that specify the goals of asthma control and management strategies, find the number of patients with uncontrolled asthma remains high, and factors associated with uncontrolled asthma are unknown. It did study that aim was to examine the relationship between asthma control and socio-demographic characteristics, health-care access and use, asthma education, and medication use among adults with active asthma residing in New England. Methods. Data from the 2006–2007, Behavior Risk Factor Surveillance System Adult Asthma Call-Back Survey were analyzed using multinomial logistic regression. Asthma control was categorized as “well controlled,” “not well controlled,” or “very poorly controlled” according to the NAEPP for3079 subjects. The two of the results in this research that unemployed or unable to work (OR = 17.9; 95% CI = 6.0–53.4), high school educated or less (OR = 2.8; 95% CI = 1.6–4.7), were associated with very poorly controlled asthma of targeted intervention(NGUYEN, ZAHRAN, IQBAL, PENG, & BOULAy, 2011).

Another study described relations between socioeconomic factors and race/ethnicity as risk factors for asthma mortality. A cross-sectional studywas conducted of US mortality records from 1991 through 1996. That results. Higher standardized mortality ratios were seen for Blacks vs Whites (3.34 vs 0.65), low vs high educational level (1.51 vs 0.69), and low vs high income (1.46 vs 0.71). Excess mortality for Blacks vs Whites was present in the highest and lowest quintets of median county income and educational level. The disparity in asthma mortality rates according to median county income and education remained after control for race/ethnicity. Conclusions. Black race/ethnicity appears to be associated, independently from low income and low education, with an elevated risk for asthma mortality (Grant et al. 2000).

The study done in Bangladish demonstrated that the low-income group (OR = 1.41, 95% CI : 1.04–1.92) and illiterate group (OR = 1.51, 95% CI : 1.01–2.24) were more vulnerable to asthma attacks than the high-income group and more educated people, respectively (Hassan, Kabir, Mahmud, Rahman, & Hossain, 2002).

Also, study did in USA fined that Current asthma prevalence differs between groups, with higher rates among those with family income below the poverty level (Akinbami, Moorman, & Liu, 2011).

Global studies on asthma pointed to socioeconomic status as one of the main variables in terms of prevalence and disease severity in various parts of the world. Social factors related to community violence have been linked to higher incidence of asthma in the current studies. The study investigated the relationship between indicators of both community violence and development and hospital admissions due to asthma. This was by an analytical ecological study of multiple groups, using public databases with information up until 2006. All Brazilian municipalities with more than 100,000 inhabitants were considered as units of analysis. Conclusions: There was a direct correlation between indicators of violence and rates of admission due asthma, and an inverse correlation with indicators of development. These results suggested that social detriment can act as a risk factor for hospital admissions due to asthma (Oliveira, Boff, Luiz, & Schaefer, 2015).

### 2.2.7 Environmental Factors

#### 2.2.7.1 Indoor Air pollution

Many people are unaware that indoor air contains pollutants, sometimes at higher percentages than outdoor air. Indoor air can be a source of asthma triggers including cigarette smoke, dust mites, molds, pet dander, gases or fumes, household or industrial chemical irritants, and wood smoke.Considering that Americans spend up to 90% of their time indoors , it is very important for individuals with asthma to take necessary steps to control their indoor environment to reduce asthma triggers (Forrest & Dudley, 2013).

##### 2.2.7.1.1 Building charater

Asthma exacerbation was more frequent among those living in buildings constructed in 1961–1975 in Sweden (OR 2.56, 95%CI 1.22–5.34) (J., K., G., & D., 2016).

According to building that use asbast, post review the recent articles by (Toyoshima, Chida, Suda, & Sato, 2011) concluded that there is no relationship between asbestos exposure alone and airway obstruction. However, one study has reported that asbestos exposure could cause asthmatic symptoms, although the subjects were also exposed to dusts other than asbestos. In addition, serum immunoglobulin E (IgE) elevation has been reported in asbestos-exposed workers and asbestos exposure may cause an atopic condition. They suspect that asbestos exposure itself is a risk factor for the development of bronchial asthma. Bronchial asthma might develop by asbestos exposure independent of dose. Previous studies of nonmalignant respiratory disease caused by asbestos exposure lack appropriate examinations of allergic status, such as sputum eosinophil population, fractional exhaled nitric oxide concentration, bronchial hyperresponsiveness, serum IgE levels, and radioallergosorbent testing. Asbestos exposure may cause airway obstruction by inducing bronchial asthma, even if confirmed asbestosis is absent. To clarify this hypothesis, further investigation will be needed (Toyoshima et al., 2011).

#### 2.2.7.1.2 Indoor animals

The rate of allergies and asthma is increasing in the developed world, as is our attachment and devotion to our pets. This strong attachment to pets often transcends health and other life issues. As such, many patients are reluctant to relinquish a pet, even if they believe that the presence of the animal may compromise their health. These feelings are compounded by the conflicting information that is available through medical and social formats. Despite the large number of studies that have been conducted, there is still no clear scientific consensus on the relationship between animal exposure and allergies and asthma (Chen et al., 2010).

Various studies have indicated that there may be a protective effect when children are exposed to animals from the time of birth, yet many of these studies are contradicted by other studies. Part of the contradictory findings may be explained by having different types of pets or by differing lengths and intensity of the exposure to animals. Because of the relatively low percentage of individuals with allergies, a major limitation of most of the studies conducted to date is the small sample sizes in the subgroups analyzed. This needs to be taken into account during the study design phase, and future studies need to begin with large enough populations in order to have robust samples in each of the subgroups. Seeking to address these shortcomings, large birth cohort studies to measure the development of allergies and asthma in children are currently underway. The results of these studies are needed before many in the medical community will feel confident in knowing how to advise patients on the issue of pet ownership (Simpson &Custovic, 2003). In the meantime, allergic patients and pet owners are placed in a difficult situation. The decision whether or not to retain a pet must ultimately be made by the individual after consultation with a trusted medical provider, and after weighing the potential benefits and consequences of the decision For prospective parents who wish to acquire or keep their animals, the research trends show that early life exposure to animals does not make children more likely to develop allergies or asthma and that there may actually be a protective effect. Thus having animals in the home when children are born may be beneficial to their immune development and is unlikely to be detrimental. However, if children do develop allergies or asthma, the decision of whether or not to keep household pets may need to be reevaluated. For allergic families who wish to keep their pets, there are medications and environmental management strategies which may help control their condition (Trembath, 2015).

#### 2.2.7.1.3 Heating

A population-based case-control study was conducted in Shunyi County, People's Republic of China, in January 1999 and March 2001 to investigate the issue. An increased risk of childhood asthma was associated with use of coal for heating (OR = 1.5, 95% CI: 1.1, 1.9). Those who reported using coal for cooking without ventilation also had an increased risk (OR = 2.3, 95% CI: 1.5, 3.5) (Zheng et al., 2002).

#### 2.2.7.1.4 Air cooling

As Green Leaf A/C and Heating, 2017 find that air conditioning, remove allergens from indoor air. The air in most homes is tainted with allergens, especially during the summertime. Most allergens can trigger both allergies and asthma attacks. These asthma attack triggers include pollen from plants, dust mites, mold, and mildew. Air conditioner units have filters that can improve indoor air quality by removing these allergens. Just ensure you replace the air filter of the unit regularly so that it remains functional and efficient(Green Leaf A/C and Heating, 2017).

Also, air conditioning control indoor humidity another way you can use your air conditioning unit to control asthma attacks is by controlling the humidity in your home. High humidity environments are a major trigger for asthma attacks for most children. This is because humidity can trigger the inflammation of the bronchioles. This inflammation partially closes these air pathways, limiting both the inhalation and exhalation of air . An hour or two before your child's bedtime, you should open your air conditioner to start reducing the humidity in your home(Green Leaf A/C and Heating, 2017) .

#### 2.2.7.1.5 Tobacco Smoke

The global tobacco epidemic may kill 10 million people annually in the next 20–30 years, with 70% of these deaths occurring in developing countries. Current research, treatment, and policy efforts focus on cigarettes, while many people in developing regions (Asia, Indian subcontinent, Eastern Mediterranean) smoke tobacco using water pipe (Maziak, Ward, Afifi Soweid, & Eissenberg, 2004).

Water-pipe smoking (also known as hubble-bubble smoking). This is becoming increasingly popular in some parts of Europe, e.g. Denmark. Although there is little research on this type of smoking, it has been established that it involves the inhalation of nicotine similar to other forms of tobacco use and also the inhalation of carcinogens and toxins. There is emerging evidence of risks to health from this behavior (McNeill, 2011).

Another study that data sources three databases-Pub Med, MEDLINE and EMBASE-were searched until August 2014 for the acute and long-term health effects of WPS using the terms ‘waterpipe’ and its synonyms (hookah, shisha, goza, narghileh, arghileh and hubble-bubble) in various spellings. Several studies of WPS have demonstrated its adverse health effects on many organs but primarily the cardiovascular and respiratory systems where there is documentation of coronary artery disease (CAD) and obstructive pulmonary disease and increased risk to develop lung cancer. WPS has acute and long- term effects on the respiratory system. The former are reflected in increased respiratory rate (RR) and CO, in addition to changes in pulmonary function (PF) and exercise capacity. Chronically, CO levels may be elevated and PF can become permanently altered, leading to chronic obstructive pulmonary disease (COPD). Chronic bronchitis, emphysema and exacerbation of asthma are other pulmonary manifestations of WPS. The association of physician-diagnosed asthma in Lebanon with WPS was of borderline significance after adjusting for cigarette smoking and other variables Mechanisms of WP-induced respiratory disease possible mechanisms of respiratory diseases in WPS were explored in vitro and in vivo studies. WPS resulted in increased airway resistance, lung inflammation, oxidative stress and catalase activity in animal lungs. Rats exposed to WPS over several weeks had higher red blood cell counts and haematocrit, supporting an association with chronic polycythaemia. WP smoke exposure led to decreased neutrophils, lymphocytes, eosinophils and interferon-γ and higher nitric oxide in the bronchi alveolar ravage fluid of asthmatic mice, similar to cigarette smoke exposure, and thus may contribute to asthma exacerbations by suppressing helper T1 cells. In humans, levels of inflammatory cytokines were decreased in the exhaled breath of WP smokers, while the bronchi alveolar lavage fluid of WPS with COPD had increased metalloproteinase two and nine gene expression similar to that of cigarette smokers with COPD. These findings need further investigation to understand their implication to human disease (Ziad M El-Zaatari, Hassan A Chami, 2015).

Bronchial asthma is a common disease and an important cause of morbidity among both children and adults. Tobacco smoking, both active and passive i.e., exposure to environmental tobacco smoke (ETS) has important effects on asthma. Smoking by adults causes bronchial irritation and precipitates acute episodes. It also increases bronchial responsiveness and causes airway sensitization to several occupational allergens. Smoking may also increase the disease severity. Continued smoking by adult asthmatics is the likely cause of irreversibility of airway obstruction and development of chronic obstructive pulmonary disease. ETS exposure affects asthma in a similar fashion. Parental smoking is commonly associated with increased asthma symptoms, respiratory infections, acute episodes and frequent hospitalization of children. Bronchial responsiveness and airway sensitization may also increase. Childhood exposure to smoking is also considered as a risk factor for the development of asthma. Similarly, in utero exposure to maternal smoking may be independently responsible for early onset asthma. ETS exposure in adult asthmatics from smoking by spouses, siblings or colleagues is equally troublesome. There is increased morbidity and poorer asthma control. Asthmatic symptoms sharply decline after the ETS exposure is reduced (Jindal & Gupta, 2004).

Also study at 2017 demonstrated the common risk factors for this disease were genetic predisposition, cigarette smoking, family history of asthma and allergic diseases (Alavinezhad & Boskabady, 2017).

Another study demonstrated that asthma and active cigarette smoking interact to cause more severe symptoms, accelerated decline in lung function, and impaired short-term therapeutic response to corticosteroids. Cigarette smoking may modify inflammation that is associated with asthma .The mechanisms of corticosteroid resistance in asthmatic smokers are unexplained, but could be as a result of alterations in airway inflammatory cell phenotypes (e.g. increased neutrophils or reduced eosinophils), changes in the glucocorticoid receptor-a to -b ratio (e.g. over-expression of glucocorticoid receptor b), and increased activation of pro-inflammatory transcription factors (e.g. nuclear factor) or reduced his tone deacetylase activity. In conclusion, every effort should be made to encourage asthmatics who smoke to stop, although the effects of smoking cessation upon reversing the adverse effects of tobacco smoke on asthma control, therapeutic response to corticosteroids and airway pathology have yet to be fully elucidated. Alternative or additional therapies to inhaled corticosteroids are needed for asthmatic patients who are unable to quit smoking (Thomson, Chaudhuri, & Livingston, 2004).

"An hour a day in a room with a smoker is nearly a hundred times more likely to cause lung cancer in a non-smoker than 20 years spent in a building containing asbestos." Sir Richard Doll, 1985.

Another study that examined the risk of respiratory symptoms related to passive smoke exposure among German adults within the European Community Respiratory Health Survey by the questionnaire data of the population-based sample (n = 1,890) were analyzed. Multiple logistic regression models were carried out for current asthma (asthma symptoms or medication), chronic bronchitis (cough with phlegm for ≥ 3 months per year), and wheezing as dependent variables, and self-reported exposure to passive smoke at home and at the workplace as independent variables after adjusting for city, age, gender, active smoking, and socioeconomic status as well as occupational exposure to dusts and/or gases. The study find the odds for chronic bronchitis were significantly higher in subjects reporting involuntary tobacco smoke exposure in the workplace (odds ratio [OR], 1.90; 95% confidence interval [CI], 1.16 to 3.11). Likewise, the adjusted OR for asthma was slightly elevated (OR, 1.51; 95% CI, 0.99 to 2.32). The risk of chronic bronchitis (OR, 3.07; 95% CI, 1.56 to 6.06), asthma (OR, 2.06; 95% CI, 1.07 to 3.97), and wheezing (OR, 2.12; 95% CI, 1.25 to 3.58) increased significantly with a daily exposure of > 8 h. So the study concluded that the control of passive smoke exposure in the workplace might reduce the risk of respiratory symptoms independently of exposure to other airborne contaminants (Radon et al., 2002).

Study by (Piipari et al. 2004) on the effect of smoking on adulthood asthma has provided contradictory results. The current authors conducted a population-based incident case- control study to assess the effects of current and past smoking on the development of asthma in adults. During a 2.5 yr study period, all new asthma cases clinically diagnosed (n=521) and randomly selected controls (n=932) from a geographically defined district in southern Finland were recruited. The risk of developing asthma was significantly higher among current smokers with an adjusted odds ratio (OR) of 1.33 (95% confidence interval 1.00–1.77) and among ex-smokers with an adjusted OR 1.49 (1.12–1.97) compared with never-smokers. Among current smokers, the risk increased up to 14 cigarettes day-1, and a similar trend was observed in relation to cumulative smoking. In conclusion, the current results support the hypothesis that smoking causes asthma in adulthood.

Tobacco smoking, both active and passive i.e., exposure to environmental tobacco smoke (ETS) has important effects on asthma. Smoking by adults causes bronchial irritation and precipitates acute episodes. It also increases bronchial responsiveness and causes airway sensitization to several occupational allergens. Smoking may also increase the disease severity. Continued smoking by adult asthmatics is the likely cause of irreversibility of airway obstruction and development of chronic obstructive pulmonary disease. ETS exposure affects asthma in a similar fashion. Parental smoking is commonly associated with increased asthma symptoms, respiratory infections, acute episodes and frequent hospitalization of children. Bronchial responsiveness and airway sensitization may also increase. Childhood exposure to smoking is also considered as a risk factor for the development of asthma. Similarly, in utero exposure to maternal smoking may be independently responsible for early onset asthma. ETS exposure in adult asthmatics from smoking by spouses, siblings or colleagues is equally troublesome. There is increased morbidity and poorer asthma control. Asthmatic symptoms sharply decline after the ETS exposure is reduced (Jindal & Gupta, 2004).

Tobacco smoking is the most common cause of chronic airway diseases such as chronic bronchitis (CB) and chronic obstructive pulmonary disease (COPD). On the other hand, asthma is an airway disorder, which commonly starts in childhood. Smoking is not directly responsible as a cause of asthma in most of these patients. There however is enough of evidence, which points towards the adverse effects of smoking on asthma. Both active smoking and exposure to environmental tobacco smoke (ETS) i.e. passive smoking are known to influence asthma in one or the other way. The absence of a demonstrable relationship of smoking with asthma in some earlier studies can be possibly explained because of its irritant effects on the airways triggering acute episodes, for which an asthmatic would tend to avoid smoking. While epidemiological relationship of smoking with asthma in children is known for about two decades, the recent studies from India done on large populations have strongly supported the smoking association of asthma in adults. For example, the Indian study on Epidemiology of Asthma, Respiratory symptoms and chronic bronchitis (INSEARCH) demonstrated odds ratios of 1.82 for cigarette and 2.87 for bide smoking in a sample population of 169575 adults of over 15 years of age.

The other study, which used data on self-reported asthma from the India's third National Family Health Survey (NFHS), reported odds ratios of 1.72 and 1.35 for women and men respectively. In a study, which used the data of Korean Youth Risk Behavior Survey on association of asthma symptoms with smoking and drinking, 21.7% of 3432 adolescent asthmatics were current smokers compared to 10.9% in the asymptomatic group. The epidemiological relationship of ETS-exposure with asthma in children, though variable from different countries, is significantly high in most of the recent studies. Therefore, tobacco smoking has several adverse associations with asthma. The odds ratios for prevalence of asthma are high for both active smoking and ETS exposures. Smoking exposure is known to adversely impact the control of asthma in both children and adults. There is aggravation of respiratory symptoms, acute respiratory infections and bronchial hyper-responsiveness amongst nonsmoker individuals exposed to smoking of parents, sibs and friends.ETS exposure from parents is also related to the severity of asthma and emergency department visits of asthmatic children. In our own study on 200 never-smoker patients of asthma of 15 to 50 years of age, the control was poor and morbidity greater amongst patients exposed to ETS at home and/or at work. Poor asthma control amongst ETS exposed individuals is further supported by the findings of a recent study, which demonstrated a significant reduction in episodes of poor asthma control after a decrease in ETS exposure in children. There were fewer hospitalization and emergency department visits after the caregivers were provided with no-smoking cessation counseling or ETS-exposure education. In a similar study, smoking of caregivers was strongly associated with child exposure to ETS amongst inner-city asthmatic children in Chicago. Measures for smoking cessation amongst the caregivers are therefore important for adequate control of asthma. In another study among 3761 children of below 12 years of age, household ETS exposure was shown to predict asthma attacks for girls (OR: 3.11, 95% C.I. = 1.24 – 1.76) So smoking adversely affects the health and treatment-outcomes of asthma. There are increased requirements of drugs for smoker and ETS exposed asthmatics. Smoking is also an important factor in the development of airway remodeling, fixed airway obstruction and an exaggerated lung function decline (Jindal, 2014).

The relationship between passive smoking and the development of hyperactive airway disease is the subject of intense investigation. In 2006, the US general surgeon reported that exposure to Environmental Tobacco Smoke (ETS) in early life is causal for wheezing. Although the same report indicated that, the role of ETS in asthma onset was still inconclusive. A report by International Study of Asthma and Allergies in childhood

(ISAAC) published in 2012 suggested that the relationship is causal. The Syrian center of ISAAC reported that exposure to narghileh smoke has stronger association with wheezing than exposure to cigarette ETS. In this short review, we will illustrate components of Environmental Tobacco Smoke (ETS) from cigarettes, and then summarize current knowledge on the mechanisms involved in the pathogenesis of wheezing and asthma following exposure to Est. will as well give the essential on ETS of waterpipe.

Environmental Tobacco Smoke (ETS) There are several components of smoke generated by a burning cigarette. First, there is Mainstream Smoke (MSS), which is generated at the cold tip of the cigarette and inhaled directly by the smoker. Second, the side stream smoke (SSS) which is generated at the burning end of the cigarette and emitted directly into the atmosphere. Finally, there is the smoke exhaled by the smoker after inhaling the MSS. This exhaled smoke is referred to as the Exhaled Mainstream Smoke (EMSS). ETS consists of both SSS and EMSS and is inhaled by passive smokers as well as smokers. Smokers, in addition to inhaling MSS, are also exposed to ETS. Wirth, in a general review, reported cigarette ETS to be more toxic than MSS for several reasons. ETS has a low ignition temperature (600°-800°). In addition, several toxic sub- stances such as carbon monoxide, nicotine, volatile organic compounds and benzopyrenes are present in higher concentrations. Finally, particles present in ETS are of smaller size (0.1 µm) which allows them to penetrate deeply into the lungs resulting in oxidative stress and the liberation of Oxidative Reactive Substances, which are among the most critical mediators for airway inflammation (Yousser, 2015).

In 2006, the US general surgeon reported that exposure to Environmental Tobacco Smoke (ETS) in early life is causal for wheezing. Although the role of ETS in asthma onset was still inconclusive. However, a report by International Study of Asthma and Allergies in childhood published in 2012 suggested that the relationship is causal. Particles present in ETS are of smaller size (0.1 µm) which allows them to penetrate deeply into the lungs resulting in oxidative stress and the liberation of Oxidative Reactive Substances, which are among the most aggressive mechanisms for airway inflammation. There are susceptibility genes for asthma in predisposed individuals, however clinical expression might not be apparent unless there is appropriate environmental exposure such as inhalation of parental ETS by children or exposure to allergens. This exposure to ETS leads to epigenetic chang- es. Epigenetic changes are defined as heritable changes that affect gene expression without altering the DNA sequence. ETS seems to influence innate immunity predisposing to asso- ciated respiratory diseases and increasing the risk for IgE-mediated sensitization, which could lead to the inflammatory and structural changes seen in allergic diseases, especially asthma. Exposure to water pipe ETS (locally called Narghileh, Shisha, or Hookah) is to be considered especially in the Middle East. We hope the next WHO report would consider the harm of in-home environmental tobacco smoke exposure, and the need to protect the rights of children worldwide(Yousser, 2015).

Experts believe that smoking is probably the single largest cause of preventable deaths in the United States. Tobacco can be bad for your health even if you are not the one smoking. Environmental tobacco smoke (ETS) is also called "secondhand smoke." This refers to the smoke that is released in the air when a smoker exhales. It also refers to the smoke released from a burning cigarette, cigar or pipe. Tobacco smoke has more than 7,000 chemicals. This includes trace amounts of poisons like formaldehyde, arsenic and cyanide. More than 70 of the substances in tobacco smoke can cause cancer. Many more irritate the lungs and airways. The National Toxicology Program says secondhand smoke is human carcinogen (cancer causing agent).Children are at high risk of lung damage and illness from inhaled smoke. Studies have shown a clear link between secondhand smoke and asthma in children. But the studies have not proven that secondhand smoke causes asthma in children (Asthma and Allergy foundatio of America, 2015b).

Exposure to secondhand tobacco smoke (SHS) also known as environmental tobacco smoke (ETS) has been well established scientifically as a human health hazard. Despite this and warnings from health agencies, concerns over the economic impact of smoke-free bans have limited political resolve to enact these regulations. Arguments against smoke-free bans include the contention that air filters can eliminate the health risks from SHS exposure. In this study, we assessed the effectiveness of air filters commonly used in homes and businesses in reducing the concentrations of total suspended particulates, fine particles and carbon monoxide from SHS as a measure of their potential to reduce the toxicity associated with SHS exposure. Our results demonstrate that these filters are not effective at reducing carbon monoxide levels or PM 2.5, which have been correlated with human health toxicity/disease. Thus, our findings, from a public health perspective, do not support the use of common air filters as available alternative to smoke-free bans (Pritsos & Muthumalage, 2015).

In 2006, the US general surgeon reported that exposure to Environmental Tobacco (ETS) in early life is causal for wheezing. Although the same report indicated that the role of ETS in asthma onset was still inconclusive, a report by International Study of Asthma and

Allergies in childhood (ISAAC) published in 2012 suggested that the relationship is causal. The Syrian center of ISAAC reported that exposure to narghileh smoke has stronger association with wheezing than exposure to cigarette ETS. In this short review, we will illustrate components of Environmental Tobacco Smoke (ETS) from cigarettes, and then summarize current knowledge on the mechanisms involved in the pathogenesis of wheezing and asthma following exposure to ETS. We will as well give the essential on ETS of waterpipe (Yousser, 2015).

Among 2734 pupils (49% females) surveyed, we found an association between exposure to ETS of the mother smoking cigarette or narghileh and ever wheezing, nocturnal cough and severe wheeze; however, the strongest association was found when the mother smoked narghileh. Mother smoking narghileh was also associated with exercise wheeze. Father smoking narghileh, but not cigarettes, was associated with nocturnal cough, severe wheeze and exercise wheeze. The association with current wheeze became significant when mother smoked both cigarettes and narghileh; however, the effect was additive and not synergic (Mohammad et al., 2015)

#### 2.2.7.2 Outdoor pollution

Both the prevalence and severity of respiratory allergic diseases such as bronchial asthma have increased in recent years. Among the factors implicated in this "epidemic" are indoor and outdoor airborne pollutants. Urbanization with its high levels of vehicle emissions and Westernized lifestyle parallels the increase in respiratory allergy in most industrialized countries, and people who live in urban areas tend to be more affected by the disease than those of rural areas(D’Amato et al., 2002).

##### 2.2.7.2.1 Carbon monoxide and NO2 (Electricity station, generators, oven and exhausts product)

Carbon monoxide (CO) is a poisonous gas responsible for hundreds of deaths and numerous non-fatal poisonings each year in the United States. A colorless, odorless, and tasteless gas is produced as a by-product of incomplete combustion of carbon-based fuels such as natural or liquefied propane gas, kerosene, oil, gasoline, wood, or coal. The health impacts of low-level exposures, the actual number of people affected, technologies for measuring levels in the environment or clinical specimens, and mitigation protocols. The literature supports the following findings regarding CO hazards in the home: The severity of health effects from CO exposure depends on various factors, including the age and physical health status of an individual, the duration of CO exposure, and the CO concentration in the air. The elderly, pregnant women, fetuses, young infants, and those with certain pre-existing health problems (e.g., those with cardiac or lung conditions) are most susceptible to health effects from CO exposure. Both short-term exposures to high concentrations of CO and repeated longer-term exposures to lower concentrations of CO can result in serious health effects. Some research shows that repeated exposures to CO, even at levels previously believed to be low, are capable of producing numerous, and persistent, adverse physical, cognitive, and emotional health effects in humans(Peter, Anderson, Menkedick R., & Wooton A., 2005).

Common sources of elevated CO levels in homes include malfunctioning, improperly or inadequately vented gas heating systems(gas heating, gas heating Coal/wood heating Kerosene/oil heating Charcoal grills) and other combustion appliances such as ovens and generators, and cars that are started or left running in attached garages (Peter et al., 2005). Fireplaces and woodstoves, vented space heaters. Malfunctioning or improperly operated unvented appliances including: Kerosene heaters , Unvented space heaters ,Ranges and ovens ,also properly functioning consumer products can pose a CO hazard when they are operated incorrectly, including gas grills or hibachis used indoors or in confined spaces, Gasoline-powered electric generators used in confined spaces ,Gasoline-powered vehicles started or left idling in attached garages, even with the garage door open. Althoug Lack of proper ventilation in attached garages, also Behavior (e.g., idling automobiles in attached garages, using gas ovens for space heating, misuse of heating and combustion appliances, cigarette smoking) (Peter et al., 2005).

Regarding NO 2 inside the home sourses :

Many chemical species of nitrogen oxides (NO2) exist, but the air pollutant species of most interest from the point of view of human health is nitrogen dioxide (NO2). Nitrogen dioxide is soluble in water, reddish-brown in colour, and a strong oxidant. Nitrogen dioxide is an important atmospheric trace gas, not only because of its health effects but also because (a) it absorbs visible solar radiation and contributes to impaired atmospheric visibility; (b) as an absorber of visible radiation it could have a potential direct role in global climate change if its concentrations were to become high enough; it is, along with nitric oxide (NO), a chief regulator of the oxidizing capacity of the free troposphere by controlling the build-up and fate of radical species, including hydroxyl radicals; and (d) it plays a critical role in determining ozone (O3) concentrations in the troposphere because the photolysis of nitrogen dioxide is the only key initiator of the photochemical formation of ozone, whether in polluted or unpolluted atmospheres. Its Sources, emissions of nitrogen oxides from natural sources far outweigh those generated by human activities. Natural sources include intrusion of stratospheric nitrogen oxides, bacterial and volcanic action, and lightning. Because natural emissions are distributed over the entire surface of the earth, however, the resulting background atmospheric concentrations are very small. The major source of anthropogenic emissions of nitrogen oxides into the atmosphere is the combustion of fossil fuels in stationary sources (heating, power generation) and in motor vehicles (internal combustion engines) (WHO, 2000).

Pervious study at 2013 that aimed to quantify the association of indoor NO2 and its main source (gas cooking) with childhood asthma and wheeze. Methods We extracted the association between indoor NO2 (and gas cooking) and childhood asthma and wheeze from population studies published up to 31 March 2013. Data were analysed by inverse- variance-weighted, random-effects meta-analysis. Sensitivity analyses were conducted for different strata. Publication bias and heterogeneity between studies were investigated. A total of 41 studies met the inclusion criteria. The summary odds ratio from random effects meta-analysis for asthma and gas cooking exposure was 1.32 [95% confidential interval (CI) 1.18–1.48], and for a 15-ppb increase in NO2 it was 1.09 (95% CI 0.91–1.31). Indoor NO2 was associated with current wheeze (random effects OR 1.15; 95% CI 1.06–1.25).Conclusions This meta-analysis provides quantitative evidence that, in children, gas cooking increases the risk of asthma and indoor NO2 increases the risk of current wheeze (Lin, Brunekreef, & Gehring, 2013).

Asthma and Allergy foundation of America, 2015 found smoke is a common asthma trigger. There are two sources of smoke to watch out for in the living room: fireplaces and tobacco products. Smoke from wood-burning fireplaces, wood stoves, pellets and “clean burning” stoves can pollute your indoor air. The smoke can contain fine particles that irritate your airways. It also contains nitrogen dioxide and carbon monoxide. These are odorless gases known to cause swelling in the airways in sensitive groups, like those with asthma. It is best to avoid using the fireplace to heat your living room. Gas and kerosene space heaters release nitrogen dioxide too. If they are not vented, these will pollute your air too. In fact, children who were around gas heaters, as babies are more likely to develop asthma. Tobacco smoke is a major asthma trigger and a health hazard for you and those around you. Secondhand smoke causes serious health issues (including asthma) in children and adults (Asthma and Allergy foundatio of America, 2015).

### 2.2.7.2.2 |Main street

According to a growing body of scientific literature, people living near freeways and major roadways have a higher risk of developing (or exacerbating) a wide range of health impacts. A recent comprehensive critical literature review found causal associations between traffic air pollution and asthma exacerbation, and suggestive casual associations with the onset of childhood asthma,cardiovascular mortality and morbidity, and lung cancer (Brauer, Hystad, & Reynolds, 2012).

Motor vehicles emit at least 40 different air pollutants, usually concentrated within 100 – 500 metres of freeways and busy roadways, and research points to a need for increased awareness of the public health concerns associated with roadway proximity in creating land-use policy, building design and environmental/air quality management programs (Brauer et al., 2012).

There was studied a sample of U.S. male veterans drawn from the general population of southeastern Massachusetts. Information on respiratory symptoms and potential risk factors was collected by questionnaire. It assessed distance from residential addresses to major roadways using geographic information system methodology by adjusting for cigarette smoking, age, and occupational exposure to dust, men living within 50 m of a major roadway were more likely to report persistent wheeze (OR) = 1.3; 95% confidence interval [CI] = 1.0–1.7) compared with those living more than 400 m away. The risk was observed only for those living within 50 m of heavily trafficked roads (≥10,000 vehicles/24 h): OR = 1.7; CI = 1.2–2.4) (Garshick, Laden, Hart, & Caron, 2003).

The effect of road vehicle traffic pollution on asthma is still not clearly understood. However, any effect is likely to be most marked among those who live within 150 m of a main road, because this is the distance within which concentrations of primary vehicle traffic pollutants are raised above ambient background levels. They have investigated the relation between proximity of the family home to the nearest main road, estimated objectively using geographical information system software, and the risk of wheeze in the past year in a case-control sample of 6,147 primary school- children (age 4 to 11 yr) and a random cross-sectional sample of 3,709 secondary schoolchildren (age 11 to 16 yr) in Nottingham, United Kingdom. Among children living within 150 m of a main road, the risk of wheeze increased with increasing proximity by an odds ratio (OR) of 1.08 (95% confidence interval [CI] 1.00 to 1.16) per 30-m increment in primary schoolchildren, and 1.16 (1.02 to 1.32) in secondary schoolchildren. Most of the increased risk was localized to within 90 m of the roadside. Among primary school children, effects were stronger in girls than boys (p interaction 0.02) were. Living within approximately 90 m of a main road is associated with a proximity-related increase in the risk of wheezing illness in children (Venn, Lewis, Cooper, Hubbard, & Britton, 2002).

legislation was also passed in New Jersey in 2008 (State of New Jersey, 2008). Called “Terrell James' Law”, it concerned the siting of new schools with respect to highway entry/exit ramps, and states that no new ramp can be constructed within 1,000 feet (approximately 300 m) of an existing school, and vice versa (unless no alternative can be found). In San Francisco, land use guidance suggests a potential hazard exists if average daily traffic exceeds 100,000 vehicles/day within a 200 meter radius of a site (roughly equivalent to the State of California definition above), 50,000 vehicles/day within a 50 meter radius or 10,000 vehicles/day on an immediately adjacent street (Bhatia and Rivard, 2008).

The latter two thresholds are equivalent with respect to area traffic volume density outdoor air pollution is caused by small particles and ground level ozone (smog) coming from car exhaust, outdoor smoke, road dust, and factory emissions. Outdoor air quality is also affected by pollen from trees, plants, crops, and weeds which vary by season. Particle pollution can be high any time of year and is typically higher near busy roads and where people burn wood.When inhaled, outdoor pollutants and pollens can aggravate lung functioning, and may lead to chest discomfort, coughing, digestive problems, dizziness, fever, lethargy, sneezing, shortness of breath, throat irritation, and watery eyes. Outdoor air pollution and pollen may also worsen chronic respiratory diseases, such as asthma.Schools and daycare centers located near busy roadways should take efforts to limit exposure to air pollution by avoiding outdoor activities during heavy traffic times, especially for children with respiratory conditions such as asthma. The Florida Environmental Public Tracking program recently conducted an analysis to determine how many schools and daycare centers were close to busy roadways (Forrest & Dudley, 2013).

#### 2.2.7.2.3 Trash containers

Despite the efforts that are directed to the recycling and recovery of solid wastes, landfills will remain an integral part of most solid waste management plans. Landfills have some adverse environmental impacts including the emission of green house gases (GHGs).

GHGs (mainly methane and carbon dioxide) are the main cause of the global warming phenomena that leads to climate change. Climate change may directly affect human health through increase in average temperature. Furthermore, indirect impact on human health may be caused by climatic change through degradation in water and food quality and quantity. According to USEPA, globally, landfills are the third- largest anthropogenic source of methane emissions, while in the US, landfills were the second-largest source in 2006. In Jordan, domestic solid waste sector contributes to about 90% of the methane emissions from anthropogenic sources at the national level. Nowadays, both governmental and nongovernmental agencies have joined efforts with environmentalists to stop landfills from contributing to global warming phenomena. These efforts are focusing on mitigating the impacts of landfill gases (QDAIS, 2008)**.**

#### 2.2.7.2.4 Residency nearby farms

Generally, farmers and agricultural workers have a lower prevalence of asthma than the general population. This may be because of the healthy worker effect in which those who do not tolerate the dusty work conditions leave that occupation. There is a recent body of literature from Europe and Australia that suggests that children growing up on farms have a lower prevalence of asthma, hay fever, respiratory, and allergic, or atopic, diseases compared to children not raised on farms. It is hypothesized that early exposure to antigens in traditional agricultural operations provides life-long protection against the development of allergy Occupational asthma  is a form of asthma that occurs to an antigen that is unique or present at higher concentrations in the work place (Kirkhorn, Clinical, & Resources, 2000).

Other recent studies of children suggest that factors encountered in a farm environment might protect against the development of allergy. However, it remains uncertain whether living on a farm in childhood is associated with a decreased risk of atopic diseases in adulthood. They analyzed data from 6,251 randomly selected adults 20 to 44 yr of age participating in the European Community Respiratory Health Survey 35. Subjects answered a detailed questionnaire and underwent specific IgE measurements to five allergens. After adjustment for potential confounders, including pet exposure in childhood, number of siblings, severe respiratory infection in childhood, and parental history of allergy, living on a farm in childhood was associated with a reduced risk of atopic sensitization in adulthood (OR 0.76, CI 95% 0.60–0.97). Compared with other adults, those who had lived on a farm as a child were less frequently sensitized to cat (OR 0.63, CI 95% 0.41–0.96) and to Timothy grass (OR 0.68, CI 95% 0.50–0.94), and were at lower risk of having nasal symptoms in the presence of pollen (OR 0.80, CI 95% 0.64–1.02). The protective effect of farming environment in childhood observed in this population-based sample of young adults provides evidence in favor of the hypothesis that environmental factors encountered in childhood may have a lifelong protective effect against the development of allergy (Leynaert, Neukirch, Jarvis, Chinn, & Burney, 2001).

#### 2.2.7.2.5 Residency nearby Main corrals

Animal protein, dust and gases are the major sources that irritate or sensitize the respiratory tract. You could also be directly exposed to a chemical substance through skin contact (Roberge, 2008) .

Allergens are found in secretions from saliva, feces, urine and skin of cats (most common), dogs, rabbits, horses, other furry animals and birds. They are commonly found in upholstery, carpets and clothing (Ferrone, MacPherson, Wilton, & Woychyshyn, 2007)**.**

#### 2.2.7.2.6 Residency nearby professionals and factories

Study at 2016 that found particle pollution could be very dangerous to breathe. Breathing particle pollution may trigger illness, hospitalization and premature death, risks that are showing up in new studies that validate earlier research. Particle pollution is produced through two separate processes—mechanical and chemical. Mechanical processes break down bigger bits into smaller bits with the material remaining essentially the same, only becoming smaller. Mechanical processes primarily create coarse particles. Dust storms, construction and demolition, mining operations, and agriculture are among the activities that produce coarse particles (Billings et al., 2016).

### 2.2.7.3 Other environmental factors

#### 2.2.7.3.1 Soil

Study in 2017 found that industry workers are exposed to asthma- causing substances such as soil components, mold, pesticides, ozone and cleaning chemicals (SHARP, 2017).

Study in 2011 also found the majority of the molds that grow on damp building materials are found in the soil and are adapted to grow on a wide variety of materials. Outdoors, molds live in the soil, on plants, and on dead or decaying matter (AIHA, 2011).

#### 2.2.7.3.2 Cement

The study was conducted in a cement factory in the United Arab Emirates to assess cement dust exposure and its relationship to respiratory symptoms among workers. The prevalence of respiratory symptoms was higher among the exposed workers. The few factory workers (19.5%) who used masks all the time had a lower prevalence rate of respiratory symptoms than those not using them. High dust level was the only variable that influenced the workers to use the mask all the time (Ahmed & Abdullah, 2012).

#### 2.2.7.3.3 Perfume

Study at 2007 that concluded that there is a positive association between exposure to perfume and bronchial asthma as Ferrone et al., 2007 that found and concluded, used of perfume have relation to bronchial asthma and trigger asthma (Ferrone et al., 2007). Also NationalAsthmaConuncilAustralia, 2016 found perfume is one of bronchial asthma triggers (National Asthma Council Australia, 2016).

#### 2.2.7.3.4 Exhausts fuels from vehicles, wood and plastic materials

A study the in the Respiratory Health in Northern Europe cohort constituted the study population. Exposure at each participant's home was calculated using dispersion models. also used, 50 m distance to nearest major road as a simpler indicator of exposure. The adjusted model included 3,609 participants, of which 107 were classified as onset cases and 55 as true incident cases of asthma. There was a positive association between asthma onset (OR) per 10 mg?m-3 1.46, 95% confidence interval (CI) 1.07–1.99) and incident asthma (OR per 10 mg?m-3 1.54, 95% CI 1.00– 2.36) and the levels of nitrogen dioxide (NO2), which remained statistically significant after adjusting for potential confounders. The relationship between asthma and NO2 was not significantly modified by sex, hay fever or wheeze. The risk of developing asthma was also significantly related to living close to a major road. The current study suggests that elevated levels of vehicle exhaust outside the home increase the risk of onset and incident asthma among adults (Modig, Torén, Janson, Jarvholm, & Forsberg, 2009).

The researcher examined the relation between exposure to exhausts burn of wood and exhausts burn of wood and plastic have relation to bronchial asthma.

This result is similar to previous studies as one of them done by asthma and Allergy foundatio of America, 2015 about fireplaces and tobacco products. It found smoke from wood-burning fireplaces; wood stoves, pellets and “clean burning” stoves can pollute your indoor air. The smoke can contain fine particles that irritate your airways. It also contains nitrogen dioxide and carbon monoxide. These are odorless gasses known to cause swelling in the airways in sensitive groups, like those with asthma. It is best to avoid using the fireplace to heat your living room. Gas and kerosene space heaters release nitrogen dioxide too. If they are not vented, these will pollute your air too. In fact, children who were around gas heaters as babies are more likely to develop asthma (Asthma and Allergy foundatio of America, 2015a).

So counseling for community to decrease exposure to sources of NO2 and NO2 by increase health education about the sources of NO2 & CO2 and how to avoid them.

**2.2.7.3.5 Home activities - (household– cleaning products).**

There are some studies that concluded that there is a positive association between exposure to cleaning substance and bronchial asthma. As study did by Sock et al 2007 and Concluded frequent use of common household cleaning sprays may be an important risk factor for adult asthma .Within the follow-up of the European Community Respiratory Health Survey in 10 countries, we identified 3,503 persons doing the cleaning in their homes (Zock et al., 2007) also the same Ferrone et al., 2007 study concluded the chemical cleaning product that used in home have relation to bronchial asthma and trigger asthma (Ferrone et al., 2007).

**2.2.7.3.6 Mold**

Molds are the most common forms of fungi found on the earth. Fungi are classified as neither plants nor animals, and include yeasts, mildews, puffballs, and mushrooms. Most molds re- produce through the formation of spores, tiny microscopic cells that float through the indoor and outdoor air on a continual basis. We are all exposed to mold spores in the air we breathe on a daily basis, both indoors and outdoors. When mold spores land on a moist surface indoors, they may begin to grow and digest the surface. Left unchecked, molds can eventually destroy the surfaces they grow on Molds can be any color. Molds, their fragments, and metabolic by-products have been associated with adverse health effects. Some diseases are known to be caused by specific molds. However, in many occupational settings health conditions suspected to be mold-related cannot be linked to a specific mold as the only possible cause. In a well-, known case an initial finding that Stachybotryschartarum was linked to acute pulmonary hemorrhage/ hemosiderosis in infants living in a water-damaged environment in Cleveland, Ohio was subsequently disproved. Molds are found almost everywhere in our environment, both outdoors and indoors. Their spores float continually in the air we breathe. Molds can grow on just about any substance, as long as moisture and oxygen are available. Mold growth may occur when excessive moisture accumulates in buildings or on building materials including carpet, ceiling tile, insulation, paper, wallboard, wood, surfaces behind wallpaper, or in heating, ventilation, and air The causes of molds in buildings. It is impossible to eliminate all molds and mold spores in the indoor environment. However, moisture control is the most important strategy for reducing indoor mold growth. Common sources of moisture in buildings include plumbing, roof, and window leaks; flooding; condensation on cold surfaces (e.g., pipe sweating); poorly maintained drain pans; and wet foundations due to landscaping or gutters that direct water into or under the building. Water vapor from unvented or poorly vented kitchens, showers, combustion appliances, or steam pipes can also create conditions that promote mold growth .Mold can grow wherever there is dampness. Damp or wet building materials and furnishings should be cleaned and dried within 24 to 48 hours to prevent the growth of mold (OSHA, 2006).

Another study that objective was to investigate whether reported mold or dampness exposure in early life is associated with the development of allergic disor- ders in children from eight European birth cohorts. By analysed data from 31742 children from eight ongoing European birth cohorts. Exposure to mold and allergic health outcomes were assessed by parental questionnaires at different time points. Meta-analyses with fixed- and random-effect models were applied. The number of the studies included in each analysis varied based on the outcome data available for each cohort. The result of this study, exposure to visible mould and/or dampness during first 2 years of life was associated with an increased risk of developing asthma: there was a significant association with early asthma symptoms in meta-analyses of four cohorts [0–2 years: adjusted odds ratios (aOR), 1.39 (95%CI, 1.05–1.84)] and with asthma later in childhood in six cohorts [6–8 years: aOR, 1.09 (95%CI, 0.90–1.32) and 3–10 years: aOR, 1.10

(95%CI, 0.90–1.34)]. A statistically significant association was observed in six cohorts with symptoms of allergic rhinitis at school age [6–8 years: aOR, 1.12 (1.02–1.23)] and at any time point between 3 and 10 years [OR, 1.18 (1.09–1.28)]. Conclusion: These findings suggest that a mouldy home environment in early life is associated with an increased risk of asthma particularly in young children and allergic rhinitis symptoms in school-age children (Tischer et al., 2011).

#### 2.2.7.3.7 House dust

Indoor dust, unlike outdoor dust, consists of a combination of cotton fibers, cellulose, mattress parts, human and animal hair and dander, mould spores, dead insect bodies and minute scraps of food. Depending upon what part of the country you live in and elements around you, the composition of dust will vary but in general, many of the components are similar (Asthma.ca, 2016).

Around 30 to 40 percent of the contaminants inside your home are brought in from outdoors. They can enter your home on your shoes, clothing, or your pets can track them in on their paws and fur. Not surprisingly, the greatest concentration of household dust is Found in carpeting near the entryway(Turner, Gibson, & Reed, 2010).

Dust is found almost everywhere indoors. It is airborne, but settles on surfaces such as open shelving, window ledges, curtains, blinds, upholstery, carpets and can be carried in many ways, including: human hair, animal dander, dead insect bodies, food scraps, chalk dust and talcum powder and trigger bronchial asthma (Ferrone et al., 2007).

#### 2.2.7.3.8 Upper respiratory tract infection

Viral respiratory tract infections are frequent and usually self-limited illnesses. For patients at risk for asthma, or with existing asthma, viral respiratory tract infections can have a profound effect on the expression of disease or loss of control. New evidence has shown that wheezing episodes early in life with the common cold virus, human rhinovirus, is a major risk factor for the later diagnosis of asthma at age six years. For those with existing asthma, exacerbations are a major cause of morbidity, need for acute care and, rarely, death. Viral respiratory tract infections, most frequently with rhinovirus, are the predominant microorganisms associated with asthma exacerbations. Evidence is also emerging that deficiencies in antiviral activity and the integrity of the airway epithelial barrier may make individuals with asthma more likely to have severe viral respiratory infections of the lower airway, and thus increase the risk of exacerbation (Busse, Lemanske Jr., & Gern, 2010).

It is still uncertain how viral respiratory infections cause acute exacerbations of bronchial asthma, although several mechanisms have been proposed. We studied the relationship between the airway narrowing and the inflammatory and bronchospastic factors in peripheral venous blood and urine, in 30 patients with asthma at the exacerbations caused by upper respiratory tract infections (URTIs). Acute exacerbations caused decreases in peak expiratory flow rate (PEFR) in all 30 patients with asthma. Asthma exacerbations caused the rises in serum levels of interleukin-6, soluble intercellular adhesion molecule-1 and eosinophilic cationic protein, concentrations of urinary leukotriene E4 and plasma histamine, compared with those in patients with asthma at a stable condition and those in 30 control subjects ($p < 0.05$). The values of PEFR at the exacerbations correlated with the levels of these factors. Treatment with oral glucocorticoids reversed the decreases in PEFR and the increases in these factors. At the onset of URTIs, rhinovirus and influenza type A virus were identified in 13 and 7 patients, respectively. Each of par influenza virus, adenovirus, and enterovirus was identified in one patient. These findings suggest that respiratory viral infections may cause acute asthma exacerbations via the production of mediators that induce inflammation and bronchospasm (Yasuda et al., 2005).

Rhinovirus induced acute asthma is the most frequent trigger for asthma exacerbations.

That assessed by inflammatory mediators were released from bronchial epithelial cells after infection with rhinovirus Results: IFN-g–induced protein 10 (IP-10) and RANTES were released in the greatest quantities, followed by IL-6, IL-8, and TNF-a. the study concluded the IP-10 release is specific to acute virus-induced Asthma (Wark et al., 2007).

**2.2.7.3.9 Gastroesophagealreflux disease (GERD)**

(GERD) is a common ,chronic, relapsing condition that carries a risk of significant morbidityand potential mortality from complications. While many patients self-diagnose, self- treat and do not seek medical attention for their symptoms, others suffer from more severe disease with esophageal damage ranging from erosive to ulcerative esophagitis.

More than 60 million adult Americans suffer from heartburn at least once a month and over 25 million experience heartburn daily. The National Ambulatory Medical Care

Survey (NAMCS) found that 38.53 million annual adult outpatient visits were related to GERD. For patients presenting with GERD symptoms, 40-60% or more have reflux esophagitis. Up to 10% of these patients will have erosive esophagitis on upper endoscopy. GERD is more prevalent in pregnant women and a higher complication rate exists among the elderly. Patients with GERD generally report decreases in productivity, quality of life and overall well-being(Standiford, Grant M Greenberg, & Harrison, 2013).

Asthma and GERD are common conditions that often coexist with 50-80% of asthmatics having GERD and up to 75% having abnormal pH testing. However, only 30% of patients who have both GERD and asthma will have GERD as the cause for their asthma. The causal relationship between asthma and GERD is difficult to establish because either condition can induce the other (GERD causing asthma as above, and asthma causing increased reflux by creating negative intrathoracic pressure and overcoming LES barrier). Furthermore, medications used for asthma, such as bronchodilators, are associated with increased reflux symptomatology. Historical clues to GERD-related asthma may include asthma symptoms that worsen with big meals, alcohol, and supine position, or adult-onset and medically refractory asthma. Diagnostic testing with pH probe and EGD have limited utility in establishing causality in this population (Standiford et al., 2013).

The prevalence of GERD increases in asthmatics compared with normal controls. The prevalence of GERD in asthmatics is estimated at between 34% and 89% which probably reflects the variation in GERD definition and populations studied. Although a proportion of patients with GERD have no obvious symptoms, data showed that, among the asthmatics, 77%, 55%, and 24% experienced heartburn, regurgitation, and swallowing difficulties, respectively perform edendoscopy and esophageal biopsy on 186 adult asthmatics, and revealed that 43% of the patients with asthma had esophagitis or Barrett’s esophagus, or both. In healthy volunteers, only 13.8% have abnormal endoscopic findings in esophagus, and 8.5% presented with erosive esophagitis. Measured lower esophageal sphincter (LES) pressure and studied gastroesophageal reflux (GER) patterns using 24-h esophageal pH monitoring in 44 controls and 104 adult asthmatics. They found that 82% of adult asthmatics had abnormal gastroesophageal reflux. Compared with the controls, asthmatics had significantly lower LES pressure, greater acid exposure time, more frequency reflux episodes, and longer clearance times in both the upright and supine positions.

Several studies have demonstrated that significant proportion of patients with asthma suffered GERD without classic GERD symptoms of heartburn or regurgitation. Harding et al1 reported that 24-h esophageal pH tests were abnormal in 29% asthmatics without reflux symptoms. Another study performed by Harding et al20evaluated 26 patients with stable asthma without reflux symptoms using esophageal manometry and 24-h esophageal pH testing, and found that the prevalence of abnormal 24-h esophageal pH tests in asthmapatients without reflux symptoms was 62%. Al-Asoom et al16found that 36.4% of asthmatic patients diagnosed by esophageal pH moni- toring as having GER did not complain of heartburn and hoarseness of voice; such as the reflux was silent.

GERD may induce or aggravate asthma. A cross-sectional international population survey in 2,661 participants showed that, compared with those without GERD, individuals with GERD had increase risk of pulmonary conditions like wheezing, breathlessness at rest and nocturnal breathlessness. In this study, association of GERD with physician-diagnosed asthma was marginally significant (OR = 2.2; 95% CI = 1.04-4.70) El-Serag et al22 studied101,366 patients with erosive esophagitis or esophageal stricture and found that, compared with the controls, patients with reflux esophagi- tis were at an increased risk of asthma (OR = 1.5; 95% CI = 1.4-1.6). Recently, El-Serag et al23 conducted a large case-control study in 1980 children with GERD and 7920 controls without GERD, and reported GERD was a significant risk factor for asthma (OR=1.9; 95% CI = 1.6- 2.3). A cross-section study reported that, in the week prior to completing the questionnaire, 41% of the asthmatics noted reflux-associated respiratory symptoms, including cough, dyspnea, and wheeze and 28% used their inhalers while experiencing GER symptoms; inhaler use correlated with the severity of heartburn ($r = 0.28$, $p < 0.05$) and regurgitation ($r = 0.40$, $p < 0.05$). Using the Medline 1966-1999 database,reviewed the studies on the relationship between GER and asthma, and concluded that GERD caused asthma symptoms but had minimal effects on pulmonary function. Asymptomatic GERD did not worsen asthma. Antireflux therapy might have a role in asthma patients with symptomatic GERD, possibly being most beneficial for those with reflux associated respiratory symptoms. Both medical and surgical antireflux therapy could improve asthma symptoms and asthma medication requirements without improving pulmonary function (Huang & Jiang, 2005).

Another study, that demonesterate association between gastroesophageal reflux disease (GERD) and asthma is well accepted. The prevalence of GERD increases in asthmatics compared with normal controls, whereas GERD may induce or exacerbate asthma. They interact with each other in a cause and effect relationship. But the mechanism by which GERD might induce or aggravate asthmatic symptoms remains unclear. Two mechanisms have been proposed, including (1) acid in the inflamed esophagus acting on exposed receptor causes an increase in bronchial hyper-responsiveness via the vagal reflex; (2) microaspiration of gastric contents damage the bronchial mucosa, which result in inflammation of the mucosa and bronchial hyper-responsiveness. Among the GERD diagnostic methods, ambulatory esophageal pH monitoring bears the highest sensitivity. Ambulatory esophageal pH monitoring is recommended in patients without classic reflux symptoms or those with difficult to control asthma. Both medical and surgical antireflux therapy could improve asthma symptoms, asthma medication requirements, and even pulmonary function in a proportion of asthmatics (Huang & Jiang, 2005).

#### 2.2.7.3.10 Psychological factor (depression, stress and grief )

Despite the availability of effective therapies, research indicates that over 50% of asthmatics are poorly controlled. Poor asthma control has been linked to several psychological (i.e., anxiety, depression) and behavioral factors (i.e., cigarette smoking, obesity), though little is known about the interaction of anxiety and depression and smoking on asthma control in adult asthmatics. This study assessed the interactions between anxiety and mood disorders and current cigarette smoking on asthma control in a large sample of asthmatics. Methods: 796 confirmed adult asthma patients completed a sociodemographic and medical history interview, including questions about current and past smoking status, and a brief structured psychiatric interview evaluating anxiety and mood disorders using the Primary Care Evaluation of Mental Disorders (PRIME-MD). Asthma control was evaluated using the Asthma Control Questionnaire (ACQ)Results: After adjusting for age, sex, and asthma severity, general linear model analyses indicated a significant association between current smoking and ACQ score (B (SE) = 0.336 (0.147), p=.022), such that patients who are currently smoking had worse asthma control relative to non-smokers. There were significant associations between anxiety disorders and asthma control (B (SE) = 0.317 (0.093), p=.001), and between mood disorders and asthma control (B (SE) = 0.451 (0.095), p= > .001), such that patients who had an anxiety disorder had worse asthma control compared to those without an anxiety disorder, and patients who had a mood disorder had worse asthma control relative to those without a mood disorder. There were no interaction effects between current smoking and anxiety or mood disorders on asthma control. Discussion: Findings suggest that current smoking, anxiety and mood disorders alone are associated with poorer asthma control, but that having an anxiety or a mood disorder and being a current smoker do not confer any additional risk to asthma control (Ouellet, Boudreau, Plourde, Bacon, & Lavoie, 2012).

Asthma has long been considered a condition in which psychological factors have a role. As in many illnesses, psychological variables may affect outcome in asthma via their effects on treatment adherence and symptom reporting. Emerging evidence suggests that the relation between asthma and psychological factors may be more complex than that, however. Central cognitive processes may influence not only the interpretation of asthma symptoms but also the manifestation of measurable changes in immune and physiologic markers of asthma. Furthermore, asthma and major depressive disorder share several risk factors and have similar patterns of deregulation in key biologic systems, including the neuroendocrine stress response, cytokines, and neuropeptides. Despite the evidence that depression is common in people with asthma and exerts a negative impact on outcome, few treatment studies have examined whether improving symptoms of depression do, in fact, result in better control of asthma symptoms or improved quality of life in patients with asthma (Lieshout & Macqueen, 2008). As there is growing awareness of the correlation between psychological factors, the course of asthma, and the outcomes of asthma treatment. The implications of this correlation are still poorly understood. In 2011 the researcher study the role of anxiety and depression in asthma with a focus on recent literature. Recent studies suggest an interaction between behavioral, neural, endocrine, and immune processes and suggest that psychological factors play an active role in the genesis of asthma. Notably, the role of chronic stress has been investigated, including the parental psychological state during pregnancy. There is evidence that in patients with asthma, such stress may induce hypo responsiveness of the hypothalamus-pituitary-adrenal axis, resulting in reduced cortisol secretion even though it is generally accepted that anxiety and depression are more common in asthmatic patients and that there is a close correlation between psychological disorders and asthma outcomes, such as poorer control of asthma symptoms, the implications and practical consequences of this link remain weak. New studies are introducing an intriguing model of the links between emotional stress, brain centers, the immune system, and the hypothalamus-pituitary-adrenal axis that is far removed from the original concept of 'asthma nervosa (Di Marco, Santus, & Centanni, 2011).

In addition, Bender, 2006 find, there is some evidence that both depression and risk behavior are associated with non-adherence to medications, poor treatment outcomes, and death. The relationship between depression and asthma may involve more than one causal pathway and includes the possibility that depression can lead to a sense of hopelessness that erodes adherence and other health- promoting behavior, or that depression impacts asthma directly by altering the immune system. An assessment of the interplay between risk behavior, depression, adherence, and asthma can add important new information to our understanding about how to identify and treat those at greatest risk for poorly controlled disease and asthma-related death (Bender, 2006).

(Di Marco et al., 2011) study anxiety and depression in asthma patients: impact on asthma control .There is evidence that asthma is associated with an increase in psychiatric symptoms and mental disorders. This association can make it difficult to achieve asthma control at the Asthma Outpatient Clinic of the Federal University of São Paulo Hospital São Paulo, in the city of São Paulo, Brazil. The patients were divided into two groups by asthma control status, as assessed by the asthma control test, and were subsequently compared in terms of demographic, clinical, and spirometric data, as well as scores for asthma quality of life and hospital anxiety/depression. The prevalence of anxiety and of anxiety& depression was significantly higher among patients with uncontrolled asthma than among those with controlled asthma (78% and 100%; $p = 0.04$ and $p = 0.02$, respectively), whereas there were no differences between the two groups in terms of the prevalence of depression, spirometry results, or quality of life score. In this sample, the prevalence of anxiety symptoms was higher in the patients with uncontrolled asthma than in those with controlled asthma. In the evaluation of asthma patients, the negative impact of mood states ought to be taken into consideration when asthma control strategies are being outlined (Di Marco et al., 2011).

Psychiatric symptoms of anxiety, depression and cognitive dysfunction often occur in patients suffering from somatic conditions such as asthma and chronic obstructive pulmonary disease (COPD) which constitute a major and growing public health problem. In the present study, we therefore aimed at analyzing depressive symptoms as well as symptoms of anxiety and cognitive problems in patients with mild to moderate asthma and COPD. 59 participants-17 with asthma, 24 with COPD and 18 healthy controls were en-rolled. Depressiveness was assessed with the beck depression inventory; anxiety symptoms were measured with the State-Trait Anxiety Inventory Part 1 and 2, and cognitive function levels were estimated with the Trail Making Test Part A and B. A score above the threshold indicative for depression was found by 33 % (n = 8) of COPD patients, 29 % (n = 5) of asthma patients compared to 0.05 % (n = 1) of the control group. Clinically relevant anxiety levels were found in 42 % (n = 10) of the COPD group, 41 % (n = 7) of the asthma patients and 17 % (n = 3) of the controls. Patients with COPD performed significantly worse on the Trail Making Test than other groups. Psycho emotional state and cognitive functions were found to be correlated with exposure to tobacco smoke (measured in pack-years) and airway obstruction (measured with FEV1). In conclusion, patients with mild to moderate asthma and COPD exhibit significantly higher levels of depressive and anxiety symptoms as well as cognitive dysfunctions than controls. The prevalence of these symptoms is related to the amount of exposure to tobacco smoke and the severity of airflow obstruction (beyondblue and Asthma Australia, 2015).

Chronic stress and adverse life events appear to trigger exacerbations in children with asthma, and asthma-related health status is independently correlated with anxiety and depression scores (Thomas & Griffith, 2005).

Risk behaviors, including tobacco, alcohol, and drug use, are common in adolescents and young adults. Those who engage in one risk behavior are likely to engage in additional health risk behaviors, and as the number of risk behaviors increase, depression co morbidity emerges. For young people with chronic illness, risk behavior and depression are also common. There is some evidence that both depression and risk behavior are associated with non-adherence to medications, poor treatment outcomes, and death. The relationship between depression and asthma may involve more than one causal pathway and includes the possibility that depression can lead to a sense of hopelessness that erodes adherence and other health- promoting behavior, or that depression impacts asthma directly by altering the immune system. An assessment of the interplay between risk behavior, depression, adherence, and asthma can add important new information to our understanding about how to identify and treat those at greatest risk for poorly controlled disease and asthma-related death. New behavioral studies must overcome the shortcomings frequently seen in previous research and include well-designed and controlled investigations using valid behavioral measures of risk behavior, mood disorder, and adherence; recruitment that includes sufficient numbers of subjects and gives careful consideration of selection bias; and employment of multivariate data modeling to allow for simultaneous statistical examination of multiple relationships (Bender, 2006).

### 2.2.7.3.11 Food allergy

Food allergy is a trigger for asthma symptoms (< 2% of people with asthma). In patients with confirmed food- induced allergic reactions (anaphylaxis), co-existing asthma is a strong risk factor for more severe and even fatal reactions. Food-induced anaphylaxis often presents as life-threatening asthma (Global Initiative for Asthma,2017).

National Asthma Conuncil Australia at 2016 demonstrated that some dietary can trigger exacerbating bronchial asthma (National Asthma Council Australia, 2016).

Children with allergy to food have a four –fold increase likelihood of having asthma compared with children without food allergy (GINA, 2015).

United States population-level association between FA and severe asthma exacerbations was also identified (Liu et al., 2010).

As there are many phenotypes of asthma one of them is allergic asthma and one is characterized by some association with is allergy to food also confirmed food allergy is a risk factor for asthma related deaths (GINA, 2015).

### 2.2.7.3.12 Drug allergy

About 10–20% of adults with asthma suffer from exacerbations in response to baby aspirin or NSAIDs that inhibit cyclooxygenase-1. This condition is more common in patients with severe asthma and poor lung function. The majority of the patients experience first symptoms during their third to fourth decade of life. Once ASA or NSAID hypersensitivity develops, it is present for life. Characteristically, within minutes to 2 h following ingestion of ASA, an acute severe asthma exacerbation develops. It is usually accompanied by rhinorrhea, nasal obstruction, conjunctival irritation, and scarlet flush of the head and neck. A typical history of reaction is considered adequate for diagnosis of ASA-induced asthma. Patients known to have ASA-induced asthma are recommended to avoid all ASA-containing products and NSAIDs. Where an NSAID is strongly indicated, alternative analgesics such as paracetamol are recommended. Prophylactic low-dose ASA is recommended also to be avoided; however, patients for whom ASA is considered essential can be referred to an allergy specialist for ASA desensitization.ASA and NSAID can be used in asthma patients who do not have ASA-induced asthma. Montelukast may be protective against this type of asthma and, therefore, is recommended to be part of the treatment regimen (Saudi Thoracic sociaty, 2016).

As there are many phenotype of asthma one of them is allergic asthma and one of characteristic by some association as  is allergy to drug (GINA, 2015).

### 2.2.7.3.13 Physical activities (exercise)

In 2012, adults with current asthma had a significantly lower prevalence of participating in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise during the past month compared to adults who never had asthma (77.9%) and adults with former asthma (75.3%) (Forrest & Dudley, 2013)**.**

Another study at 2012 noticed that EIA might be observed in children and adolescents of different physical conditioning backgrounds, from those not engaged in sports to competitive athletes. Physical exercise causes bronchoconstriction in the majority (~70%) of the children and adolescents who present asthma; nevertheless, EIA may occur in those who do not present asthma diagnosis (Oxford Health Plans, 2012)

People with asthma can participate in physical activity. For adults or children involved in competitive sport, prescribers need to check which asthma medicines are permitted in the sport. Exercise-induced bronchoconstriction can be managed effectively with relievers and preventers (or both) and should not stop people with asthma participating in physical activity (National Asthma Council Australia, 2015).

In conclusion, the persons with bronchial asthma need counseling and health promotion to be active and encourage them to do exercise with good preparation and special instruction before exercise to avoid exercise-induced asthma.

### 2.2.7.3.14 Obesity-Body Mass Index

Obesity is considered one the most important public health problems of the 21st century. According to the World Health Organization, in 2014, more than 1.9 billion adults were overweight, and 41 million children under the age of 5 were overweight or obese. Genetic and lifestyle factors, mainly over-nutrition (an excess of carbohydrate and fat ingestion and a low ingestion of vegetables, fruits, and whole grains) and low physical activity are important factors that contribute to the escalation of this condition. Obesity is designated by an increased body mass index (BMI) and by systemic low-grade inflammation. In addition, obesity is associated with the development of type 2 diabetes and with cardiovascular and non-alcoholic fatty liver disease risk .Obesity and asthma are two chronic conditions that affect millions of people. Genetic and lifestyle factors such as diet, physical activity, and early exposure to microorganisms are important factors that may contribute to the escalating prevalence of both conditions. The prevalence of asthma is higher in obese individuals. Recently, two major phenotypes of asthma with obesity have been described: one phenotype of early-onset asthma that is aggravated by obesity, and a second phenotype of later-onset asthma that predominantly affects women. Systemic inflammation and mechanical affect, both due to the expansion of the adipose tissue, have been proposed as the main reasons for the association between obesity and asthma. However, the mechanisms involved are not yet fully understood. Moreover, it has also been suggested that insulin resistance syndrome can have a role in the association between these conditions. The intestinal micro biota is an important factor in the development of the immune system, and can be considered a link between obesity and asthma. In the obese state, higher lip polysaccharide serum levels as a consequence of a microbiota dysbiosis have been found. In addition, changes in micro biota composition result in a modification of carbohydrate fermentation capacity, therefore modifying short chain fatty acid levels. The main objective of this review is to summarize the principal findings that link obesity and asthma (Gomez-Llorente, Romero, Chueca, Martinez-Cañavate, & Gomez-Llorente, 2017).

Asthma and obesity are  the objective of this study was to explore the association between body mass index (BMI) and asthma in men and women of diverse ethnic and socioeconomic background living in New York State, USA. In this study, we analyzed cross-sectional data on 5524 subjects aged 18 years and older who were interviewed by telephone in the 1996 and 1997 New York State Behavioral Risk Factor Surveillance System. Asthma (doctor-diagnosed), and weight and height were self- reported. BMI (kg/m2) was used as a measure of adiposity. Weighted logistic regression analysis, with stratification by gender and age, was used to examine the relationship between asthma prevalence and BMI, adjusting for race/ethnicity, education, health insurance, time since last physical examination, physical activity and smoking status. The results showed that the prevalence of asthma was 4.6% (CI: 3.6–5.5%) among men and 8.1% (CI: 7.1–9.1%) among women. In women, the prevalence of asthma was significantly increased in those with a BMI 25kg/m2 or higher (BMI 25–27.5: OR¼1.76, 95% CI: 1.06–2.94; BMI 27.5–29.9: OR¼2.45, 95% CI: 1.41–4.25; BMIX30: OR¼2.67, 95% CI: 1.66–4.29) when compared to the reference category (BMI: 22–24.9kg/m2). In men, the prevalence of asthma was increased in the lowest weight category, BMIo22kg/m2 (OR¼3.05, 95% CI:1.37–6.78) and in the highest category, BMIX30 kg/ m2 (OR2.92, 95% CI: 1.39–6.14).

This U-shaped association persisted when restricting the analysis to men who had never smoked and was more pronounced for those between 18 and 49 years of age. In conclusion, this cross-sectional study showed that men and women differ significantly in the association between BMI and asthma prevalence only with respect to the lowest weight category. While women had a monotonic association, men showed a U-shaped relationship, indicating that both extremes of weight are associated with a higher prevalence of asthma (Luder, Ehrlich, Lou, Melnik, & Kattan, 2004).

In addition, the study by National Asthma Education and Prevention Program (NAEPP) guidelines that specify the goals of asthma control and management strategies, the number of patients with uncontrolled asthma remains high, and factors associated with uncontrolled asthma are unknown. It did study that aim was to examine the relationship between asthma control and socio-demographic characteristics, health-care access and use, asthma education, and medication use among adults with active asthma residing in New England (NGUYEN et al., 2011).

**2.2.7.3.15 Family history**

Studies that concluded if you have a parent with asthma, you are 3-6 times more likely to develop asthma than who doesn't (MINNESOTA DEPARTMENT OF HEALTH ASTHMA PROGRAM, 2007).

Another study that demonstrated that genetics plays an important role in the development of asthma and allergy likely through several genes of moderate effect (i.e., genes associated with relative risks in the range of 1.2–2) Genome-wide linkage studies and case–control studies have identified 18 genomic regions and more than 100 genes associated with allergy and asthma in 11 different populations. In particular, there are consistently replicated regions on the long arms of chromosomes 2, 5, 6, 12 and 13 (Subbarao, Mandhane, and Sears 2009).

Recent genome-wide association study19 identified a new gene, ORMDL3, that exhibited a highly significantly association with asthma ($p<10^{-12}$) (for single nucleotide polymorphism rs8067378, odds ratio 1.84, 95% confidence interval 1.43–2.42) a finding that has now been replicated in several populations. Extensive heterogeneity in the genetic basis of asthma, and in gene-by-environment interactions, is likely. Failure to identify and precisely quantify environmental exposures and their timing may account for some of the difficulty that researchers have had in replicating genetic associations (Subbarao, Mandhane, and Sears 2009).

Another recent study that find the common risk factors for this disease were genetic predisposition, cigarette smoking, family history of asthma and allergic diseases (Alavinezhad & Boskabady, 2017)**.**

### 2.2.7.3.16 Seasonal variation

Seasonal trends in asthma-related hospitalizations are widely recognized; however, little is known about trends in asthma-related intensive care unit (ICU) admissions or intubations. The objective of this study is to examine monthly rates of asthma-related ICU admissions and/or intubations as a percent of total asthma-related admissions and to identify seasonality. This analysis was performed in a database of 285 hospitals representing > 3 million annual inpatient visits. Asthma-related hospital admissions for patients aged 5 and older were identified with a primary diagnosis of asthma during calendar years 2001-2002. The percents of the total admissions per month were compared. Monthly means were calculated and data were presented as moving averages. 76,916 hospital admissions were identified with a primary diagnosis of asthma. Just over 10% (n = 7,803) were admitted to the ICU and/or intubated, with the majority among patients > 35 years of age (> 70%). A peak in asthma-related hospitalizations occurred in the winter months (10.3%) and a nadir in the summer months (5.9%; $p < 0.004$) with similar trends for ICU admissions. Despite this finding, ICU admissions and intubations remained relatively constant as a percent of total asthma-related hospitalizations, ranging from 9.2 to 10.9% and did not dip during the summer months when the overall asthma-related hospitalization rates were lowest. Significant differences in seasonal variation were also noted by age group and by region, but not by gender. These findings suggest a need for year-round vigilance and improved compliance with asthma therapy, especially during the summer when asthma attacks are perceived to be infrequent (Pendergraft, Stanford, Beasley, Stempel, & McLaughlin, 2005).

Weather and climate are known to influence human health. Seasonal changes of temperature promote alterations in respiratory morbidity and in total and cause-specific mortality. Data on the prevalence of asthma and asthma-related symptoms and its disparities between winter and summer in the acid rain-plagued city of Zunyi in southwestern China have not been widely available. In order to describe the epidemiology of asthma and asthma-related symptoms and its prevalence changes between winter and summer, we have completed two cross-sectional surveys of people aged ≥18 years in winter and summer in the inner-city areas of Zunyi city, GuizhouProvince, China. The subjects were selected using a simple random sampling method. Data on asthma and asthma-related symptoms and selected home environmental factors were assessed by questionnaire. The studied Chinese adult population residing in Zunyi recorded a lower prevalence rate of asthma than those of Western countries. There was significant difference in asthma prevalence among adult respondents between winter (1.8%) and summer (0.8%) in inner city Zunyi. Asthma and asthma-related symptoms occurred more frequently in winter than summer, and that difference mainly correlated with environmental risk exposures, including coal combustion, frequency of stove cooking, fan or range hood usage, mattress material, pet possession, must and mould in the bedroom, etc. The prevalence of adult asthma and asthma-related symptoms was higher in winter compared to the summer in Zunyi, China. Our study suggests that asthma may be an important component of the public health burden of indoor air pollution, especially in winter (Jie, Kebing, Yin, & Jie, 2016).

To quantify the number of asthma attacks treated in the emergency room of a public hospital and to study seasonal fluctuations, taking into consideration the local climate, which is characterized by having only two seasons: a rainy/humid season and a dry season. Methods: A retrospective survey was conducted in a community general hospital. 37,642 emergency room consultations related to asthma, bronchitis, pneumonia, upper-airway infection or other respiratory complaints were registered during a two-year period. The data from each patient chart were collected for later analysis. Results: Among the respiratory conditions treated, asthma (24.4%) was the second most common diagnosis. Most of the asthma consultations (56.6%) involved children below the age of fifteen. Regression analysis revealed a seasonal variation in the number of asthma consultations, which was significantly higher in March ($p = 0.0109$), the low points being in August ($p = 0.0485$) and September ($p = 0.0169$). The correlation between climate and asthma was most significant in relation to changes in humidity, although the effect was delayed by one month ($p = 0.0026$) or two months ($p = 0.0002$). Conclusion: Visits to the emergency room for the treatment of asthma attacks were more frequent during the rainy season, increasing at one to two months after the annual increase in humidity and decreasing in the dry season. This positive correlation raises the possibility of a causal relationship with proliferation of house dust mites and molds (VALENÇA, RESTIVO, & NUNES, 2006).

#### 2.2.7.3.17 Asthma with atopic dermatitis, conjunctivitis and rhinitis

The prevalence of asthma in children with allergic rhinitis was 21.6%. Also allergic conjunctivitis is closely associated with asthma and allergic rhinitis(Kim et al., 2013).

Co-morbid allergic rhinitis (AR) and asthma has been studied and the study concluded AR is prevalent in children with asthma. The burden of co-morbid disease in asthmatic children is associated with increased likelihood of asthma-related (Pinto Pereira et al., 2010).

The prevalence of conjunctivitis was slightly more common among women, while rhinitis was more common among men. Patients with both conjunctivitis and rhinitis were more likely to undergo positive skin prick test (SPTs), and they had a higher rate of positive skin prick test. The coexistence of two or more co morbidities increased the risk of having an immunoglobulin E (IgE)-mediated allergy (based on the skin prick test compared to having each of the conditions alone. In conclusion, allergic conjunctivitis can occur either alone or with asthma and/or rhinitis. It is not always accompanied by rhinitis (Michailopoulos et al., 2017)**.**

Atopic dermatitis (AD) is a chronic inflammatory skin disease posing a significant burden on health-care resources and patients' quality of life. It is a complex disease with a wide spectrum of clinical presentations and combinations of symptoms. AD affects up to 20% of children and up to 3% of adults; recent data show that its prevalence is still increasing, especially in low-income countries. First manifestation of AD usually appears early in life and often precedes other allergic diseases such as asthma or allergic rhinitis (Nutrition and Health Department Nestlé Research Center Lausanne Switzerland, 2015).

# Chapter 3

# Methodology

This chapter presents the methodology used in this study. It begins with the study design, then study setting, study population, study sample, sampling, eligibility criteria, study instrument, study period, data collection, data analysis, scientific rigor, administrative and ethical consideration, response rate, pilot study, constrains and strength of study.

### 3.1 Study design

In this Study, the methodology provides quantitative (interviewed–questionnaire) for adult patients with bronchial asthma (cases) and persons free of bronchial asthma (control). Case-control study is an analytical epidemiologic study design in which individuals who have the disease under study, called cases, are compared to individuals free of disease called (controls) regarding past exposures (Kaelin & Bayona, 2004). A case-control study is designed to help determine if an exposure is associated with an outcome (i.e., disease or condition of interest). In theory, the case-control study can be described simply. First, identify the cases (a group known to have the outcome) and the controls (a group known to be free of the outcome). Then, look back in time to learn which subjects in each group had the exposure(s), comparing the frequency of the exposure in the case group to the control group. It is especially useful when there is a need to study multiple exposures, and when a study can be done relatively quickly and inexpensively (Lewallen & Courtright, 1998).

Exposure differences between cases and controls are helpful to find potential risk protective factors. The purpose is to determine if there are one or more factors associated with the disease under study (Kaelin & Bayona, 2004).

Therefore, it is the design that can be used to identify potential risk factors associated with development of bronchial asthma of adult in Gaza Strip.

### 3.2 Study Setting

The study was conducted at the UNRWA health centers (clinic based) that distributed across Gaza Strip.

### 3-3 Population

All adult patients ≥18 years with bronchial asthma that attend UNRWA clinics (Jabalia, Beach, Gaza town, Al Nussirate, Khanionis and Rafah health centers) that selected by the researcher randomly from 22 UNRWA clinics that distributed all over Gaza Strip. It represents about 6340 cases.

### 3-4 Study population

The researcher planed to select (480) precipitants from the selected clinics, 240 cases and then found to them 240 controls.

### 3.5 Study Sample

The researcher reached successfully for 214 cases with specific criteria and then found to them 214 controls with specific criteria, ratio case/control (1:1). Therefore, the total number of sample is 428.

### 3.6 Sampling

#### 3.6.1 Sample size calculation

Illustrated by annex (1)

By EPI info with power 80, Alfa 0.05, exposed control 15 % and Controls / Case ratio = 1 so the total is 416 (208 cases and 208 control).

The researcher increased the actual sample size by 15% to compensate for missing and non-responders that suspected to occur.

Post data collection the final sample size was 428.

These steps illustrated by figure (3.1).

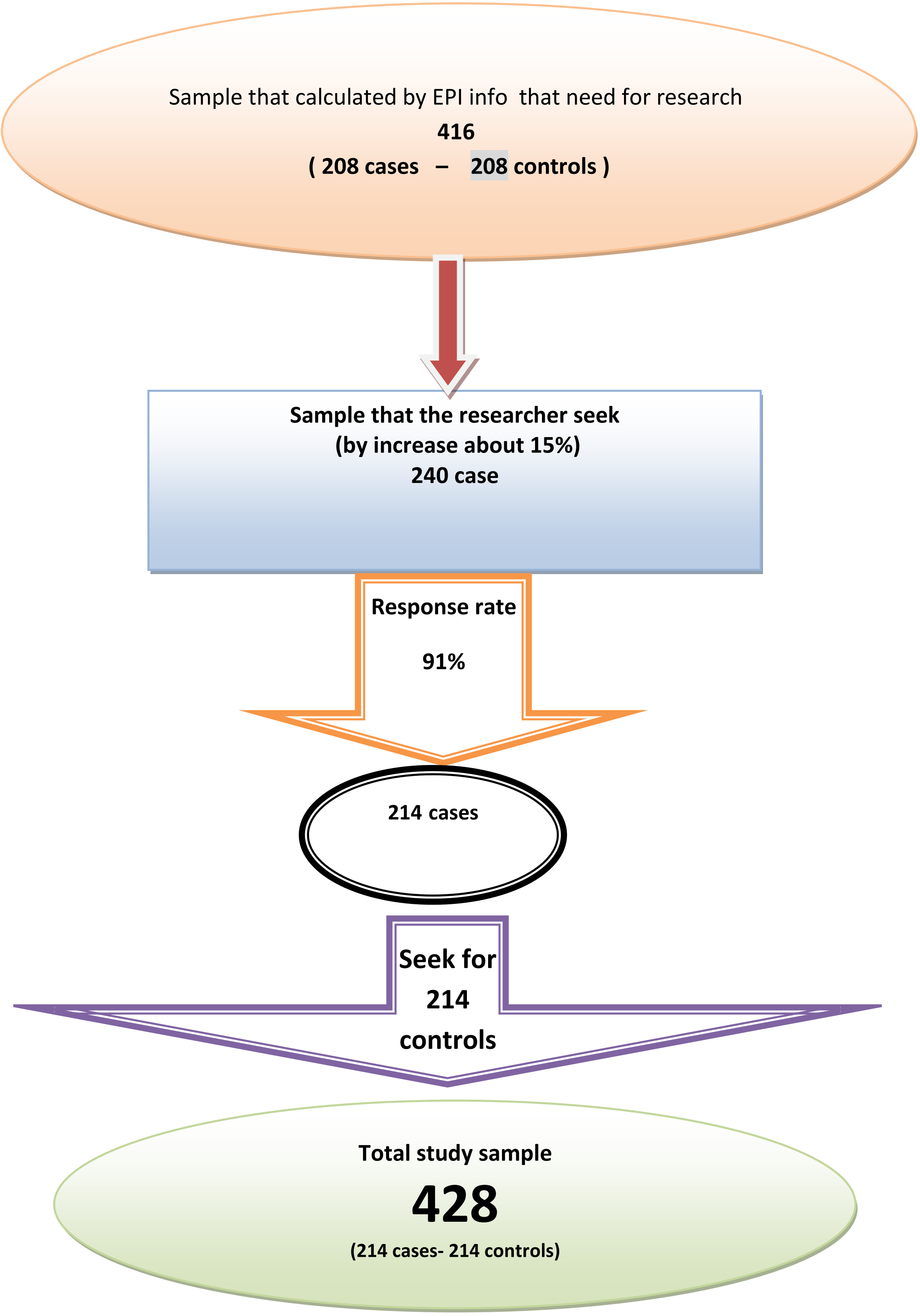


**Figure (3.1) Sample size included in the study by number.**

### 3.6.2 Sample processing:

The researcher used stratified proportional sampling to select cases for the research. First, the researcher selected all Gaza Governorate for study as the following:

North, Gaza, Middle, Khanionis and Rafah governorates. Then, the researcher made a list of clinics that belong to UNRWA for each governorates. Next by simple random sample (drowning lots) selected one clinic from each governorate except Gaza governorate 2 clinics (as its thigh representative governorate).

The clinics that selected randomly determined as the following:

1-Two clinics (Beach health center and Gaza town health center) from Gaza governorate.

2-Jabalia health center from north governorate.

3-Jabanise health center from Khaniouns governorate.

4-Al–Nussirate health center from middle governorate.

5-Rafah health center from Rafah Governorate.

These steps illustrated by figure (3.2)

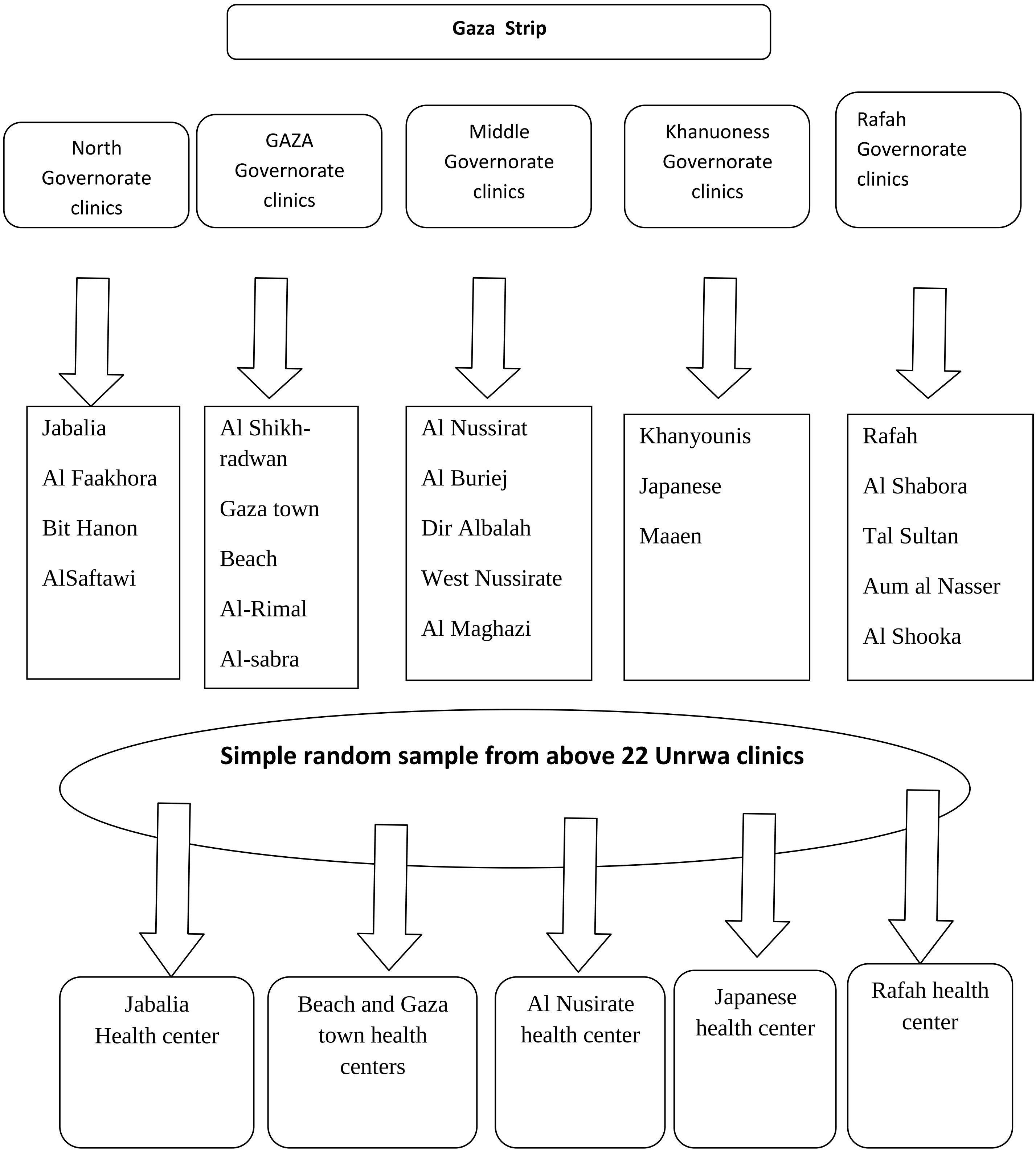


**Figure (3.2) Steps of sample processing**

After that, the researcher with his team prepared the amount of questionnaires for each health centers that belong to each governorate needed. Which represented by percentage according to the distribution of population in Gaza Strip.

At the same time, the researcher with his team prepared the amount of questionnaires for each controls to be completed immediately in the same session with specific criteria with each completed cases as illustrated in table (3.1)

**Table (3.1 ) distribution of study sample**

| Governorates | clinics | repre-sentative percent For population | Cases needed (240) | | Cases success-fully reached | Control For cases | Total study sample |
|---|---|---|---|---|---|---|---|
| **North** | Jabalia | 18.7% | 45 | **Response Rate 91%** | 40 | 40 | 80 |
| **Gaza** | Gaza& Beach | 35% | 84 | | 75 | 75 | 150 |
| **Middle** | Al-Nusirate | 16.4% | 39 | | 35 | 35 | 70 |
| **Khanyonis** | Japanese | 19.2 | 46 | | 41 | 41 | 82 |
| **Rafah** | Rafah | 10.7 | 25 | | 23 | 23 | 46 |
| **Total** | | | 240 | | 214 | 214 | 428 |

Post of the above and after a long period of hard work the study sample was 428 participants (214 cases with 214 controls) distributed as the following:

North governorate (80) ➡ Jaballia health center (40 cases – 40 controls).

Gaza governorate (150) ➡ Gaza town - Beach health centers (75 cases – 75 controls).

Mid-zone governorate (70) ➡ AL-Nusirate health center (35 cases – 35 controls)

Khanyouns governorate (81) ➡ Japanese health center (41 cases – 41 controls).

Rafah governorate (46) ➡ Rafah health center (23 cases - 23 controls).

This illustrated in figure ( 3.3)

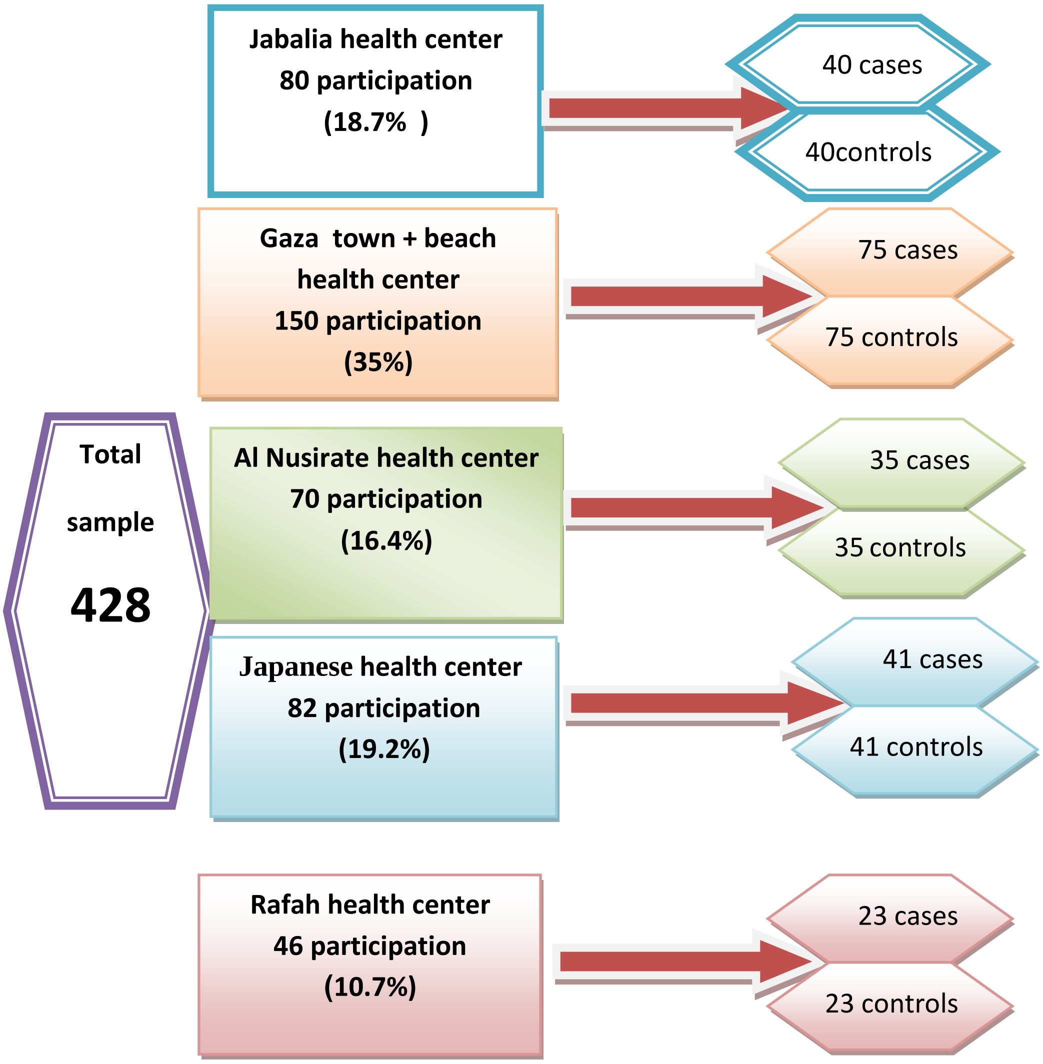


Figure (3.3) Distribution of studied samples in each clinic

In each selected clinic above the researcher selected the target cases by systemic random sample by arranging patients who suffer from bronchial asthma with eligibility criteria in a list from which the researcher selected every 4$^{th}$ patient. He phoned them and gave them information about the study. Post taking a permission from them, he made appointment at health center by date and time for the interview to complete the questionnaire. Next, after the end of each interview of cases the control was selected by simple random sample from the same clinic with special eligibility criteria. At Jabalia health center the researcher finally collected and completed 80 participants 40 cases and 40 controls.

Gaza town and beach health center in gaza governorate the researcher selected 150 participants 75 cases and 75 controls, as well as in Al Nusirate health center in middle zone the researcher selects 70 participants 35 cases and 35 controls. In Japanese health center at khanyonis governorate 82 participants (41cases & 41 controls) and finally from Rafah health center at Rafah governorate in khnyonis and Rafah 46 participants (23 cases & 23 controls).

### 3.6.3 Eligibility criteria

#### 3.6.3.1 Inclusion and exclusion criteria of cases

A case means patients who had bronchial asthma with the following characters:

**Inclusion**

1-Adult patient >=18 years with asthma that registered and follow at the selected UNRWA clinics for receiving any kind of health services.

2- Consecutive patients with physician diagnosed asthma >=18 were eligible for entry into the study.

3--Duration of asthma of one year or more.

4- Attendance of selected UNRWA clinic for 12 months or longer for receiving their anti-asthmatic drugs consecutively

**Exclusion**

1-Patient visiting other facilities governmental (MOH) or NGOs.

2- Self-diagnosed asthma

3- Newly diagnosed asthma less than12 month.

4- Cardiac patients.

#### 3.6.3.2 Inclusion and exclusion criteria of control

Definition:

Persons without bronchial asthma, we matched age, gender and residency (persons from same severed clinic for cases) who attend the same UNRWA clinics for any reasons with the following specific criteria.

**Inclusion**

Age more than 18 years with matching -/+ up to 5 years.

Residency within area that severed from the same clinic for cases.

Persons live in same catchment area for clinics that cases belong to them.

**Exclusion**

Patient with cardiac disease

Individual less than 18 years.

Relative to selected case.

Persons outside catchment area of clinic.

Difference of age more than 5 years.

**3.7 Study instrument**

The researcher prepared the required tool (questionnaire) annex (2) in Arabic and annex (3) in English copy .

The researcher consulted 10 experts at the arbitration stage before the finalization of tool, annex (4) demonstrates names of experts.

This instrument prepared and built by the researcher himself and divided the instrument (quantitative questionnaire) into the following parts:

*The first part about age, gender, governorate, family size, marital state and income.

*Then second part about residence nearby electricity station, electricity generator, farm, corrals, trash containers, professions and factories, main street and ovens.

*Third part about building character such as type of building, type of floor, land cover, painting, home size, number of rooms inside home, site of cooking, fuel used for cooking, cooling and heating for home, animals and plant inside home. Also in this part, the educational level included.

*Fourth part about smoking (cigarette, arrjila and passive smoking) .

*Fifth part is about exposure to many factors that include soil, cement, perfume, exhausts of vehicles, exhausts wood and plastic, chemical cleaning substance, dust, mold, deprivation of petroleum, frying oil, and entrance site of kitchen, seasonal variation, and psychological problems (depression, stress and grief).

*Finally in sixth part the researcher asked about food allergy, drug allergy, exercise, BMI and Family history.

### 3.8 Study Period

The study started in June 2016 and completed by October 2017.The study took 14 months in execution, this study was initially proposed in 2016. The researcher proposal has been submitted to and defended in front of the SPH assigned committee. Post approval, the researcher prepared the required tool (questionnaire). The researcher consulted 10 experts at the arbitration stage before the finalization of tool. The arbitration stage took about one month, post end this stage, the tool (questionnaire) was ready for data collection and the researcher trained two data collectors and carried out the requiring training prior to piloting and filed work. Piloting took two week.

### 3.9 Data Collection

The researcher and two data collectors took more than five month in data collection.
The initial analysis of data was done in March 2016.Then the researcher did analysis for this data. The researcher and two data collectors conducted exit interviews for select participations at selected clinics. This was completed within 5months for all sample size (428 individuals 214 case - 214control).
After checking and reviewing all filled questionnaires on the same day data entered computer by using SPSS (Statistical package for Social Science) software version 20 to be analyzed. After finishing the data entry process, checking codes used to avoid double entries.

### 3.10 Data analysis

The collected data captured and analyzed using SPSS (Statistical Package for Social Sciences) version 20.
1-Descriptive analysis including means, medians, standard deviations, ranges, confidence intervals, frequencies, and percentages.
2-Chi square based on cross tabulation methods to find out the relation between categorical variables.
3-To predict the risk factors associated with asthma, logistic regressions used to assess the odds ratio and confidence interval at 95% and alpha less than 0.05.

### 3.11 Scientific rigor (Quantitative (questionnaire))

#### 3.11.1 Validity

The questionnaire evaluated by tenth experts in public health field to assess and evaluate all components and the context of instrument (questionnaire) ,in order to ensure that it is highly valid and relevance, and their comments will be taken in consideration annex (4). In addition, a pilot study was conducted before the actual data collection to examine responses to the questionnaire and how they understand it. This enhanced the validity of the questionnaire after modifying it to be better understood. Finally, the questionnaire was nicely formatted in order to ensure face validity, this includes appealing layout, and logic sequence of questions and clarify of instructions.

#### 3.11.2 Reliability

The following steps done to assure instruments reliability, training of data collectors of client interviewing steps and the way of asking questions. This assured standardization of questionnaire filling. Then, the data entry in the same day of data collection allowed possible interventions to check the data quality or to refill the questionnaire when required.

### 3.12 Administrative Considerations

In order to launch this study, the proposal was submitted to Al Quds University-school of Public health research committee for discussion and academic approval.
An administrative approval was obtained from the Chief of UNRWA health program.

### 3.13 Ethical consideration:

**Helsinki committee** (Ethical committee in the Gaza Strip): an official letter of approval to conduct the study was obtained from the Helsinki committee Annex (5)

**Informed consent**

*Every participant in this study receive complete explanation about the research, purpose, confidentiality and sponsorship

* Every subject of the study population knew that participation in the research was optional and that they had the right to refuse.

*confidentiality was given and maintained.

### 3.14 Response rate

91% as the researcher seek and looking for collect date from 240 cases and then arrange to them 240 controls with specific criteria to reach 480 participates but the researcher just reached 428 participates (214 cases and 214 controls). High response rate could be attributed to researcher who used appropriate communication and interviewing skills, also interviewed questionnaire high response rate than the self–administered questionnaire as well as the patient at UNRWA clinics respect worker at UNRWA and highly cooperative with them.

### 3.15 Pilot study

A piloting process was conducted before starting data collection. The piloting process aiming to help indentifying problem in research design, test data collections tool for validity, reliability and objective. Additionally, piloting allows the data collectors to gain experience dealing with data collection instrument and the type of people who will ultimately participate in the study.

Piloting was performed on 40 subjects 20 cases and 20 controls from Al-Buriej health center in middle zone. As a result of piloting some modifications of data collection tools were done. Subjects who were selected for piloting were excluded from the study.

### 3.16 Constraints of the study

No basic information about bronchial asthma among adult in Gaza Strip.

The study was self-funded there for the researcher faced financial problem

Limited access to international publication.

### 3.17 Strength of study

First study that done in Gaza Strip about risk factors of bronchial asthma.

Can be considered as base line for future studies.

Pay attention of policy maker about bronchial asthma.

Applicable preventive measures for risk factors that identified in the study.

## Chapter (4)

## Results and findings

This chapter presents the main result and findings of the study in a comparative way between cases and controls. This chapter demonstrates the descriptive variation between cases and controls with illustration the different risk factors contributed to the development of bronchial asthma by using different statistical tools.

It is worth reminding the reader that the study sample was 428 divided into 214 cases (diagnosed with bronchial asthma) and 214 controls (free from bronchial asthma).

The study showed obvious variations in demographic factors, socioeconomic status, environmental, psychological and others factors.

### 4.1 Demographic characteristic

**Table (4.1): Distribution of participants by demographic related data (N=428).**

| | Age group( years) | Control | | Case | | Total | |
|---|---|---|---|---|---|---|---|
| | | No. | % | No. | % | No. | % |
| Age group ( years) | 18 -30 | 61 | 28.5 | 59 | 27.6 | 120 | 28.0 |
| | 31 - 40 | 63 | 29.4 | 57 | 26.6 | 120 | 28.0 |
| | 41 to 50 | 41 | 19.2 | 46 | 21.5 | 87 | 20.3 |
| | More than 50 | 49 | 22.9 | 52 | 24.3 | 101 | 23.6 |
| | Total | **214** | **100.0** | **214** | **100.0** | **428** | **100.0** |
| gender | Male | 140 | 65.4 | 140 | 65.4 | 280 | 65.4 |
| | Female | 74 | 34.6 | 74 | 34.6 | 148 | 34.6 |
| | Total | **214** | **100.0** | **214** | **100.0** | **428** | **100.0** |
| Governorate | North | 40 | 18.7 | 40 | 18.7 | 80 | 18.7 |
| | Gaza | 75 | 35.0 | 75 | 35.0 | 150 | 35.0 |
| | Meddle zone | 35 | 16.4 | 35 | 16.5 | 70 | 16.4 |
| | Khanyounis | 41 | 19.2 | 41 | 19.2 | 82 | 19.2 |
| | Rafah | 23 | 10.7 | 23 | 10.7 | 46 | 10.7 |
| | Total | **214** | **100.0** | **214** | **100.0** | **428** | **100.0** |
| Mari-tal Status | Not Married | 46 | 21.5 | 30 | 14.0 | 76 | 17.8 |
| | Married | 168 | 78.5 | 184 | 86.0 | 352 | 82.2 |

Table (4.1) showed that the majority age of participants were in the age group from 18 to 40 years old (56%) then above 50 years old (23.6%) and finally from 41 to 50 years (20.3 %). The mean age of subject in general was 40 years with (SD 13.7).

Regarding the gender, the researcher noted that male were dominate and represented 65.4% and female 34.6% from the subject.

The precipitants was selected from all over Gaza Strip Governorates as the following, Gaza governorate 150 participates (35%) of subjects, Khanyounis governorate 82 participates that represent (19%) of subjects, North governorate 80 participates that represent (18.7%) of subjects, Med zone governorate 70 participates that represent (16%) of subjects, Rafah governorate 46 participates that represent (10.7%) of subjects .

Regarding of marital status, most subjects in general were married 82.2%, also married cases (86%) were more than controls (78.5%).

### 4.1.1 Family size

**Table (4.2) Relationship between case and control according to family size**

| **Family size (members)** | **Control** | | **Case** | | **Total** | | **$X^2$** | **Sig.** |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **<=5** | 74 | 36.1 | 58 | 27.6 | 132 | 31.8 | 8.777 | 0.012 |
| **6 - 8** | 94 | 45.9 | 89 | 42.4 | 183 | 44.1 | | |
| **Above 8** | 37 | 18.0 | 63 | 30.0 | 100 | 24.1 | | |
| **Total** | 205 | 100.0 | 210 | 100.0 | 415 | 100.0 | | |

Table (4.2) shows the effect of family size, the researcher found the most prominent subjects were from 6 to 8 members (44.1%) then members less than 5 members (31.8%) and the least above 8 members (24%).

The researcher found the difference between case and controls was statistically significant (P-value=0.012) between asthma and large family size more than eight members.

In another word, large family size is positively associated with bronchial asthma, among cases (30%) have large family (>8), while it was only 18 % among controls. This difference reached statistical significant level.

## 4.2 Socioeconomic factors (Education, Occupation and Income)

### 4.2.1 Education

**Table (4.3) relationship between cases and controls according to educational level**

| Education level | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Illiteracy** | 18 | 8.4 | 28 | 13.1 | 46 | 10.7 | 34.610 | 0.001 |
| **Primary** | 13 | 6.1 | 31 | 14.5 | 44 | 10.3 | | |
| **Preparatory** | 23 | 10.7 | 45 | 21.0 | 68 | 15.9 | | |
| **Secondary** | 57 | 26.6 | 58 | 27.1 | 115 | 26.9 | | |
| **Diploma** | 23 | 10.7 | 16 | 7.5 | 39 | 9.1 | | |
| **University** | 80 | 37.4 | 36 | 16.8 | 116 | 27.1 | | |
| **Total** | 214 | 100.0 | 214 | 100.0 | 428 | 100.0 | | |

This table (4.3) shows that the difference between cases and controls according to educational level was statistically significant (P value = 0.001).

The researcher found two categories, first pre-university (illiteracy, primary school, preparatory school and secondary school) and second group that belong to it diploma and university.

The cases among first group (illiteracy, primary school, preparatory school and secondary school) (13.1%, 14.5%, 21% & 27.1%) respectively were more than controls (8.4%, 6.1%, 10.7% & 26.6%) these differences were statically significant (P value = 0.001) that mean whenever the level of education decrease the relation with bronchial asthma increase.

On the otherwise the second group that higher education level (diploma-university) the cases represent (7.5% & 16.8%) respectively less than controls (10.7% & 37%) respectively) these differences are statically significant (P value = 0.001).

In conclusion, from above there is a relation between low level of education and bronchial asthma.

### 4.2.2 Income

**Table (4-4) Relationship between cases and controls according to monthly income**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **500 NIS and less** | 27 | 16.5 | 43 | 28.7 | 70 | 22.3 | 32.033 | 0.001 |
| **501 to less than 1000** | 26 | 15.9 | 47 | 31.3 | 73 | 23.2 | | |
| **1000 to 1500 NIS** | 52 | 31.7 | 42 | 28.0 | 94 | 29.9 | | |
| **Above 1500 NIS** | 59 | 36.0 | 18 | 12.0 | 77 | 24.5 | | |
| **Total** | 164 | 100.0 | 150 | 100.0 | 314 | 100.0 | | |

Table (4.4) shows positive difference between cases and controls and this difference is statistically difference (P value = 0.001) between bronchial asthma and income.

This relation is clear between low incomes below 1000 NIS, which demonstrate high percentage of cases of asthma in two categories in study below 500 NIS. In addition, from 500 to 1000 NIS (28.7%) and (31.3%) respectively in comparison with controls for the same categories (16.5%) and (15.9) respectively.

But when the income increase from 1000 to1500 NIS and above 1500 NIS the percentage increase in control (31.7%) and (36%) respectively in comparison with cases (28%) and(12%) respectively. That means there is relation between low income (below 1000 NIS) and bronchial asthma.

### 4.2.3 Work

**Table (4.5) relationship between cases and controls by work**

| | **Control** | | **Case** | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Not working** | 119 | 55.6 | 153 | 71.5 | 272 | 63.6 | 11.660 | 0.001 |
| **working** | 95 | 44.4 | 61 | 28.5 | 156 | 36.4 | | |
| **Total** | 214 | 100.0 | 214 | 100.0 | 428 | 100.0 | | |

Table (4.5) shows the difference between cases and controls were statistically significant (p value =0.001) according to work.

As the table demonstrated, subject who did not work among cases (71.5%) were more than controls (55.6%) and this relation is statistically significant that means asthma more contributed to subject whom not working.

In another word, not working is positively associated with bronchial asthma, among cases (71.5%) have not work, while it was only (55%) among controls.

## 4.3 Environmental Factors:

## 4.3.1 Indoor pollutions

## 4.3.1.1 Building character

#### 4.3.1.1.1 House roof

**Table (4.6) Relationship between cases and controls according to type of house roof**

| House roof | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Cement** | 183 | 85.5 | 168 | 78.5 | 351 | 82.0 | 3.563 | 0.039 |
| **Asbast** | 31 | 14.5 | 36 | 16.8 | 67 | 15.7 | 0.442 | 0.297 |
| **Asbast+liner** | 3 | 1.4 | 5 | 2.3 | 8 | 1.9 | 0.510 | 0.362 |
| **Zinko** | 18 | 8.4 | 25 | 11.7 | 43 | 10.0 | 1.267 | 0.167 |
| **Zinko +liner** | 4 | 1.9 | 3 | 1.4 | 7 | 1.6 | 0.145 | 0.500 |
| **Carmid** | 1 | 0.5 | 0 | 0.0 | 1 | 0.2 | 1.002 | 0.500 |
| **Carmid +liner** | 1 | 0.5 | 0 | 0.0 | 1 | 0.2 | 1.002 | 0.500 |

This table (4.6) shows high percentage of subject live in house that builds completely from cement (82%). In addition, the highest percent of them among controls (85.5%) in comparison with cases (78.5%).

Nevertheless, roof of houses for cases that consist of asbast (16.8%), asbast with linear (2.3%) and zinko (11.7%) are more than controls (14.5%), (1.4%) and (8.4%) respectively.

In another word there are relations between type of roof of house (asbast, asbast with linear and zinko) and bronchial asthma but not reached statistically significant.

#### 4.3.1.1.2 House land

**Table (4-7) Relationship between cases and controls according to type of house land**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | % | **No.** | % | **No.** | % | | |
| **House Land** | | | | | | | | |
| **flagstone** | 127 | 59.3 | 153 | 71.5 | 280 | 65.4 | 6.982 | 0.005 |
| **kramica** | 89 | 41.6 | 54 | 25.2 | 143 | 33.4 | 12.865 | 0.001 |
| **batoon** | 18 | 8.4 | 21 | 9.8 | 39 | 9.1 | 0.254 | 0.369 |
| **sand** | 3 | 1.4 | 2 | 0.9 | 5 | 1.2 | 0.202 | 0.500 |
| **other** | 2 | 0.9 | 0 | 0.0 | 2 | 0.5 | 2.009 | 0.249 |

Table (4.7) shows statistically significant difference between cases and controls according to use flagstones for house land, cases (71.5%) more than controls (59.3%) that means there is association between bronchial asthma and use of flagstones for house land.

Also, the use of batoon is more in cases (9.8%) than controls (4.8%) and this relation is statistically not significant.

#### 4.3.1.1.3 Land cover

**Table (4.8) Relationship between cases and controls according to land cover that used**

| **Land Cover** | **Control(214)** | | **Case(214)** | | Total | | $X^2$ | **Sig.** |
|---|---|---|---|---|---|---|---|---|
| | **No.** | % | **No.** | % | **No.** | % | | |
| | | | | | | | | |
| **Carpet** | 97 | 45.3 | 58 | 27.1 | 155 | 36.2 | 15.384 | 0.001 |
| **Mokait** | 56 | 26.2 | 31 | 14.5 | 87 | 20.3 | 9.017 | 0.002 |
| **Mat** | 93 | 43.5 | 121 | 56.5 | 214 | 50.0 | 7.327 | 0.004 |
| **Other** | 0 | 0.0 | 3 | 1.4 | 3 | 0.7 | 3.021 | 0.124 |

This table (4.8) shows who had carpet (45.3%) and mokait (26.2%) among controls more than cases (27.1%) and (14.5%) respectively.
Otherwise cases who had MAT more among cases (56.5%) than control (43.5%) and this difference between cases and controls is statistically significant P value (0.004). On another word, uses of MAT for land cover have relation to bronchial asthma.

#### 4.3.1.1.4 House painting

**Table (4-9) Relationship between case and control attributed to houses paint**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **House paint** | | | | | | | | |
| **Oil** | 173 | 34.1 | 29 | 13.6 | 102 | 23.8 | 24.919 | 0.001 |
| **polygad** | 146 | 68.2 | 164 | 76.6 | 310 | 72.4 | 3.791 | 0.033 |
| **gypsum** | 31 | 14.5 | 7 | 3.3 | 38 | 8.9 | 16.635 | 0.001 |
| **others** | 10 | 4.7 | 22 | 10.3 | 32 | 7.5 | 4.864 | 0.021 |

This table (4.9) shows that low percentage for cases in both who painted their house by oil (13.6%) and used gypsum (3.3%) in comparison with controls (34%) and (14.5%) respectively.
In opposite side who used polygad are more among cases (76.9%) in comparison with control (68.2%) and this relation is statistically significant (P = value 0.033). In another word the uses of polygad for painting has relation with bronchial asthma.

#### 4.3.1.1.5 House size

**Table (4-10) Relationship between cases and controls according to house size**

| **House Size (by meter)** | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | % | **No.** | % | **No.** | % | | |
| **100m2 >** | 45 | 21.5 | 73 | 34.6 | 118 | 28.1 | 13.872 | 0.003 |
| **101 to 140** | 62 | 29.7 | 69 | 32.7 | 131 | 31.2 | | |
| **141 to 175m2** | 49 | 23.4 | 37 | 17.5 | 86 | 20.5 | | |
| **Above 175m2** | 53 | 25.4 | 32 | 15.2 | 85 | 20.2 | | |
| **Total** | 209 | 100.0 | 211 | 100.0 | 420 | 100.0 | | |

The table (4.10) shows, that the highest percent of cases among who live inside house less or equal than100 m2 (34.6%) and this percent was higher than controls (21.5%) and this difference was statistically significant (P value = 0.003). In addition, cases who lived in house that size between 100 m2 - 140m2 (32.7%) more than controls (29.7%) and this difference was statistically significant (P value = 0.003).

In otherwise wider house (above140 m2 to 175 m2 and above 175 m2), the study found they more in controls (23.4%) and (25.4%) respectively in comparison with cases (17.5%) and (15.5%) respectively and this difference was statistically significant (P value = 0.003).

In the other word when the houses (less than140 m2) has relation with have bronchial asthma.

**4.3.1.1.6 Number of rooms inside house**

**Table (4-11) Relationship between cases and controls according to number of rooms in house**

| | **Control** | | **Case** | | **Total** | | **$X^2$** | **Sig.** |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Number of rooms** | | | | | | | | |
| **<=2 rooms** | 28 | 13.1 | 52 | 24.3 | 80 | 18.7 | 12.196 | 0.007 |
| **3 rooms** | 82 | 38.5 | 79 | 36.9 | 161 | 37.7 | | |
| **4 rooms** | 61 | 28.6 | 59 | 27.6 | 120 | 28.1 | | |
| **Above 4** | 42 | 19.7 | 24 | 11.2 | 66 | 15.5 | | |
| **Total** | 213 | 100.0 | 214 | 100.0 | 427 | 100.0 | | |

This table (4.11) shows that percentage of cases (24%) that their house consist of 2 room and less are more than controls (13.1%), but controls who their house consist of 3 rooms (38.5%), 4 rooms (28.6%) and above 4 rooms (19.7%) more than cases (36.9%), (27.6%) and (11.2%) respectively. All the previous relations are statistically significant (P value 0.007).

In the other word, bronchial asthma has positive relation with low number of rooms as (two rooms or less).

#### 4.3.1.1.7 Bed room size

**Table (4-12) Relationship between cases and controls according to bedroom size**

| | **Control** | | **Case** | | **Total** | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Rooms Size ( bed room)** | | | | | | | | |
| 2x2 meter | 5 | 2.3 | 16 | 7.5 | 21 | 4.9 | 6.071 | 0.108 |
| 2x3 meter | 36 | 16.9 | 34 | 15.9 | 70 | 16.4 | | |
| 3x4 meter | 130 | 61.0 | 122 | 57.0 | 252 | 59.0 | | |
| 4x4 & more meter | 41 | 19.7 | 42 | 19.5 | 84 | 19.7 | | |
| Total | 213 | 100.0 | 214 | 100.0 | 427 | 100.0 | | |

This table (4.12) showed that cases (7.5%) more than controls (2.3%) who had bedroom measurement (2X2m) or less. That means there is relation between bronchial asthma and small room size (2X2m). This difference did not reach statistical significant level.

#### 4.3.1.1.8 Number of person in bedroom (crowding index)

**Table (4-13) Relationship between cases and controls according to number of persons who sleeping in the room.**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Number of person who sleeping in the room( persons)** | | | | | | | | |
| **1 to 2** | 142 | 66.7 | 135 | 63.1 | 277 | 64.9 | 2.175 | 0.337 |
| **3 to 4** | 59 | 37.7 | 59 | 27.6 | 118 | 27.6 | | |
| **Above 4** | 12 | 5.6 | 20 | 9.3 | 32 | 7.5 | | |
| **Total** | 213 | 100.0 | 214 | 100.0 | 427 | 100.0 | | |

This table (4.13) shows that persons who sleep in bed room (above four members) more among cases (9.3%) than controls (5.6%).

In opposite side percentage for persons who sleep in bedroom (one to two) and (three to four) persons in the same room are lower in cases (63.1%) and (27.6%) in comparison with controls (66.7%) and (37.7%) respectively.

In another word, there is a relation between bronchial asthma and overcrowding (above four persons) in bedroom. This difference did not reach statistical significant level.

#### 4.3.1.1.9 Kitchen character

##### 4.3.1.1.9.1 Cooking place and Kind Fuel of cooking.

**Table (4-14) Distribution of subject according Cooking place and Kind Fuel for cooking**.

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Cooking place** | | | | | | | | |
| **Inside home** | 212 | 99.1 | 211 | 98.6 | 423 | 98.8 | 0.202 | 0.500 |
| **Outside home** | 2 | 0.9 | 3 | 1.4 | 5 | 1.2 | | |
| **Total** | 214 | 100.0 | 214 | 100.0 | 428 | 100.0 | | |
| **Kind Fuel of cooking** | | | | | | | | |
| **Electricity** | 10 | 4.7 | 6 | 2.8 | 16 | 3.7 | 2.002 | 0.367 |
| **Gas** | 201 | 93.9 | 202 | 94.4 | 403 | 94.2 | | |
| **Wood** | 3 | 1.4 | 6 | 2.8 | 9 | 2.1 | | |
| **Total** | 214 | 100.0 | 214 | 100.0 | 428 | 100.0 | | |

This table (4.14) shows the cooking place outside home (1.4%) in comparison with controls (0.9%). In addition, who use wood for cooking more among cases (2.8%) in comparison with controls (1.4%). In another word, there are relation between bronchial asthma and cooking outside house and using wood as fuel for cooking. All the previous difference did not reach statistical significant level .

#### 4.3.1.2 Plant and animal inside the home

**Table (4.15) Relationship between cases and controls according to plant and animal inside the home**

| Plant in home | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **NO** | 164 | 77.0 | 173 | 81.2 | 337 | 79.1 | 1.151 | 0.170 |
| **YES** | 49 | 23.0 | 40 | 18.8 | 89 | 20.9 | | |
| **Total** | 213 | 100.0 | 213 | 100.0 | 426 | 100.0 | | |
| **Animals in home** | | | | | | | | |
| **NO** | 196 | 92.5 | 198 | 93.4 | 394 | 92.9 | 0.143 | 0.425 |
| **YES** | 16 | 7.5 | 14 | 6.6 | 30 | 7.1 | | |
| **Total** | 212 | 100.0 | 212 | 100.0 | 424 | 100.0 | | |

Table (4.15) shows no relation between plant and animal inside the home with bronchial asthma. As table demonstrate low percentage of both plant and animal inside home among cases (18.8%) and (6.6%) respectively in comparison with controls (23%) and (7.5%) respectively.

#### 4.3.1.3 Types of heating

**Table (4-16) Relation between cases and controls by ways of heating**

| Heating Types | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No. | % | No | % | | |
| **Electricity** | 103 | 50.5 | 52 | 26.3 | 155 | 38.6 | 29.35 | 0.001 |
| **Gas** | 25 | 12.3 | 21 | 10.6 | 46 | 11.4 | | |
| **Wood** | 33 | 16.2 | 49 | 24.7 | 82 | 20.4 | | |
| **Coal** | 5 | 2.5 | 8 | 4.0 | 13 | 3.2 | | |
| **Nothing** | 38 | 18.6 | 68 | 34.3 | 106 | 26.4 | | |
| **Total** | 204 | 100.0 | 198 | 100.0 | 402 | 100.0 | | |

As table (4.16) shows, the most modern way for heating either by electricity (50.5%) or Gas (12.3%) were high in controls in comparison with cases (26.3%) and (10.6%) respectively. Otherwise, traditional way for heating either by wood and coal are more common in cases about (24.7%) and (4% ) in opposite for controls (16.2%) and (2.5%) respectively. In addition, subjects who not use anything for heating are more prominent in cases about (34%) in comparison with controls (18.6%).

In conclusion heating by traditional ways either through using wood or coal combustion or persons who not use things for heating have relation to bronchial asthma and these relations are statistically significant P value = 0.001**.**

#### 4.3.1.4 Factors related to Air-cooling

**Table (4-17) Relationship between cases and controls according to ways of air cooing.**

| **Types of air-cooling** | **Control** | | **Case** | | **Total** | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Air-Condition** | 22 | 10.3 | 4 | 1.9 | 26 | 6.2 | 24.601 | 0.001 |
| **Fan** | 115 | 54.0 | 90 | 42.5 | 205 | 48.2 | | |
| **Nothing** | 76 | 35.7 | 118 | 55.7 | 194 | 45.6 | | |
| **Total** | 213 | 100.0 | 212 | 100.0 | 425 | 100.0 | | |

As table (4.17) shows the percentage for subject who used fan (54%) and air- conditioning (10.3%) were more among controls in comparison with case (42.5%) and (1.9%) respectively and this difference is statistically significant (P-value less than 0.001). On the other wise subject who not use things for conditioning are more common in cases (55.7%) in comparison with controls (35.7%) and also this difference is statistically significant (P-value less than 0.001). In another word, bronchial asthma has relation with persons not use thing for cooling air in comparison with other that use air condition.

## 4.3.1.5 Smoking

### 4.3.1.5.1 Cigarette smoking

**Table (4. 18) Relationship between cases and controls according to cigarette smoking**

| **Cigarette Smoking** | **Controls** | | **Cases** | | **Total** | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | % | **No.** | % | **No.** | % | | |
| NO | 154 | 72.0 | 142 | 66.4 | 296 | 69.2 | 1.577 | 0.125 |
| YES | 60 | 28.0 | 72 | 33.6 | 132 | 30.8 | | |
| **Total** | **214** | **100.0** | **214** | **100.0** | **428** | **100.0** | | |
| **Number of Cigarette per day** | | | | | | | | |
| Less than 10 | 36 | 60.0 | 31 | 43.1 | 67 | 50.8 | 3.921 | 0.141 |
| From 11 to 20 | 17 | 28.3 | 27 | 37.5 | 44 | 33.3 | | |
| Above 20 | 7 | 11.4 | 14 | 19.4 | 21 | 15.9 | | |
| **Total** | **60** | **100.0** | **72** | **100.0** | **132** | **100.0** | | |
| **Years of smoking** | | | | | | | | |
| Less than 10 | 20 | 33.3 | 24 | 33.3 | 44 | 33.3 | 3.075 | 0.215 |
| From10 to 20 | 15 | 25.0 | 27 | 37.5 | 42 | 31.8 | | |
| Above 20 years | 25 | 41.7 | 21 | 29.2 | 46 | 34.8 | | |
| **Total** | **60** | **100.0** | **72** | **100.0** | **132** | **100.0** | | |

Regarding cigarette smoking table (4.18) the researcher found the highest percentage of participant was smoking among cases (33.6%) in comparison with controls (28%). This difference did not reach statistical significant level.

Regarding number of cigarette smoking per day the researcher found less than 10 cigarette smoking per day more in controls (60%) than cases (34%) but if number of cigarettes increase from10-20 and above 20 more among cases (37.5%) and (19.4%) in comparison with controls (28.3%) and (11.4%) respectively. That means there is relation between bronchial asthma and amount of increasing number of cigarette smoking per day. These differences did not reach statistical significant level.

**Relationship between cases and controls according to cigarette smoking by gender.**

**Table (4.19) Relationship between case and control according to male cigarette smoking**

| Male Smoking cigarette | control | | cases | | sig | OR | CI |
|---|---|---|---|---|---|---|---|
| | No | % | No | % | | | |
| NO | 82 | 58.6 | 69 | 49.3 | 0.120 | ( 1.455 | (2.332 - 0.907) |
| YES | 58 | 41.4 | 71 | 50.7 | | | |
| Total | 140 | 100.0 | 140 | 100.0 | | | |

This table (4.19) show that male who use cigarette smoking between cases 70 persons that represent 50.7% more than controls 58 persons that represent 41.4% . That means there is relation between bronchial asthma and cigarette smoking. This difference did not reach statistical significant level.

**Table(4.20) relationship between case and control according to female cigarette smoking.**

| Smoking Cigarette | Case | | controls | | Total | | $X^2$ | Sig. | OR | CI |
|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | | | |
| **NO** | 73 | 98.6 | 72 | 97.3 | 145 | 98.0 | 0.34 | 0.567 | 0.49 3 | 0.044-5.559 |
| **YES** | 1 | 1.4 | 2 | 2.7 | 3 | 2.0 | | | | |
| **Total** | 74 | 50.0 | 74 | 50.0 | 148 | 100. | | | | |

This table (4.20) shows that just 3 female who smoking cigarettes (one between cases and two between controls) between all women in study as female in this study represent about (34.6%) from all subjects. It is explained by that most smoker women deny smoking habits. That leading to underestimation of these risk factors as table shows that women who smoked just one participate represent (1.4%) among case and control two participates represent (2.7%).

#### 4.3.1.5.2 Arrjila smoking

**Table (4.21) relationship between cases and controls according to Arial smoking**

| | Control | | Case | | Total | | $X^2$ | Sig. | OR | CI |
|---|---|---|---|---|---|---|---|---|---|---|
| | **No.** | % | **No.** | % | **No.** | % | | | | |
| **Arrjila** | | | | | | | | | | |
| NO | 161 | 75.2 | 136 | 63.6 | 297 | 69.4 | 6.87 5 | **0.009** | **1.742** | **(1.148 - 2.643)** |
| YES | 53 | 24.8 | 78 | 36.4 | 131 | 30.6 | | | | |
| **Total** | **214** | **100.** | **214** | **100.0** | **428** | **100.0** | | | | |
| **Times Arrjila Smoking per week** | | | | | | | | | | |
| Once | 44 | 83.0 | 74 | 94.9 | 118 | 90.1 | 4.96 | 0.028 | | |
| =>2 | 9 | 17.0 | 4 | 5.1 | 13 | 9.1 | | | | |
| **Total** | **53** | **100.0** | **78** | **100.0** | **131** | **100.0** | | | | |

Regarding arrjila table (4.21) shows most of subject among cases who use it about (36.4%) in comparison with controls (24.8%). As shown in the table the relation was statistically significant between who use arrjila and bronchial asthma (P-value = 0.001) so there is association between arrjila and bronchial asthma. Also the table shows arrjila smoking increase the risk of bronchial asthma by (70%) as OR (CI) = 1.742 (1.148-2.643).

Also, table (4.21) shows one time smoking arrjila per week more in cases (94%) than controls (83%) So the researcher found the most important things was just smoking arrjila once weekly is enough to explore the effect of arrjila smoking.

#### 4.3.1.5.3 Passive smoking

**Table (4.22) Relationship between cases and controls according to passive Smoking (smoker in home)**

| | Control | | Case | | Total | | $X^2$ | Sig | OR | CI |
|---|---|---|---|---|---|---|---|---|---|---|
| | **No** | % | **No.** | % | **No.** | % | | | | |
| **Smokers in home (passive Smoker)** | | | | | | | | | | |
| NO | 145 | 67.8 | 128 | 59.8 | 273 | 63.8 | **2.923** | **0.05** | **1.4** | **(0.950- 2.098)** |
| YES | 69 | 32.2 | 86 | 40.2 | 155 | 36.2 | | | | |
| **Total** | **214** | **100.0** | **214** | **100.0** | **428** | **100.0** | | | | |
| **Number of smokers in home** | | | | | | | | | | |
| One | 48 | 69.6 | 64 | 74.4 | 112 | 72.3 | 0.467 | 0.79 | | |
| Two | 12 | 17.4 | 13 | 15.1 | 25 | 16.1 | | | | |
| Three& more | 9 | 13.0 | 9 | 10.5 | 18 | 11.6 | | | | |
| **Total** | **69** | **100.0** | **86** | **100.0** | **155** | **100.0** | | | | |

Table (4.22) shows higher percentage of passive smoking among cases (40.2%) in than controls (32.2%). In another word, passive smoking has a positive relation with bronchial asthma but not reach statistically significant level (P value = 0.050).

Regarding number of smoker at home who leads to the effect of passive smoking, the researcher found that most of cases exposed to one smoker at home (74.4%) in comparison with controls (69.6%). This relation did not reach statistically significant level. The researcher found the most important things was just one smoking person inside the home is enough to explore the effect of passive smoking to members at that home.

#### 4.3.1.5.4 Exposure to tobacco smoking (passive smoking, Arrjila smoking and Cigarette smoking)

**Table (4.23) Relationship between cases and controls according to exposure to tobacco smoking in general (passive smoking, Arrjila smoking and Cigarette smoking)**

| **Tobacco smoking (cigarette, arrjila and passive)** | Control | | Case | | Total | | **$X^2$** | **Sig.** | **OR** | **CI** |
|---|---|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | | | | |
| **NO** | 92 | 43.0 | 63 | 29.4 | 155 | 36.2 | 8.506 | 0.002 | 1.807 | (1.212-2.695) |
| **YES** | 122 | 57.0 | 151 | 70.6 | 273 | 36.8 | | | | |
| **Total** | **214** | **100.0** | **214** | **100.0** | **428** | **100.0** | | | | |

Table (4.23) shows firstly that highest percentage of subject exposed to tobacco smoke (Cigarette, Arrjila and passive smoking) around 63.8%. Highest percentage of exposure to different types of smoking inside the home among cases were (70.6%) in comparison with (57%) among controls. The researcher found relationship between bronchial asthma and exposure to all type of tobacco smoking and this relation is statistically significant (P-value =0.002). Also, the researcher found that exposure to tobacco smoking increase the risk for bronchial asthma about (80%) as OR (CI) =1.807 (1.212-2.695).

In the other word, exposure to tobacco smoking has association to bronchial asthma and considered as a risk factor of bronchial asthma.

### 4.3.2 Outdoor pollution

#### 4.3.2.1 Residence nearby electricity station, generators and ovens.

**Table (4.24) Relationship between cases and controls according to residence nearby electricity station, generators and ovens.**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Electricity Station** | 25 | 11.7 | 18 | 8.4 | 43 | 10.0 | 1.267 | 0.167 |
| **Big Generators** | 67 | 31.3 | 49 | 22.9 | 116 | 27.1 | 3.832 | 0.032 |
| **Small Generator** | 85 | 39.7 | 63 | 29.4 | 148 | 34.6 | 4.999 | 0.016 |
| **Oven** | 39 | 18.2 | 38 | 17.8 | 77 | 18.0 | 0.016 | 0.500 |
| **Total** | 138 | 64.5 | 113 | 52.8 | 251 | 68.6 | 6.021 | 0.009 |

This table (4.24) showed difference between cases and controls and this difference is statistically significant just among subject who live nearby big generators and small generators as higher percentage of them among controls in both subject who live nearby big generators and small generators (31.3%) and (39.7%) respectively in comparison with cases (22.9%) and (29.4%) respectively and this relation were statistically significant as (P value = 0.032 and 0.016 respectively). In conclusion no relation between living nearby big and small generators and bronchial asthma.

Also annex (6), the difference between cases and controls according to distances by meter between residency and electricity station, electricity generators (BIG or small) and ovens were not statistically significant.

#### 4.3.2.2 Residence nearby main Streets

**Table (4.25) Relationship between cases and controls according to residence nearby main Streets**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Main Streets** | 141 | 65.9 | 126 | 58.9 | 267 | 62.4 | 2.240 | 0.081 |

This table (4.25) shows no relationship between living nearby the Main Street and bronchial asthma. As cases that living nearby main street contribute 126 subject represent 58.9% less than controls 141 that represent 65.9% and this difference not statistically significant (P value = 0.081).

Also, annex (7) show the difference between cases and controls according to distance between residence and main street by meter were not statistically significant.

#### 4.3.2.3 Residence nearby trash container

**Table (4.26) Relationship between cases and controls according to residence nearby trash container**

| **Trash containers** | **Control** | | **Case** | | **Total** | | **$X^2$** | **Sig.** |
|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| | 50 | 23.4 | 81 | 37.9 | 131 | 30.6 | 10.572 | 0.001 |

This table (4.26) shows that living nearby trash containers were more among cases (37.9%) than controls (23.4%) and this difference were statistically significant (P value < 0.001) so there was relation between bronchial asthma and residence nearby trash containers.

Although annex (8) demonstrate that the percentage of cases that living nearby trash container within distance less than 200 m and between 200-500 m are more among cases (53.1%) and (23.5%) respectively in comparison with controls (44%) and (18%) also this difference is statistically not significant.

#### 4.3.2.4 Residence nearby farms

**Table (4-27) relation between cases and controls according to residence nearby farms**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Citrus** | 53 | 24.8 | 46 | 21.5 | 99 | 23.1 | 0.644 | 0.246 |
| **Olive** | 68 | 31.8 | 57 | 26.6 | 125 | 29.2 | 1.367 | 0.144 |
| **Seasonal crops** | 39 | 18.2 | 34 | 15.9 | 73 | 17.1 | 0.413 | 0.304 |
| **Grape** | 24 | 11.2 | 24 | 11.2 | 48 | 11.2 | 0.000 | 0.561 |

This table (4.27) showed that no relationship between living nearby the farms and bronchial asthma. As the controls between subjects are more than cases according to residency near farms either citrus, olive and seasonal crops also this difference was not statistically difference.

#### 4.3.2.5 Residence near corrals

**Table (4.28) Relationship between cases and controls according to residence near corrals**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Chicken** | 67 | 31.3 | 78 | 36.4 | 145 | 33.9 | 1.262 | 0.154 |
| **Dove** | 56 | 26.2 | 68 | 31.8 | 124 | 29.0 | 1.635 | 0.121 |
| **Ducks** | 28 | 13.1 | 40 | 18.7 | 68 | 15.9 | 2.518 | 0.073 |
| **Sheep** | 34 | 15.9 | 35 | 16.4 | 69 | 16.1 | 0.017 | 0.500 |
| **Cows** | 17 | 7.9 | 17 | 7.9 | 34 | 7.9 | 0.000 | 0.571 |
| **Other Animals** | 18 | 8.4 | 15 | 7.0 | 33 | 7.7 | 0.296 | 0.359 |

This table (4.28) showed that there was difference between cases and controls according to residency near corrals as cases more in all nearby corals of chicken, dove, ducks, sheep and cows as following (36.4%), (31.8%), (18.7%), (16.4%) and (34%) in comparison with controls (31.3%), (26.2%), (13.1%), (15.9%) and (7.9%) respectively. In conclusion, there are relations between residency nearby all the above types of corrals and bronchial asthma but not reach significant statistically. Annex (10) show the cases that living less than 200m more nearby corrals of chicken (52.6%), dove (64.7%), ducks (70%), sheep (65.7%) and cow (41%) than controls (40.3%), (41.1%), (39.3%), (44.2) and (50%) respectively but this relation not statistically significant.

#### 4.3.2.6 Residence near professions and factories

**Table (4.29) Residence near professions and factories**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No.** | % | **No.** | % | **No.** | % | | |
| **Concrete** | 49 | 22.9 | 27 | 12.6 | 76 | 17.8 | 7.743 | 0.004 |
| **Crusher** | 17 | 7.9 | 22 | 10.3 | 39 | 9.1 | 0.705 | 0.281 |
| **Carpentry** | 59 | 27.6 | 40 | 18.7 | 99 | 23.1 | 4.744 | 0.019 |
| **Metal works** | 56 | 26.2 | 50 | 23.4 | 106 | 24.8 | 0.451 | 0.288 |
| **Mechanical for cars** | 48 | 22.4 | 39 | 18.2 | 87 | 20.3 | 1.169 | 0.168 |
| **Manufacturing Perfume** | 17 | 7.9 | 13 | 16.1 | 30 | 7.0 | 0.574 | 0.285 |

This table (4.29) show that no relation between bronchial asthma and residence nearby professions and factories. The relation just nearby crusher as cases (10.3%) in comparison with controls (7.9%) also this relation is not statistically significant.

### 4.3.3 Other environmental factors

#### 4.3.3.1Soil

**Table (4.30) Relationship between cases and controls according to exposed and degree of effect due to soil**

| | **Not exposed** | | **Exposed with no effect** | | **Expose with mild effect** | | **Exposed with Moderate effect** | | **Exposed with severe effect** | | $X^2$ | **Sig.** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No.** | **%** | **No.** | **%** | **No.** | **%** | **NO** | **%** | | |
| **Soil** | | | | | | | | | | | | |
| **Control** | 76 | 35.5 | 105 | 49.1 | 13 | 6.1 | 10 | 4.7 | 10 | 4.7 | 116.188 | 0.001 |
| **Case** | 65 | 30.4 | 21 | 9.8 | 27 | 12.6 | 43 | 20.1 | 58 | 27.1 | | |
| **Total** | 141 | 32.9 | 126 | 29.4 | 40 | 9.3 | 53 | 12.4 | 68 | 15.9 | | |

As table (4.30) showed that the percentage of subject were more affected by exposure to soil among cases were more (59.3%) and divided in to mild effect (12.6%), moderate effect (20.1%), severe effect (27.1%) than controls who exposed and affected (15.5%) that divided in to (6.1%), (4.7%) and (4.7%) respectively and these difference were statistically significant as (P value = 0.001) but the study find the percentage of participations that exposed to soil with no effect were among controls (49.1%) more than cases (9.8%). In conclusion, there is effect between exposure to soil and bronchial asthma.

**Relationship between cases and controls according to kind of effect of exposure to soil. Annex (12)**

The effect of exposure to soil between case and controls, that the shortness of breath (71.1%) more among cases than controls (29.7%) and this difference was statistically significant (P value less than 0.001) but conjunctivitis more among control 27.1% in comparison with cases 10.9% and this difference was statistically significant (P value 0.018). That conclude the more effect type of exposure due to soil is shortness of breath.

**4.3.3.2 Cement**

**Table (4-31) Relationship between cases and controls according to exposure and degree of effect due to cement**

| | **Not exposed** | | **Exposed with no effect** | | **Exposed with mild effect** | | **Exposed with Moderate effect** | | **Exposed with severe effect** | | $X^2$ | **Sig** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **No.** | **%** | **No** | **%** | **No.** | **%** | **No.** | **%** | **No.** | **%** | | |
| **Cement** | | | | | | | | | | | | |
| **Control** | 101 | 47.2 | 79 | 36.9 | 14 | 6.5 | 11 | 5.1 | 9 | 4.2 | 101.5 | 0.001 |
| **Case** | 83 | 38.8 | 13 | 6.1 | 23 | 10.7 | 35 | 16.4 | 60 | 28.0 | | |
| **Total** | 184 | 43.0 | 92 | 21.5 | 37 | 8.6 | 46 | 10.7 | 69 | 16.1 | | |

This table (4.31) shows the degree of effect and exposure of cement on cases and controls. The highest percent of exposed and affected by cement between cases (55.1%) arrange as that arrange as the following severe (28%) then moderate (16.4%) and last mild

(10.7%) in comparison with controls that exposed and affect (15.8%) and arrange as following (4.2%), (5.1%) and (6.5%) respectively, so all percentage of cases that exposed and affected by cement (55.1%) in comparison with controls (15.8%) that meaning exposure to cement has relation to bronchial asthma and this relation is statistically significant (P value less than 0.001).

**Relationship between cases and controls according to kind of effect of exposure to cement. Annex (13)**

The effect of cement on participants that contribute106 (69.3%) complain of shortness of breath, 95 (89.6%) of them belong to cases more than controls that contributed11 (10.4%) participations. Also, the difference between case and controls is statistically significant (P value less than 0.001). Then allergic conjunctivitis 24 of participants that contribute (15.7%), 13 (54.1%) of them belong to cases more than controls that contributed 11(45.8%) and the difference between case and controls is statistically significant (P value = 0.006). In conclusion the difference between four types of effect that study in our research (skin dermatitis, shortness of breath, allergic rhinitis and allergic conjunctivitis), all of them are more in cases but just of shortness of breath and allergic conjunctivitis are statistically significant.

### 4.3.3.3 Perfume

**Table (4.32) Relationship between cases and controls according to exposed and degree of effect due to perfume**

| | Not exposed | | Exposed with no effect | | Exposed with mild effect | | Exposed with moderate effect | | Exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | No | % | No | % | | |
| **Perfume** | | | | | | | | | | | | |
| **Control** | 58 | 27.2 | 119 | 55.9 | 15 | 7.0 | 16 | 7.5 | 5 | 2.3 | 151.560 | 0.001 |
| **Case** | 33 | 15.4 | 25 | 11.7 | 33 | 15.4 | 51 | 23.8 | 72 | 33.6 | | |
| **Total** | 91 | 21.3 | 144 | 33.7 | 48 | 11.2 | 67 | 15.7 | 77 | 18.0 | | |

This table (4.32) shows the effect of perfume on cases and controls, the researcher notice that (72.8%) of cases exposed and effected to perfume in comparison with controls (16.8%) and this difference was statistically significant P value less than 0.001 .

On other wise, the highest percent of sever effect due to exposed to perfume (33.6%) then moderate (23.8%) and last mild (15.4%) among cases in comparison with controls (2.3%), (7.5%) and (7%) respectively. On another word, all percentage of cases that affected by asthma (72.8%) in comparison with controls (16.8%) that meaning the perfume has positive relation to bronchial asthma.

**Relationship between cases and controls according to kind of effect of exposure to perfume. Annex (14)**

The effect of perfume on participants, 124 of participations that contribute (64.6%) complain of shortness of breath, 116 (93.5%) of them belong to cases more than controls that contributed eight (6.4%) participations. Also, the difference between case and controls is statistically significant (P-value less than 0.001). Then 89 of participations that contribute (46.4%) complain of allergic rhinitis, 66 (54.1%) of them belong to cases more than controls that contributed 23 (25.8%) and the difference between case and controls according to allergic rhinitis is statistically significant (P value = 0.006). In conclusion the difference between four types of effect that study in our research (skin dermatitis, shortness of breath, allergic rhinitis and allergic conjunctivitis), all of them are more in cases but just of shortness of breath and allergic rhinitis are statistically significant.

### 4.3.3.4 Exhausts fuels of vehicles, exhausts of wood, plastic and exposed to derivatives of petroleum

**Table (4-33a) Relationship between cases and controls according to exposed and degree of effect to exhausts of vehicle fuel, wood product, plastic product and derivatives of petroleum.**

| | Not exposed | | Exposed with no effect | | Exposed with mild effect | | Exposed - moderate effect | | Exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % | No | % | | |
| **Exhausts' fuels from vehicles** | | | | | | | | | | | | |
| **Control** | 47 | 22.0 | 106 | 49.5 | 30 | 14.0 | 17 | 7.9 | 14 | 6.5 | 111.7 | 0.001 |
| **Case** | 26 | 12.1 | 27 | 12.6 | 34 | 15.9 | 63 | 29.4 | 64 | 29.9 | | |
| **Total** | 73 | 17.1 | 133 | 31.1 | 64 | 15.0 | 80 | 18.7 | 78 | 18.2 | | |

**Table (4-32 b) Relationship between cases and controls according to exposed and degree of effect to exhausts of vehicle fuel, wood product, plastic product and derivatives of petroleum.**

| | Not exposed | | exposed with no effect | | exposed with mild effect | | exposed with moderate effect | | exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % | No | % | | |
| **Exhausts burn of wood** | | | | | | | | | | | | |
| **Control** | 71 | 33.3 | 97 | 45.5 | 21 | 9.9 | 14 | 6.6 | 10 | 4.7 | 129.31 | 0.001 |
| **Case** | 55 | 25.7 | 12 | 5.6 | 35 | 16.4 | 53 | 24.8 | 59 | 27.6 | | |
| **Total** | 126 | 29.5 | 109 | 25.5 | 56 | 13.1 | 67 | 15.7 | 69 | 16.2 | | |
| **Exhausts burn of plastic** | | | | | | | | | | | | |
| **Control** | 86 | 40.4 | 81 | 38.0 | 23 | 10.8 | 12 | 5.6 | 11 | 5.2 | 118.559 | 0.001 |
| **Case** | 65 | 30.4 | 11 | 5.1 | 22 | 10.3 | 54 | 25.2 | 62 | 29.0 | | |
| **Total** | 151 | 35.4 | 92 | 21.5 | 45 | 10.5 | 66 | 15.5 | 73 | 17.1 | | |
| **Derivatives of petroleum** | | | | | | | | | | | | |
| **Control** | 85 | 40.1 | 96 | 45.3 | 11 | 5.2 | 12 | 5.7 | 8 | 3.8 | 81.245 | 0.001 |
| **Case** | 74 | 34.9 | 30 | 14.2 | 22 | 10.4 | 37 | 17.5 | 49 | 23.1 | | |
| **Total** | 159 | 37.5 | 126 | 29.7 | 33 | 7.8 | 49 | 11.6 | 57 | 13.4 | | |

The table (4.33) shows firstly regarding to exposed and effect of exhausts' fuels from vehicles the cases were more (75.2%) than controls (28.4%) and the cases that exposed and effect with highest percentage among severe effects (29.9%) and moderate effect (29.4%) last mild effect (15.9%) in comparison with controls (6.5 %), (7.9%) and (14%) respectively and this relation were statically significant P value less than 0.001.

Regarding to Exhausts 'burn of wood that exposed and affect the percent of cases were more (68.8%) than controls (21.2%) and cases that exposed with sever effect were highest percentage (27.6%), then moderate (24.8%) last mild (16.4%) in comparison with controls (4.7 %), (6.6%) and (9.9%) respectively and this relation were statically significant p value less than 0.001.

Regarding to Exhausts 'burn of plastic the percent of cases that exposed and affected were more (64.5%) than controls (21.6%) and cases that exposed with sever effect were highest percentage among (29%) then moderate effect (25.2%) last mild effect (10.3%) in comparison with controls (5.2 % ), (5.6%) and (10.8%) respectively and this relation were statically significant p value less than 0.001.

Regarding to Exposed to derivatives of petroleum the percent of cases were more (51%) than controls (14.7%) and cases that exposed and affected with sever effect were highest percentage (23.1%), then moderate effect(17.5%) last mild effect (10.4%) in comparison with controls (3.8 %), (5.7%) and (5.2%) respectively and this relation were statically significant p value less than 0.001. The conclusion from table (4.32) there are relations between exposed to exhaust's of fuels from vehicle, wood, plastic product and derivation of petroleum and all this relation are statistically significant.

**Relationship between cases and controls according to kind of effect of exposure to exhausts of vehicle fuel, wood product, plastic product and derivatives of petroleum. Annex (15)**

Exhausts fuels from vehicle, shortness of breath that contributed 161 (71.9%) of participations distributed as 135 (83.8%) cases which was more than controls that contributed 26 (16.1%) and this difference was statistically significant (p value = 0.001).

Then, allergic rhinitis due exposed to exhausts fuels from cars that contributed 78 (34.8%) participation distributed as cases 49 (62.8%) of in comparison with controls 29 (37.2%). Also, this difference was statistically significant (P value 0.016). Next allergic conjunctivitis contributed 22 (9.8%) of participant that exposed to exhausts fuels and complained from conjunctivitis divided into 12 (54.5%) in cases and 10 (45.5%) in controls .also the difference between cases and controls it is statistically significant (P value 0.047).

Finally skin dermatitis that contributes 2.7 % from all participants that exposed exhausts fuels from cars the difference between cases and controls not statistically significant.

In conclusion, exposed to exhausts fuels from cars have affects cases more than controls and lead to shortness of breath, allergic rhinitis and conjunctivitis.

Exhausts burn of wood, most kind of effect due to exhausts burn of wood were shortness of breath that contributed 147 (75.4%) of participants distributed as 122 (82.9%)among cases which was more than controls that contributed 25(17%) participation and this difference was statistically significant p value less than 0.001 .

Then, allergic conjunctivitis contributed 30 (15.4%) of participants that exposed to exhausts burn of wood and complained from conjunctivitis divided into 17 cases (56.6%) and 13 (43.3%) controls the difference is statistically significant (P value = 0.012).

In addition, allergic rhinitis and allergic dermatitis more common in cases than controls but it is statistically not significant.

In conclusion, exposed to exhausts burn from wood have affects cases more than controls and lead to shortness of breath and allergic conjunctivitis.

Exhausts burn of plastic shows the most kind of effect due to exhausts were shortness of breath that contributed 149 (81.9%) of participants distributed as 121 (81.2%) cases which was more than controls that contributed 28 (18.7%) and this difference was statistically significant (P value 0.001) .

Then, allergic conjunctivitis due exposed to exhausts of plastic that contributed 20 (11%) participants distributed as cases 11 (55%) of in comparison with controls nine (45%). In addition, this difference between cases and controls was statistically significant (P value = 0.035).

Derivatives of petroleum shows the most kind of effect were shortness of breath that contributed110 (80.3%) of participants distributed as 89 (80.9%) cases which was more than controls that contributed 21(19.1%) and this difference was statistically significant (P value = 0.001).

### 4.3.3.5 Exposed and degree of effect due to home activities

**Table (4.34a) Relationship between cases and controls according to exposed and degree of effect due to home activities**

| | **Not exposed** | | **Exposed with no effect** | | **Expose with mild effect** | | **Exposed-moderate effect** | | **Exposed with severe effect** | | **$X^2$** | **Sig.** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Chemical cleaning substance** | | | | | | | | | | | | |
| Control | 66 | 31.0 | 87 | 40.8 | 22 | 10.3 | 22 | 10.2 | 16 | 7.5 | 85.253 | 0.001 |
| Case | 48 | 22.6 | 22 | 10.4 | 24 | 11.3 | 48 | 22.6 | 70 | 33.0 | | |
| Total | 114 | 26.8 | 109 | 25.6 | 46 | 10.8 | 70 | 16.5 | 86 | 20.2 | | |

**Table (4.33b) relationship between cases and controls according to exposed and degree of effect due to home activities**

| | Not exposed | | Exposed with no effect | | Expose with mild effect | | Exposed with moderate effect | | Exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Entrance of kitchen at time of cooking** | | | | | | | | | | | | |
| **Control** | 50 | 23.4 | 142 | 66.4 | 10 | 4.7 | 6 | 2.8 | 6 | 2.8 | 80.592 | 0.001 |
| **Case** | 45 | 21.2 | 66 | 31.1 | 27 | 12.7 | 33 | 15.6 | 41 | 19.3 | | |
| **Frying of oil** | | | | | | | | | | | | |
| **Control** | 60 | 28.4 | 124 | 58.8 | 11 | 5.2 | 10 | 4.7 | 6 | 2.8 | 95.375 | 0.001 |
| **Case** | 41 | 19.4 | 51 | 24.2 | 29 | 13.7 | 35 | 16.6 | 55 | 26.1 | | |
| **Total** | 101 | 23.9 | 175 | 41.5 | 40 | 9.5 | 45 | 10.7 | 61 | 14.5 | | |

The table (4.34) shows firstly regarding to exposed and effect of chemical cleaning substances the cases were more (66.9%) than controls (28%) and the cases that exposed and effect with highest percentage among severe effects (33%) and moderate effect (22.6%) last mild effect (11.3%) in comparison with controls (7.5%), (10.2%) and (10.3%) respectively and this relation were statically significant (P value = 0.001).

The table (4.34) shows firstly regarding to exposed and effect of frying of oil the cases were more (56.4%) than controls (12.7%) and the cases that exposed and effect with highest percentage among severe effects (26.1%) and moderate effect (16.6%) last mild effect (13.7%) in comparison with controls (2.8 %), (4.7%) and (5.2%) respectively and this relation were statically significant (P value = 0.001).

Regarding to exposed and affected to entrance of kitchen at time of cooking the percent of cases were more (47.6%) than controls (10.3%) and cases that exposed with severe effect were highest percentage (19.3%), then moderate effect (15.6%) last mild effect ( 12.7%) in comparison with controls (2.8 %), (2.8%) and (4.7 %) respectively and this relation were statically significant (P value = 0.001).

**Relationship between cases and controls according to kind of effect of exposure to chemical cleaning substances and some kitchen activities annex (16)**

Chemical cleaning substances, the most kind of effect due to it was shortness of breath that contributed 141(70.9%) of participations distributed as 116 (82.2%) cases which was more than controls that contributed 25 (17.3%) and this difference was statistically significant (P value = 0.001).Then, allergic rhinitis that contributed 67 (33.7%) participation distributed as cases 39 (58.2%) of in comparison with controls 28 (41.8%). In addition, this difference was statistically significant (P value = 0.009).

Frying oil, the most kind of effect due to it was shortness of breath that contributed 115 (82.7%) of participants distributed as 103 (89.5%) cases which more than controls that contributed 12 (10.4%) and this difference was statistically significant (P value = 0.001).

Entrance of kitchen at time of cooking, the most kind of effect due entrance of were shortness of breath that contributed 104 (84.6%) of participants distributed as 91(87.5%) cases which was more than controls that contributed 13 (12.5%) and this difference was statistically significant (P value = 0.004).

### 4.3.3.6 Mold

**Table (4-35) Relationship between cases and controls according to mold inside the home**

| | **Not exposed** | | **Exposed with no effect** | | **Exposed with mild effect** | | **Exposed with moderate effect** | | **Exposed with severe effect** | | **$X^2$** | **Sig.** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Mold inside home** | | | | | | | | | | | | |
| **Control** | 113 | 53.6 | 80 | 37.9 | 7 | 3.3 | 7 | 3.3 | 4 | 1.9 | 71.74 | 0.001 |
| **Case** | 113 | 53.1 | 22 | 10.3 | 21 | 9.9 | 26 | 12.2 | 31 | 14.6 | | |
| **Total** | 226 | 53.3 | 102 | 24.1 | 28 | 6.6 | 33 | 7.8 | 35 | 8.3 | | |

This table (4.35) shows that the difference between cases and controls according to the exposed and effect of mold inside home was statistically difference (P value = 0.001).

The researcher found the percent of cases that exposed and affected by mold (36.7%) are more than controls (8.5%) among subjects and this difference is statistically difference (P value = less than 0.001).

The researcher found that the effect of exposure to mold between subject among cases are highest especially with severe effect (14.6%) then moderate (12.2%) and finally mild (9.9%) in comparison of subject among controls (1.9%), (3.3%) and (3.3%) respectively. That meaning that there is relation between presence of mold and bronchial asthma.

**Relationship between cases and controls according to kind of effect of exposure to mold annex (17)**

The most type of effect due to exposure to mold was shortness of breath that contributed 75 (77.3%) of participations distributed as 66 (88%) cases which was more than controls that contributed (12%) and this difference was statistically significant (P value = 0.001).

Then, allergic rhinitis that contributed 28 (28.9%) participation distributed as cases 19 (67.8%) of in comparison with controls 9 (32.1%). In addition, this difference was statistically significant (P value 0.047).

#### 4.3.3.7 Dust

**Table (4-36) Relationship between cases and controls according to effect of dust inside the home**

| | Not exposed | | Exposed with no effect | | Exposed with mild effect | | exposed with moderate effect | | exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % | No | % | | |
| **Dusts inside home** | | | | | | | | | | | | |
| **Control** | 52 | 24.6 | 104 | 49.3 | 25 | 11.8 | 18 | 8.5 | 12 | 5.7 | 136.219 | 0.001 |
| **Case** | 21 | 9.9 | 22 | 10.4 | 36 | 17.0 | 52 | 24.5 | 81 | 38.2 | | |
| **Total** | 73 | 17.3 | 126 | 29.8 | 61 | 14.4 | 70 | 16.5 | 93 | 22.0 | | |

As the table (4.36) shows that the the percent of cases that exposed and affected by dust (79.75%) are more in cases than controls (26%) among subjects and this difference is statistically significant (P value 0.001).

Percentage of cases were more affected by exposed to dust among cases as mild effect (17%), moderate effect (24.5%), severe effect (38%) in comparison with controls (11.8%), (8.5%) and (5.7%) respectively and these difference were statistically significant as (P value less than 0.001) but the study find the percentage of participations that exposed to dust with no effect were among controls (49.3%) than cases (24.6%) and this difference was statistically significant (P value = 0.001).This show that the exposure of dust has relation to bronchial asthma.

**Relationship between cases and controls according to kind of effect of exposure to dust at home annex (18)**

The most type of effect due to exposure to dust was shortness of breath that contributed 161 (70.6%) of participations distributed as 136 (84.5%) cases which was more than controls that contributed 25 (15.5%) and this difference was statistically significant (P value = 0.001).

Then, allergic rhinitis that contributed 96 (28.9%) of participation distributed as cases 63 (65.6%) of in comparison with controls 33 (34.4%). Also, this difference was statistically significant (P value = 0.007).

### 4.3.3.8 Upper Respiratory Tract Infection (URTI)

**Table (4-37) Relationship between cases and controls according to exposed and degree of upper respiratory tract infection (URTI)**

| | Not exposed | | Exposed with no effect | | Expose with mild effect | | Exposed with moderate effect | | Exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % | No | % | | |
| **URTI** | | | | | | | | | | | | |
| **Control** | 51 | 23.8 | 110 | 51.4 | 20 | 9.3 | 26 | 12.1 | 7 | 3.3 | 183.005 | 0.001 |
| **Case** | 15 | 7.5 | 17 | 7.9 | 19 | 8.9 | 59 | 27.6 | 103 | 48.1 | | |
| **Total** | 67 | 15.7 | 127 | 29.7 | 39 | 9.1 | 85 | 19.9 | 110 | 25.7 | | |

Regarding to exposed and affected to URTI the percent was more in cases (84.6%) than controls (24.4%) and cases that exposed with severe effect were highest percentage (48.1%), then moderate effect (27.6%) last mild effect (8.9%) in comparison with controls (3.3%), (12.1%) and (9.3%) respectively and this relation were statically significant (P = value 0.001). In another word there is relationship between exposed of URTI with bronchial asthma and considered as risk factor.

**Relationship between cases and controls according to kind of effect of exposure to (URTI) annex (19)**

The most type of effect due to exposure of URTI was shortness of breath that contributed 188 (80.3%) of participations distributed as 161 (85.6%) cases which was more than controls that contributed 27 (14.4%) and this difference was statistically significant (P = value 0.001).

Then, allergic rhinitis due to exposure to URTI that contributed 78 (33.3%) participation distributed as cases 53 (67.9%) of in comparison with controls 25 (32.1%). In addition, this difference was statistically significant (P value = 0.013).

#### 4.3.3.9 Gastro esophageal reflux disease (GERD):

**Table (4.38) Relationship between cases and controls according to exposed and degree of effect of Gastro esophageal reflux disease (GERD):**

| | Not exposed | | Exposed with no effect | | Expose with mild effect | | Exposed with moderate effect | | Exposed with severe effect | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % | No | % | | |
| **GERD** | | | | | | | | | | | | |
| **Control** | 92 | 43.0 | 102 | 47.7 | 5 | 2.3 | 7 | 3.3 | 8 | 3.7 | 91.893 | 0.001 |
| **Case** | 77 | 36.0 | 34 | 15.9 | 34 | 15.9 | 39 | 18.2 | 30 | 14.0 | | |
| **Total** | 169 | 39.5 | 136 | 31.8 | 39 | 9.1 | 46 | 10.7 | 38 | 8.9 | | |

Table (4.38) show that exposed and affected to GERD. The percent of cases were more (48.1%) than controls (9.3%) and cases that exposed with moderate effect were highest percentage (18.2%), then mild effect (15.9%) last sever effect (14%) in comparison with controls (3.3%), (2.3%) and (3.7%) respectively and this relation were statically significant (P value = 0.001).

In addition, annex (20) shows the all types of effect (shortness of breath, allergic dermatitis, allergic conjunctivitis and allergic dermatitis) due to exposure of GERD did not reach statistically significant level.

### 4.3.3.10 Psychological conditions

**Table (4-39) Relationship between cases and controls according to psychological conditions**

| | **Not exposed** | | **Exposed with no effect** | | **Expose with mild effect** | | **Exposed with moderate effect** | | **Exposed with severe effect** | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **No** | % | **No** | % | **No** | % | **No** | % | **No** | % | | |
| **Depression** | | | | | | | | | | | | |
| **Control** | 59 | 27.6 | 114 | 53.3 | 21 | 9.8 | 9 | 4.2 | 11 | 5.1 | 104.205 | 0.001 |
| **Case** | 42 | 19.6 | 37 | 17.3 | 26 | 12.1 | 56 | 26.2 | 53 | 24.8 | | |
| **Total** | 101 | 23.6 | 151 | 35.3 | 47 | 11.0 | 65 | 15.2 | 64 | 15.0 | | |
| Grief | | | | | | | | | | | | |
| **Controls** | 56 | 26.2 | 115 | 53.7 | 19 | 5.9 | 17 | 7.9 | 7 | 3.3 | 125.723 | 0.001 |
| **Case** | 27 | 12.6 | 34 | 15.9 | 29 | 13.6 | 62 | 29.8 | 53 | 29.0 | | |
| **Total** | 83 | 19.4 | 149 | 34.8 | 48 | 11.2 | 79 | 18.5 | 69 | 16.1 | | |
| Stress | | | | | | | | | | | | |
| **Control** | 49 | 22.9 | 120 | 56.1 | 16 | 7.5 | 18 | 8.4 | 11 | 5.1 | 131.166 | 0.001 |
| **Case** | 20 | 9.3 | 33 | 15.4 | 32 | 15.0 | 64 | 29.9 | 65 | 30.4 | | |
| **Total** | 69 | 16.1 | 153 | 35.7 | 48 | 11.2 | 82 | 19.2 | 76 | 17.8 | | |

Regarding to exposed and affected by depression, the percent of cases were more (63.1%) than controls (19.1%) and cases that exposed with moderate effect were the highest percentage(26.2%), then severe effect (24.8%) last mild effect (12.1%) in comparison with controls (4.2%), (5.1%) and (9.8%) respectively and this relation was statically significant (P value = 0.001).

Regarding to exposed and affected by grief the percent of cases were more (72.4%) than controls (17.1%) and cases that exposed with moderate effect the highest percentage (29.8%), then severe effect (29%) last mild effect (13.6%) in comparison with controls (7.9%), (3.3%) and (5.9%) respectively and this relation were statistically significant (P value = 0.001).

Regarding to exposed and affected by stress the percent of cases were more (75.3%) than controls (21%) and cases that exposed with severe effect were the highest percentage (30.4%), then moderate effect (29.9%) last mild effect (7.5%) in comparison with controls (5.1%), (8.4%) and (10.3%) respectively and this relation was statistically significant (P value = 0.001). So in other word, there are relation between psychological problem (depression, grief, stress) with bronchial asthma**.**

**Relationship between cases and controls according to kind of effect of exposure to psychological problems. Annex 21**

First the types of effect of depression among participants, the researcher found just shortness of breath was statistically significant (P value 0.042) as shortness of breath represent 148 (85.1%) among participants that exposed and affected by depression and almost of them among cases (118 out of 148) than controls (30 out of 148) and this difference was statistically significant (P value = 0.024) .

Then regarding the types of effect of grief, the researcher found just shortness of breath was statistically significant (P value 0.022) as shortness of breath represent 178 (90.8%) among participants that exposed and affected by grief and almost of them among cases (134 out of 178) than controls (30 out of 148) and this difference was statistically significant (P value = 0.022).

Finally regarding the types of effect of stress among participates, the researcher found just shortness of breath was statistically significant (P value 0.008) as shortness of breath represent 184 (89.3%) among participates that exposed and affected by stress and most of them more in cases (149 out of 184) than controls (35 out of 184) and this difference was statistically significant (P value = 0.008) .

Otherwise for all depression, grief, stress (allergic rhinitis, allergic dermatitis, allergic conjunctivitis) not statistically significant.

**4.3.3.11Food allergy**

**Table (4-40) Relationship between cases and controls according to Allergy because of food**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No | % | | |
| **Allergy for food** | **21** | **9.8** | **60** | **28.0** | **81** | **18.9** | **23.161** | **0.001** |
| Chocolate | 5 | 23.8 | 12 | 20.0 | 17 | 21.0 | 0.136 | 0.465 |
| Tomato | 3 | 14.3 | 13 | 21.7 | 16 | 19.8 | 0.535 | 0.351 |
| Mango | 1 | 4.8 | 11 | 18.3 | 12 | 14.8 | 2.270 | 0.122 |
| Canned food | 3 | 14.3 | 8 | 13.3 | 11 | 13.6 | 0.012 | 0.585 |
| Fruit | 5 | 23.8 | 12 | 20.0 | 17 | 21.0 | 0.136 | 0.465 |
| Vegetable | 1 | 4.8 | 3 | 5.0 | 4 | 4.9 | 0.002 | 0.725 |
| Freezers | 1 | 4.8 | 7 | 11.7 | 8 | 9.9 | 0.833 | 0.332 |
| Meat | 0 | 0.0 | 4 | 6.7 | 4 | 4.9 | 1.473 | 0.293 |
| Spices | 4 | 19.0 | 27 | 45.0 | 31 | 39.3 | 4.435 | 0.030 |
| Soft-drink | 0 | 0.0 | 13 | 21.7 | 13 | 16.0 | 5.420 | 0.014 |
| Others | 5 | 23.8 | 22 | 36.7 | 27 | 33.3 | 1.157 | 0.212 |

This table (4.40) shows that allergy to food contributed 81(18.9%) of all participations was more common between cases (60 participants representative 28% of cases) than controls (21participants, representative 9.8% of controls) and this difference was statistically significant (P value less than 0.001). Also, cases about all types of food that mentioned in table above were more than controls, but the positive relation appear clear at spices and soft drink that were more in cases (45%), (21.7%) than control (19%), (zero) respectively with P value statistically significant (0.030), (0.014) respectively. In another word there is relationship between bronchial asthma and allergic to food.

### 4.3.3.12 Drugs allergy

**Table (4-41) Relationship between cases and controls because of Drugs allergy**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Drug allergy** | 12 | 5.6 | 45 | 21.0 | 57 | 13.3 | 22.041 | 0.001 |
| **Paracetamol allergy** | 0 | 0.0 | 1 | 2.3 | 1 | 1.8 | 0.278 | 0.782 |
| **Baby aspirin** | 3 | 25.0 | 18 | 40.0 | 21 | 36.8 | 0.916 | 0.272 |
| **NSAID** | 4 | 33.3 | 32 | 72.7 | 36 | 64.3 | 6.373 | 0.016 |
| **others** | 8 | 61.5 | 6 | 14.0 | 14 | 25.0 | 12.022 | 0.000 |

This table (4.41) shows that in generals allergy to drug between all subjects was (57 participants) that contributed about 13.3% of all participations. Also most of them between cases (45 participations representative 21% of cases) more than controls (12 participations representative 5.6% of controls) and this difference was statistically significant (P value less than 0.001). That meaning there is positive relation between drug allergy and bronchial asthma.

The relation between drug allergy and the bronchial asthma was clear with NSAID as cases (72.7%) more than controls (33.3%) with (P value 0.016).That meaning there is positive relation between NSAID and bronchial asthma.

Also, there is relation between bronchial asthma and allergy to baby aspirin as table showed the cases with baby aspirin allergy more among cases (40%) in comparison with controls (25%) but this relation not reach statistically significant.

### 4.3.3.13 Exercises

**Table (4-42) relationship between cases and controls according to exercises**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Exercise (Physical Activity)** | | | | | | | | |
| **NO** | 108 | 50.9 | 131 | 61.5 | 239 | 56.2 | 4.813 | 0.018 |
| **YES** | 104 | 49.1 | 82 | 38.5 | 186 | 43.8 | | |
| **Total** | 212 | 100.0 | 213 | 100.0 | 425 | 100.0 | | |
| **Period of exercise by (minutes)** | | | | | | | | |
| **<= 20** | 16 | 19.8 | 10 | 15.2 | 26 | 17.7 | 1.431 | 0.698 |
| **21 - 30** | 33 | 40.7 | 28 | 42.4 | 61 | 41.5 | | |
| **31 to 40** | 1 | 1.2 | 0 | 0.0 | 1 | 0.7 | | |
| **Above40** | 31 | 38.3 | 28 | 42.4 | 59 | 40.1 | | |
| **Total** | 81 | 100.0 | 66 | 100.0 | 147 | 100.0 | | |
| **Times per weeks** | | | | | | | | |
| **<=2** | 32 | 36.0 | 31 | 43.1 | 63 | 39.1 | 1.559 | 0.459 |
| **3 to 4** | 30 | 33.7 | 18 | 25.0 | 48 | 29.8 | | |
| **Above 4** | 27 | 30.3 | 23 | 31.9 | 50 | 31.1 | | |
| **Total** | 89 | 100.0 | 72 | 100.0 | 161 | 100.0 | | |
| **Type of Exercises** | | | | | | | | |
| **Walk** | 68 | 73.1 | 63 | 79.7 | 131 | 76.2 | 6.494 | 0.165 |
| **Running** | 7 | 7.5 | 8 | 10.1 | 15 | 8.7 | | |
| **Football** | 14 | 15.1 | 7 | 8.9 | 21 | 12.2 | | |
| **Body-building** | 0 | 0.0 | 1 | 1.3 | 1 | 0.6 | | |
| **Fitness** | 4 | 4.3 | 0 | 0.0 | 4 | 2.3 | | |
| **Total** | 93 | 100.0 | 79 | 100.0 | 172 | 100.0 | | |

This table (4.42) shows that physical activity (exercise) more common among controls (49.1%) than cases (38.5%) and this difference was statistically significant (P value = 0.018)

In addition the researcher found that exercise duration especially when it takes 21-30 minutes and >40 minutes are longer among cases (42.4%) and (42.4%) than controls (40.7%) and (38.3%) respectively but this relation was statically not significant

With respect to time of exercise/week and type of exercise.

## 4.3.3.14 BMI

**Table (4-43) Relationship between cases and controls according to BMI**

| BMI | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Underweight** | 6 | 3.0 | 14 | 7.1 | 20 | 5.0 | 7.667 | 0.05 |
| **Normal Weight** | 48 | 24.1 | 61 | 30.8 | 109 | 27.5 | | |
| **Overweight** | 79 | 39.7 | 59 | 29.8 | 138 | 34.8 | | |
| **Obese** | 66 | 33.2 | 64 | 32.3 | 130 | 32.7 | | |
| **Total** | 199 | 100.0 | 198 | 100.0 | 397 | 100.0 | | |

This table (4.43) shows over weight and obese more common among controls (39.7%) and (33.2%) in comparison with cases (29.8%) and (32.3%) respectively and this relation is not statistically significant

### 4.3.3.15 Family history

**Table (4.44) Relationship between cases and controls according to family history of bronchial asthma**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Family history** | | | | | | | | |
| **NO ( negative)** | 173 | 83.6 | 126 | 59.2 | 299 | 71.2 | 30.526 | 0.001 |
| **YES (positive)** | 34 | 16.4 | 87 | 40.8 | 121 | 28.8 | | |
| **Total** | 207 | 100.0 | 213 | 100.0 | 420 | 100.0 | | |

Table (4.44) shows positive family history more among cases (40.8%) in comparison with controls (16.4%) and this relation was statistically significant (P value = 0.001).
That meaning there is a relationship between family history (genetic factors) and bronchial asthma.

#### 4.3.3.16 Seasonal (weather) variations

**Table (4.45) the effect of seasonal variation**

| | **attacks** | **No attacks** | **OR** | **Confidence interval** | **Sig.** |
|---|---|---|---|---|---|
| **Summer** | 111 | 315 | 4.135 | (2.743-6.231) | 0.001 |
| **Autumn** | 118 | 306 | 3.946 | (3.065-5.079) | 0.001 |
| **Spring** | 173 | 253 | 3.544 | (2.718-4.619) | 0.001 |
| **Winter** | 203 | 224 | 3.271 | (2.362-4.530) | 0.001 |

This table (4-45) shows the effect of different seasons on bronchial asthma, researcher found the most risky season is summer more than fourfold as OR (CI) = 4.135 (2.743-6.231) then autumn less than fourfold as OR (CI) = 3.946 (3.065-5.079) next spring three and half fold as OR (CI) = 3.5 (2.718-4.619) and lastly winter more than threefold OR (CI) = 3.271 (3.362-4.530) .

**Table (4-46) relationship between cases and controls regarding the severity of seasonal (weather) variations effects.**

| | | exposed with no effect | | expose with mild effect | | exposed with Moderate effect | | exposed with Sever Effect | | $X^2$ | Sig |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | No. | % | No. | % | No. | % | No | % | | |
| **Winter** | | | | | | | | | | | |
| **Control** | | 180 | 84.1 | 124 | 6.5 | 13 | 6.1 | 7 | 3.3 | 178.9 | 0.001 |
| **Case** | | 44 | 20.7 | 36 | 16.9 | 48 | 22.5 | 85 | 39.9 | | |
| **Total** | | 224 | 52.5 | 50 | 11.7 | 61 | 14.3 | 92 | 21.5 | | |
| **Summer** | | | | | | | | | | | |
| **Control** | | 203 | 94.9 | 5 | 2.3 | 2 | 0.9 | 4 | 1.9 | 99.061 | 0.001 |
| **Case** | | 112 | 52.8 | 42 | 19.8 | 27 | 12.7 | 31 | 14.6 | | |
| **Total** | | 315 | 74.0 | 47 | 11.0 | 29 | 6.8 | 35 | 8.2 | | |
| **Autumn** | | | | | | | | | | | |
| **Control** | | 196 | 92.0 | 8 | 3.8 | 5 | 2.3 | 4 | 1.9 | 90.091 | 0.001 |
| **Case** | | 110 | 52.0 | 33 | 15.6 | 36 | 17.1 | 32 | 15.2 | | |
| **Total** | | 306 | 72.1 | 41 | 9.7 | 41 | 9.7 | 36 | 8.5 | | |
| **Spring** | | | | | | | | | | | |
| **Control** | | 188 | 87.8 | 10 | 4.7 | 8 | 3.7 | 8 | 3.7 | 145.763 | 0.001 |
| **Case** | | 65 | 30.6 | 39 | 18.4 | 50 | 23.6 | 58 | 27.4 | | |
| **Total** | | 253 | 59.6 | 49 | 11.5 | 58 | 13.6 | 66 | 15.5 | | |

## Winter

This table (4.46) shows that the difference between cases and controls according to the effect of winter was statistically significant (P value = 0.00) so there was positive relation between winter and bronchial asthma .The percent of cases that affected by winter (79.3%) was more than controls (15.9%) and this difference was statistically significant (P value = 0.01).

The researcher found that the effect of exposure to winter among cases are the highest with severe effect (39.9%) then moderate (22.5%) and finally mild (16.9%) in comparison with controls (3.3%), (6.1%) and (6.5%) respectively.

**Summer**

This table shows that the difference between cases and controls according to the effect of summer was statistically significant (P value = 0.001). The percent of cases that affected by summer (47.1%) are more than controls (5.1%). The researcher found that the effect of summer among cases are higher especially with sever effect (14.6%), moderate (12.7%) and mild (19.8%) in comparison to controls (1.9%), (0.9%) and (2.3%) respectively.

**Autumn**

This table shows that the difference between cases and controls according to autumn was statistically significant (P value = 0.001).The percent of cases that affected by autumn (47.9%) are more than controls (8%) this difference was statistically significant (P value =0.001).The researcher found that the severity of the effect of autumn among cases were highest especially with moderate effect (17.1%) then mild (15.6%) and finally severe (15.2%) in comparison with controls (2.3%), (3.8%) and (1.9%) respectively .

**Spring**

This table shows that the difference between cases and controls according to the effect of spring was statistically significant (P value = 0.001).The percent of cases that affected by spring (69.4 %) were more than controls (12.1%) and this difference was statistically significant (P value = 0.001). The effect of spring among cases with sever effect (27.4%) then moderate (23.6%) and finally mild (18.4%) in comparison to controls (15.5%), (13.6%) and (11.5%) respectively.

In conclusion, the severity of bronchial asthma increase in winter and as it is noticed in, the table the winter is has the highest severity (39.9%), spring (27.4%) autumn (15.2%) finally summer (14.6%).

**Relationship between cases and controls according to kind of effect of exposure to seasonal variation (weather). Annex (22).**

**Winter**

Shortness of breath affect 151 participants that represent (76.3%), cases (138 out of 151) 91.3% more than controls that represent (13 out of 151) 8.6% and this difference reached statistically significant level (P value= 0.001).

In addition, allergic rhinitis among participants represent 73 (36.9%) that affected by winter (53 out of 73) 72.6% cases while controls (20 out of 73) 27.4% and this difference was statistically significant (P value = 0.002).

Skin dermatitis and allergic rhinitis more among cases than controls regarding to who affected by winter, but these differences did not reached statistically significant level.

**Summer**

According to participants that affected by summer most of the effect and that reached statistically significant level among who suffer from shortness of breath 82 participants (75.2%) in which the cases represent (78 out of 82) 95.1% more than controls that represent (4 out of 82) 4.9% and this difference was statistically significant (P value = 0.001).

Allergic rhinitis, skin dermatitis and conjunctivitis were more between cases than controls regarding to affect by summer, but these differences not reached statistically significant level.

**Autumn**

Shortness of breath represent 84 participants that represent (71.8%), cases were (78out of 84) 92% more than controls that represent (6 out of 84) and this difference was statistically significant ( P value = 0.001).

Allergic rhinitis among participants that represents 38(32.5%) of participations that exposed and affected by autumn , among them cases (27 out of 38) 71% more than controls (11 out of 38) 28.9% and this difference reached statistically significant level (P value 0.007).

Also skin dermatitis and allergic conjunctivitis more between cases than controls regarding to who exposed and affected by winter, but these difference were statistically not significant.

**Spring**

Shortness of breath represent 117 (70.5%), cases (112out 117) was more than controls that represent (5 out of 117) 95.7 % and this difference was statistically significant (P value = 0.001).

Also, allergic rhinitis between participants that represent 54 (32.5%) of participants that affected by spring among them cases (39 out of 54) 72.2% more than controls (15out of 54) 27.7% and this difference was statistically significant (P value 0.004).

Also skin dermatitis and allergic rhinitis more among cases than controls regarding to who affected by winter, but these differences were statistically not significant.

**Logistic regression**

**Table (4-47) Logistics Regressions (A) for exposure to some environmental factors**

| | | B | S.E. | Wald | Sig. | Exp(B) | 95% C.I. for EXP(B) | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper |
| Step 1[a] | Soil | .152 | .139 | 1.200 | .273 | 1.165 | .887 | 1.530 |
| | Cement | -.040- | .137 | .084 | .772 | .961 | .734 | 1.258 |
| | Perfume | .601 | .143 | 17.725 | .000 | 1.824 | 1.379 | 2.413 |
| | Exhausts of vehicles | .225 | .158 | 2.027 | .155 | 1.252 | .919 | 1.708 |
| | Exhausts of wood | -.114- | .168 | .460 | .497 | .892 | .642 | 1.240 |
| | Exhausts of plastic | -.019- | .164 | .014 | .907 | .981 | .711 | 1.353 |
| | Chemical cleaning substance | -.201- | .139 | 2.091 | .148 | .818 | .623 | 1.074 |
| | Dust inside home | .319 | .136 | 5.536 | .019 | 1.376 | 1.055 | 1.796 |
| | Mold inside home | .019 | .162 | .014 | .907 | 1.019 | .742 | 1.401 |
| | Derivations of petroleum | -.182- | .168 | 1.177 | .278 | .834 | .600 | 1.158 |
| | Fraying Oil | .203 | .192 | 1.121 | .290 | 1.225 | .841 | 1.784 |
| | Entrance site of coking | -.172- | .199 | .745 | .388 | .842 | .570 | 1.244 |
| | Grief | .155 | .188 | .678 | .410 | 1.167 | .808 | 1.687 |
| | Stress | .392 | .190 | 4.245 | .039 | 1.480 | 1.019 | 2.150 |
| | URTI | .707 | .133 | 28.456 | .000 | 2.029 | 1.564 | 2.631 |
| | GERD | -.190- | .140 | 1.836 | .175 | .827 | .629 | 1.088 |
| | Constant | 5.562- | .544 | 104.510 | .000 | .004 | | |

**Table(4.48)Logistic regression (B)**

| | | B | S.E | Wald | Sig | EXP(B) | 95% C.I. for EXP(B) | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | upper |
| Step 1[a] | Family number | .001 | .040 | .000 | .988 | 1.001 | .925 | 1.083 |
| | Educational level | -.230- | .084 | 7.467 | .006 | .795 | .674 | .937 |
| | working | -.189- | .271 | .486 | .486 | .828 | .487 | 1.407 |
| | Exposure to Tobacco smoke | .507 | .262 | 3.761 | .048 | 1.661 | 1.007 | 2.773 |
| | Carpet | -.356- | .305 | 1.362 | .243 | .700 | .385 | 1.274 |
| | Mokit | -.635- | .337 | 3.542 | .060 | .530 | .274 | 1.027 |
| | Mat | -.240- | .289 | .691 | .406 | .786 | .446 | 1.386 |
| | Home size | -.001- | .003 | .044 | .834 | .999 | .994 | 1.005 |
| | No of rooms | -.060- | .129 | .219 | .640 | .941 | .731 | 1.212 |
| | Heating | .164 | .081 | 4.080 | .043 | 1.179 | 1.005 | 1.382 |
| | Air Cooling | .564 | .235 | 5.753 | .016 | 1.757 | 1.109 | 2.785 |
| | Food allergy | 1.135 | .359 | 10.022 | .002 | 3.112 | 1.541 | 6.286 |
| | Drug allergy | 1.667 | .417 | 15.953 | .001 | 5.299 | 2.338 | 12.009 |

Table (4.47) shows some risk factors for bronchial by logistic regression in our study. By the above model, the researcher found the perfume was risky on bronchial asthma by (82.4%) as OR (CI) = 1.824 (1.379-2.413) also dust in home was risky on bronchial asthma by (37.6%) as OR (CI) = 1.824 (1.005-1.796). Also, the researcher found stress increase the risk for bronchial asthma by 48% as OR (CI) =1.480 (1.019-2150) and URTI increase the risk by two fold as OR (CI) = 2.029 (1.564-2.63). So stress and URTI are risk factors of bronchial asthma.

Table (4-48) shows that as the level of education decrease the risk for bronchial asthma increase by 21% OR (CI) = 0.795 (0.674 -0.937).Exposure to all types of smoking increase the risk for bronchial asthma by 66% OR (CI) = 1.66 (1.007-2.773). In addition, the researcher found that not using things for heating at cool weather is risk factors for bronchial asthma by 17.9% OR (CI) = 1.179 (1.007-2.773). Also not using things for cooling air in hot and humid weather increase the risk for bronchial asthma by 75.7% OR (CI) = 1.757 (1.109-2.785). According to food allergy the researcher found that food allergy increase the risk for bronchial asthma by three fold OR (CI) = 3.001 (1.541-6.286). Also family history (genetic) increase the risk for bronchial asthma nearby fourfold OR (CI) =3.7(2.071-6.619). Also drug allergy increase the risk for bronchial asthma by fivefold as OR (CI) = 5.299 (2.338-12.009).

In conclusion, from two tables, perfume, dust in-home, stress, URTI, low level of education, exposure to tobacco smoke, not using things for heating or cooling, food allergy, drug allergy and family history are risk factors for bronchial asthma.

# Chapter (5)

# Discussion

As aforementioned, the present study was designed to investigate risk factors such as socio-demographic, socioeconomic, environmental, psychological and other factors of bronchial asthma in the Gaza Strip.

The study topic was selected because of the increase of prevalence of bronchial asthma all over the word (D'Amato et al., 2002).The impact of bronchial asthma on the health status as well as the burden of the cost on the health care system is high and it is cause of substantial burden to people, often causing a reduced quality of life, not only due to its physical effects, but also its psychological and social effects (GlobalAsthmaNetwork, 2014). Additionally, there are no researches about bronchial asthma in Gaza Strip.

In this chapter, the researcher attempts to explain the finding in the light of other studies and points to the implications of that in developing preventive programs aiming to decrease burden of bronchial asthma in the Gaza Strip.

## 5.1 Demographic factors

### 5.1.1 Family size

The researcher investigated the relationship between bronchial asthma and risk of family size. The study concluded that when number of family size increase above 8 the cases of asthma increase, in other words the family size has relation with the bronchial asthma when the number of family size increase above 8 members.

These results are not agreement to some studies that showed the "protective" effects of having a higher number of siblings for the risk of atopic eczema, asthma wheezing, hay fever, and allergic sensitization, one of them a study that review of the literature for 53 different studies were identified; for asthma and wheezing, 21 of 31 reported the inverse association; supported the "protective" effect of a higher number of siblings (Karmaus & Botezan, 2002).

## 5.2 Socioeconomic variations (income, work & educations).

The researcher investigated the relationship between bronchial asthma and risk of socioeconomic factors (income, level of education, working). The study concluded that low educational level (secondary school and less), low income (=<1000NIS) and joblessness have relations to bronchial asthma. The main core among them is low level of education in Gaza Strip as the researcher found that the low level of education increase the risk of bronchial asthma by 21% OR (CI) = 0.795 (0.677 - 0.937).

As unemployment rate by mid-2016 was almost 42%, among the global highs, while among youth it stood at 60% and among females at over 65% and 47% of households in Gaza suffer from moderate or severe food insecurity. More than 70% of Gaza's population receives some form of international aid, the bulk of which is food assistance. The blockade of Gaza, which entered its tenth year in 2016, continued to undermine basic human rights and economic prospects, as well as the availability of essential services, exacerbating poverty and aid dependency. Longstanding access restrictions imposed by Israel have undermined Gaza's economy, resulting in high levels of unemployment, food insecurity and aid dependency. These include restrictions on the marketing of goods in the West Bank and Israel; on the import of certain goods; and on the access of people to agricultural land and fishing waters. The partial easing of some of these restrictions over the past two years has not enabled a significant reactivation of the economy. This is compounded by recurrent hostilities, which result in the loss of assets and people being left with long-term disabilities; the energy crisis; and the internal Palestinian division all these destroy so socioeconomic status for Palestinian people who live in Gaza Strip (OCHA, 2016)**.**

In conclusion, all socio economic factors that the researcher studied have relation to bronchial asthma and the most affected factors for bronchial asthma and consider as risk factors is low educational level and these result supported by many studies as Hassan et al. 2002 in Bangladesh that showed that the low-income group (OR=1.41,95% CI:1.04–1.92) and illiterate group (OR=1.51, 95% CI:1.01–2.24) were more vulnerable to asthma attacks than the high-income group and more educated people, respectively (Hassan et al., 2002).

Also study by Global Asthma Network at 2014 the most affected people with bronchial asthma are in low- and middle-income countries, and its prevalence is estimated to be increasing fastest in those countries ( Global Asthma Network 2014).

The National Asthma Education and Prevention Program (NAEPP) did study that aimed to examine the relationship between asthma control and socio-demographic characteristics, health-care access and asthma education, and medication use among adults with active asthma residing in New England from the 2006. Behavior Risk Factor Surveillance System Adult Asthma Call-Back Survey was analyzed using multinomial logistic regression. The two of the results in this research that unemployed or unable to work (OR =17.9; 95% CI =6.0–53.4), high school educated or less (OR = 2.8; 95% CI =1.6–4.7) were associated with very poorly controlled asthma of targeted intervention (NGUYEN et al., 2011).

Also Forrest & Dudley 2013 found households with the lowest income levels per year had the highest prevalence of lifetime and current asthma (Forrest & Dudley, 2013) Another study that was done to assess whether the association with social class differed between allergic rhinitis and asthma. The Swedish Military Service Conscription Register was linked to two other national registers for 1,247,038 male conscripts in successive cohorts born between 1952 and 1977. The steepest increase in asthma and allergic rhinitis occurred in conscripts with a low socio-economic status (Hjern et al., 2005).

Another study Grant et al. 2000 described relations between socioeconomic factors and race/ethnicity as risk factors for asthma mortality by cross-sectional study was conducted of US mortality records from 1991 through 1996 was demonstrate low vs high educational level (1.51 vs 0.69), and low vs high income (1.46 vs 0.71) (Grant et al. 2000)

Fiori et al. 2012 did Study in Rio Grande do Sul State, Brazil by cross sectional study for adults(20-69 years).Asthma was more frequent among people with low family income (Fiori et al., 2012).

Also, study done in USA found that Current asthma prevalence differs between groups, with higher rates among those with family income below the poverty level (Akinbami et al., 2011).

NGUYEN et al. 2011 did the study that aimed to examine the relationship between asthma control and socio-demographic characteristics, health-care access and use, asthma education, and medication use among adults with active asthma residing in New England. One of the results in this research demonstrated that unemployed or unable to work

(OR =17.9; 95% CI= 6.0–53.4 (NGUYEN et al., 2011).

As our study demonstrate that low educational level (from secondary school and below) is most the important factors among socioeconomic factors and considered as risk factors as our study demonstrate the low level of education increase the risk for bronchial asthma by

21%  OR ( CI) = 0.795 (0.674-0.937). Also low income and joblessness have relations to bronchial asthma.

Therefore patient with bronchial asthma need more counseling about their diseases, avoid exposure to risk factors, compliance with drugs and aggravating factors as most of them have low level of education so health education, health promotion sessions and counseling could help to avoid the effect of these socioeconomic factors.

## 5.3 Indoor pollution

### 5.3.1 Building character:

The researcher investigated the relation between some of building characters as type of house, house land, land cover, house painting, home size, number of room, bed room size and number of persons in bed room (crowding index) with bronchial asthma. After investigations the study showed relations between type of building either by asbst or zinko, uses of flagstones or baton for house land, uses of Mat for land cover, polygad for painting, small house size (less than 140 m2), two rooms or less inside home, number of persons in bed room (crowding index) above 4 persons and bronchial asthma.

Most of these factors are related to low socioeconomic as poly gad for painting considered as the  cheapest type and also difficult to clean with  high mold growth over it also use of mat that cover land usually have a lot of dust and usually cleaned by old model that lead to emission of the dust.

Regarding house type (zinco-Asbast), as study demonstrated there is relation between living in  houses that building either by asbst or zinko these reflect many things low socioeconomic, at time of summer high temperate inside home, at time of winter low temperate, usually more mold and dust inside these home . In conclusion, many factors play role in this old fission of houses .This result supported by study at 2011 that concluded post review of articles there is no relationship between asbestos exposure alone and airway obstruction. However, one study has reported that asbestos exposure could cause asthmatic symptoms, although the subjects were also exposed to dusts other than asbestos. In addition, serum immunoglobulin E (IgE) elevation has been reported in asbestos-exposed workers and asbestos exposure may cause an atopic condition. They suspect that asbestos exposure itself is a risk factor for the development of bronchial asthma. Bronchial asthma might develop by asbestos exposure independent of dose. Previous studies of nonmalignant respiratory disease caused by asbestos exposure lack appropriate examinations of allergic status, such as sputum eosinophil population, fractional exhaled nitric oxide concentration, bronchial hyperresponsiveness, serum IgE levels, and radioallergosorbent testing. Asbestos exposure may cause airway obstruction by inducing bronchial asthma, even if confirmed asbestosis is absent. To clarify this hypothesis, further investigation will be needed.(Toyoshima et al., 2011).

Regarding small size of house less than 140m2 and low number of room (two or less) that may reflect low socioeconomic also they affected by in indirect way as they lead to psychological problem due to crowdedness as in this study also find relation between bronchial asthma and high member of family above 8 member and psychological problem. All character above of building have relation to low socioeconomic factors as all sample selected from UNRWA clinics and most of them live inside the camps that characterized by bad housing conditions, old building, crowdedness, bad ventilations and bad sanitations.

Regarding land cover our study demonstrated that land cover with mat have relation to bronchial asthma this relation could be due to bad way of cleaning, old mat that lead mat source of emission of dust and many small particles. The dust below them is heavy and cleaned usually not by electric vacuum.

Regarding the carpet and mokait have no relation to bronchial asthma in this study in opposite to some studies as, Sauerhoff 2008 multiple studies have reported fewer allergy and asthma symptoms associated with carpet (Sauerhoff, 2008)**.** The carpet or mokait if present in houses, they does not cover all house land and if present it cover small pieces in special area of the houses and usually clean so it is not contributed to be a risk factor for asthma. Also the usual habit of most people to remove shoes out of the house that let the mokait and carpet clean. In addition, they usually washed by water and soup at least twice yearly every EID. From the previous result, the carpet and mokait are not risk factors for bronchial asthma in Gaza Strip but mate have relation to bronchial asthma.

Dr. Berry states that main problem is dirty carpet as bio pollutants occur naturally in the outdoor and indoor environment and include pollen, animals, dander, house dust**,** Mites, mold, and cockroaches. At least some bio pollutants (e.g., animals, dander, house dust mites, and mold) have been linked to allergies and asthma. “The main conclusion of his research is that clean carpet does not support mold growth even at prolonged and elevated temperature and humidity levels. It is a conclusion for this project that for any material. Dirt + Water (High Humidity) = Mold Growth. The obvious management solution for mold indoors is to keep all carpet materials dry or at least clean (Sauerhoff, 2008).

Alan E. Luedtke published a comprehensive review of the literature regarding the indoor environment, floor coverings, dusts, and airborne exposures. More specifically, he addressed the composition of soils and dusts, floor loadings, surface loading rates, relationships to re-suspension of dust components and airborne exposures, floor coverings, dust and airborne contaminants. He summarized his findings as follows. “The majority of the contaminants that accumulate on smooth floors, in carpet, and on other surfaces appear to be outdoor-sourced. Dusts and soils on flooring were mostly the result of foot traffic. As expected, carpet usually carried a higher burden of soils, dusts, and trace contaminants per unit area than smooth surfaces. For a large number of contaminants, the levels were similar on a per gram of dust basis (Sauerhoff, 2008).

### 5.3.2 Animal and plant inside home

The researcher investigated the relationship between bronchial asthma and animal and plant inside home. The study concluded that there is no relation between animal and plant inside home and bronchial asthma as this result agreement to study demonstrated that early life exposure to animals does not make children more likely to develop allergies or asthma and that there may actually be a protective effect. Thus having animals in the home when children are born may be beneficial to their immune development and is unlikely to be detrimental. However, if children do develop allergies or asthma, the decision of whether or not to keep household pets may need to be reevaluated. For allergic families who wish to keep their pets, there are medications and environmental management strategies which may help control their condition (Trembath, 2015).

### 5.3.3 Heating

The researcher investigated the relationship between types of heating as risk factors for bronchial asthma. The researcher found traditional way for heating by either wood and coal combustion and subject who use (nothing) for heating has relation to bronchial asthma.

These result are similar to previous studies that concluded that there is a positive association between exposure to coal combustion and bronchial asthma .An increased risk was observed for use of coal for heating (Zheng et al., 2002).

Another study that investigated household factors and asthma in four Chinese cities (Lanzou, Chongquig, Wuhan, and Gangzhou). In each city, one urban area with high ambient pollution and one suburban area with low ambient pollution were chosen. Heating with coal was associated with cough with phlegm (OR = 1.29, CI 1.11–1.50), wheeze (OR = 1.22, CI 1.02– 1.45), and asthma in general, exposure from .indoor combustion for heating was more consistently associated with risk. This may reflect the higher exposure from heating devices that are used for many hours per day. Also Coal is composed primarily of carbon, but it also contains sulfur, oxygen and hydrogen. Emissions from coal combustion include a large number of air pollutants such as particulate matter, carbon monoxide, sulfur dioxide, nitrogen oxides, and organic toxics. Overall, it appears that exposure to indoor combustion sources may increase the risk of asthma or asthma severity, particularly in children (Belanger & Triche, 2008).

In conclusion, patient with bronchial asthma must increase awareness about, how to deal with heating in winter–weather, by forward them to use electricity or gas for heating and not use wood or coal as these considered as most sources of indoor air pollutions ($CO_2$ and $NO_2$) specially as most of people who use this methods stay long time in front of fire and its emissions.

Also the study found that not using things for heating is also consider as a risk factor for bronchial asthma by 17.9% OR (CI) = 1.179 (1.007-2.773) as inspiration of cold air is stimulating and exacerbating bronchial asthma. So per each winter, it's better to increase awareness of patient of bronchial asthma about the safest way for heating and avoid exposure too cold. Also Inspiration of cold air leads indirectly to drying out and subsequent constriction of the airways.Breathing in through the nose is also more difficult in extremely cold weather. In asthmatics, mouth breathing leads to even more constriction of the airways, as the air is not pre-warmed as it is breathed in (Astma- og Allergiforbundet, 2007).

Also cold air is dry as airways are lined with a thin layer of fluid. When you breathe in dry air, that fluid evaporates faster than it can be replaced. Dry airways become irritated and swollen, which worsens asthma symptoms. Cold air also causes your airways to produce a substance called histamine, which is the same chemical your body makes during an allergy attack. Histamine triggers wheezing and other asthma symptoms. Also Cold increases mucus as airways are lined with a layer of protective mucus, which helps remove unhealthy particles. In cold weather, your body produces more mucus, but it is thicker and stickier than normal. The extra mucus makes you more likely to catch a cold or other infection (Watson, Henline, & Sullivan, 2016).

### 5.3.4 Air cooling

The researcher investigates the relationship between types of air-cooling in summer as risk factors for bronchial asthma**.** The researcher found that who not use any things for air cooling is a risk factor for bronchial-asthma by more than one and half times as OR (CI) = 1.757 (1.109-2.785).

As Green Leaf A/C and Heating, 2017 find that air conditioning, remove allergens from indoor air. The air in most homes is tainted with allergens, especially during the summer time. Most allergens can trigger both allergies and asthma attacks. These asthma attack triggers include pollen from plants, dust mites, mold, and mildew. Air conditioner units have filters that can improve indoor air quality by removing these allergens. Just ensure you replace the air filter of the unit regularly so that it remains functional and efficient(Green Leaf A/C and Heating, 2017). In addition, air conditioning control indoor humidity another way you can use your air conditioning unit to control asthma attacks is by controlling the humidity in your home. High humidity environments are a major trigger for asthma attacks for most children. This is because humidity can trigger the inflammation of the bronchioles. This inflammation partially closes these air pathways, limiting both the inhalation and exhalation of air. An hour or two before your child’s bedtime, you should open your air conditioner to start reducing the humidity in your home. Keep the unit on throughout the night to keep the humidity low. You can also use a dehumidifier to keep indoor air humidity between 30% to 50% (Green Leaf A/C and Heating, 2017)

As our study demonstrate that nothing used for air-cooling at summer is risk factors for bronchial asthma (as Gaza Strip present on Mediterranean Sea which characterized by high humidity in summer) so trying to decrease humidity or indoor pollution by modern air condition or at least fan have benefit for patient with bronchial asthma and in Gaza Strip these advice to work need electricity so electricity is mandatory for patient with bronchial asthma.

### 5.3.5 Tobacco smoking (Cigarette, Arrjila and passive smoking)

In this study, the researcher investigated the effect of cigarette smoking with bronchial asthma and the result of this investigation there is relation between bronchial asthma and cigarette smoking but this relation not reach significant statistically. Most of smoking women deny smoking, as female in this study represent about (34.6%) from all female and just who use it (2%) just leading to underestimation of this risk factors.

The researcher investigated the relation between bronchial asthma and risk of Arrjila smoking. So Arrjila smoking consider as risk factor for bronchial asthma and increase its risk by 74% as OR (CI) = 1.74 (1.148 – 2.643). In conclusion, arrjila is risk factor for bronchial asthma.

The researcher investigated the relationship between bronchial asthma and risk of passive smoking. The result of it, passive smoking has relation to bronchial asthma but not reach statistical significant.

The researcher investigated the relationship between bronchial asthma and risk of exposure to tobacco smoking **(**Cigarette, Arrjila and passive smoking**)**.The study concluded that exposure to tobacco smoking **(**Cigarette, Arrjila and passive smoking**)** have relation to bronchial asthma. This relation is statistically significant and considered as risk factors for b-asthma .In the other word; tobacco smoking is risk factors for bronchial asthma
OR (CI) = 1.6(1.007 - 2.773).

Also, the researcher in this study investigated the relation between bronchial asthma and Tobacco smoking (arrjila, cigarette and passive). The result is there is relation between exposed to all type of tobacco smoke and bronchial asthma and it increase the risk for bronchial asthma by 80% as OR (CI) = 1.807 (1.212 – 2.695).

All the previous results agreement with some study as Piipari et al. 2004 done study on the effect of smoking on adulthood asthma have provided contradictory results. Conducted a population-based incident case-control study to assess the effects of current and past smoking on the development of asthma in adults in southern Finland were recruited. The risk of developing asthma was significantly higher among current smokers. In conclusion, the current results support the hypothesis that smoking causes asthma in adulthood (Piipari, Jaakkola, Jaakkola, & Jaakkola, 2004).

Thomson et al. 2004 demonstrated that most developed countriesy25percentage of adults with asthma are current cigarette smokers. Asthma and active cigarette smoking interact to cause more severe symptoms, accelerated decline in lung function, and impaired short-term therapeutic response to corticosteroids (Thomson et al., 2004).

Piipari et al. 2004 the current results are consistent with the hypothesis that smoking causes asthma in adulthood. The stronger effect of former smoking and the nonlinear relationship with the quantity of smoking seem to point to a behavioral change as a response to beginning symptoms (Piipari et al., 2004).

Jindal 2014, the recent studies from India done on large populations have strongly supported the smoking association of asthma in adults (Jindal, 2014)

Mohammad 2015, the US general surgeon reported demonstrated that exposure to Environmental Tobacco Smoke in early life is causal for wheezing. Although the same re-port indicated that, the role of ETS in asthma a report by International Study of Asthma and Allergies in childhood (ISAAC) published in 2012 suggested that the relationship is causal. The Syrian center of ISAAC reported that exposure to narghileh smoke has stronger association with wheezing than exposure to cigarette ETS (Yousser, 2015).

Tobacco smoking, both active and passive i.e., exposure to environmental tobacco smoke (ETS) has important effects on asthma. Smoking by adults causes bronchial irritation and precipitates acute episodes. Smoking may also increase the disease severity. Continued smoking by adult asthmatics is the likely cause of irreversibility of airway obstruction and development of chronic obstructive pulmonary disease. ETS exposure affects asthma in a similar fashion. Parental smoking is commonly associated with increased asthma symptoms, respiratory infections, acute episodes and frequent hospitalization of children. Bronchial responsiveness and airway sensitization may also increase. Childhood exposure to smoking is also considered as a risk factor for the development of asthma. Similarly, in utero exposure to maternal smoking may be independently responsible for early onset asthma. ETS exposure in adult asthmatics from smoking by spouses, siblings or colleagues is equally troublesome. There is increased morbidity and poorer asthma control. Asthmatic symptoms sharply decline after the ETS exposure is reduced (Jindal & Gupta, 2004).

Exposure to secondhand tobacco smoke (SHS) also known as environmental tobacco smoke (ETS) has been well established scientifically as a human health hazard (Pritsos & Muthumalage, 2015).

In conclusion, in our study, all types of smoking has relation to bronchial asthma specially arrjila smoking. Based on these results, effective health education; awareness to for patient to stop tobacco smoking is mandatory in every visit of patient to clinic and stop cigarette, passive and arrjila smoking to reduce these risks.

Therefore, Health education, health promotion at all level inside clinic and in community through all acclivities and social media to stop smoking to reduce these risk factors for bronchial asthma is mandatory.

### 4.5 Outdoor pollution

#### 5.4.1 Residency nearby Main Street

The researcher examined the relation between residency nearby main street as a risk factor and bronchial asthma , the researcher found no relation between residency nearby main street to bronchial asthma .These result are supported by previous studies that concluded the hazard of residency nearby main street affects on health need special character as in San Francisco, land use guidance suggests a potential hazard exists if average daily traffic exceeds 100,000 vehicles/day within a 200 meter radius of a site, 50,000 vehicles/day within a 50 meter radius or 10,000 vehicles/day on an immediately adjacent street (Bhatia and Rivard, 2008).

Anothor study by Garshick et al., 2003that assessed distance from residential addresses to major roadways using geographic information system ,men living within 50 m of a major roadway were more likely to report persistent wheeze compared with those living more than 400 m away. The risk was observed only for those living within 50 m of heavily trafficked roads (≥10,000 vehicles/24 h (Garshick et al., 2003).

Another study venn et al., 2002 demonstrated an effect is likely to be most marked among those who live within 150 m of a main road, because this is the distance within which concentrations of primary vehicle traffic pollutants are raised above ambient background. Most of the increased risk was localized to within 90 m of the roadside. Living within approximately 90 m of a main road is associated with a proximity-related increase in the risk of wheezing illness in children (Venn et al., 2002).

In Gaza Strip according to, the Ministry of Communications revealed the presence of about 70 thousand vehicles of all types in the Gaza Strip. "This number includes licensed and unlicensed vehicles, as well as 15,000 motorcycles, including 700 licensed motorcycles (iqtisidi , 2016) these interpretative the relation between residency near main street and bronchial asthma.

### 5.4.2 Residency nearby trash containers

The researcher examined the relation between residency nearby trash containers as a risk factors and bronchial asthma , the researcher found there is a relation between residency nearby trash containers relation to bronchial asthma. These results are similar to previous studies that concluded landfills have some adverse environmental impacts including the emission of green house gases (GHGs). GHGs (mainly methane and carbon dioxide) are the main cause of the global warming phenomena that leads to climate change. Climate change may directly affect human health through increase in average temperature. Furthermore, indirect impact on human health may be caused by climatic change through degradation in water and food quality and quantity. According to USEPA, globally, landfills are the third- largest anthropogenic source of methane emissions, while in the US, landfills were the second-largest source in 2006. In Jordan, domestic solid waste sector contributes to about 90% of the methane emissions from anthropogenic sources at the national level. Nowadays, both governmental and nongovernmental agencies have joined efforts with environmentalists to stop landfills from contributing to global warming phenomena. These efforts are focusing on mitigating the impacts of landfill gases (QDAIS, 2008).

The previous study demonstrated the effect of landfill that consisted of many trash containers that had bad smell and many people use fire to clean them that lead to emission of large amount of CO2 and NO2 and others particles so the researcher advices to clean trash container 2 times daily to avoid interaction of materials inside trash containers and bad attitude of people that live near them so also increase awareness of community about how to deal with trash containers.

### 5.4.3 Residency nearby farm

Considering residency near farm and bronchial asthma the researcher found no relation, this result agreement with studies such as a study that demonstrated farmers and agricultural workers have a lower prevalence of asthma than the general population. This may be because of the healthy worker effect in which those who do not tolerate the dusty work conditions leave that occupation. There is a recent body of literature from Europe and Australia that suggests that children growing up on farms have a lower prevalence of asthma, hay fever, respiratory, and allergic, or atopic, diseases compared to children not raised on farms. It is hypothesized that early exposure to antigens in traditional agricultural operations provides life-long protection against the development of allergy Occupational asthma is a form of asthma that occurs to an antigen that is unique or present at higher concentrations in the work place(Kirkhorn et al., 2000).

Another recent study of children suggested that factors encountered in a farm environment might protect against the development of allergy. The protective effect of farming environment in childhood observed in this population-based sample of young adults provides evidence in favor of the hypothesis that environmental factors encountered in childhood may have a lifelong protective effect against the development of allergy (Leynaert et al., 2001).

### 5.4.4 Residency nearby Main corrals

Animal protein, dust and gases are the major sources that irritate or sensitize the respiratory tract. You could also be directly exposed to a chemical substance through skin contact (Roberge, 2008) .

Allergens are found in secretions from saliva, feces, urine and skin of cats (most common), dogs, rabbits, horses, other furry animals and birds. They are commonly found in upholstery, carpets and clothing (Ferrone et al., 2007)**.**

### 5.4.5 Residency nearby professionals and factories

The researcher investigates the relations between residency nearby professionals and bronchial asthma. The researcher found relation between bronchial asthma and living near-by crusher although this relation not reaches significant statistically. Its agreement to study at 2016 that found particle pollution can be very dangerous to breathe. Breathing particle pollution may trigger illness, hospitalization and premature death, risks that are showing up in new studies that validate earlier research. Particle pollution is produced through two separate processes—mechanical and chemical. Mechanical processes break down bigger bits into smaller bits with the material remaining essentially the same, only becoming smaller. Mechanical processes primarily create coarse particles. Dust storms, construction and demolition, mining operations, and agriculture are among the activities that produce coarse particles (Billings et al., 2016).

### 5.5 Others environmental factors

#### 5.5.1 Soil &Cement

Found particle pollution can be very dangerous to breathe. Breathing particle pollution may trigger illness, hospitalization and premature death, risks that are showing up in new studies that validate earlier research. Particle pollution is produced through two separate processes—mechanical and chemical. Mechanical processes break down bigger bits into smaller bits with the material remaining essentially the same, only becoming smaller. Mechanical processes primarily create coarse particles. Dust storms, construction and demolition, mining operations, and agriculture are among the activities that produce coarse particles (Billings et al., 2016).

Industry workers are exposed to asthma- causing substances such as mold, pesticides, soil components, ozone, and cleaning chemicals (SHARP, 2017).

Majority of the molds that grow on damp building materials are found in the soil and are adapted to grow on a wide variety of materials. Outdoors, molds live in the soil, on plants, and on dead or decaying matter (AIHA, 2011).

The study was conducted in a cement factory in the United Arab Emirates to assess cement dust exposure and its relationship to respiratory symptoms among workers. The prevalence of respiratory symptoms was higher among the exposed workers. The few factory workers (19.5%) who used masks all the time had a lower prevalence rate of respiratory symptoms than those not using them. High dust level was the only variable that influenced the workers to use the mask all the time (Ahmed & Abdullah, 2012).

#### 5.5.2 Perfume

The researcher examined the relation between perfume as a risk factors and bronchial asthma, the researcher found the perfume has relation to bronchial asthma and considered as a risk factors for bronchial asthma. It increase the risk of bronchial asthma by 82% as OR (CI) = 1.824 (1.379 – 2.413). This result is similar to previous studies that concluded that there is a positive association between exposure to perfume and bronchial asthma as

Ferrone et al., 2007 that found and concluded, used of perfume have relation to bronchial asthma and trigger asthma (Ferrone et al., 2007) .

Also in 2016 a study found that perfume is one of bronchial asthma triggers (National Asthma Council Australia, 2016).

### 5.5.3 Exhausts burn of wood and plastic

The researcher examined the relation between exposure to exhausts burn of wood and plastic as a risk factor and bronchial asthma, the researcher found the exposure to exhausts burn of wood and plastic have relation to bronchial asthma.

This result is similar to previous studies as one of them done by asthma and Allergy foundatio of America, 2015 about fireplaces and tobacco products. It found smoke from wood-burning fireplaces; wood stoves, pellets and "clean burning" stoves can pollute your indoor air. The smoke can contain fine particles that irritate your airways. It also contains nitrogen dioxide and carbon monoxide. These are odorless gasses known to cause swelling in the airways in sensitive groups, like those with asthma. It is best to avoid using the fireplace to heat your living room. Gas and kerosene space heaters release nitrogen dioxide too. If they are not vented, these will pollute your air too. In fact, children who were around gas heaters as babies are more likely to develop asthma (Asthma and Allergy foundatio of America, 2015a).

So counseling for community to decrease exposure to sources of $NO_2$ and $NO_2$ by increase health education about the sources of $NO_2$ & $CO_2$ and how to avoid them.

### 5.5.4 Home activities (Household– cleaning products)

The researcher examined the relation between household– cleaning products as a risk factors and bronchial asthma , the researcher find the household– cleaning products

Has relation to bronchial asthma .These result are similar to previous studies that concluded that there is a positive association between exposure to cleaning substance and bronchial asthma. As study did by Sock et al 2007 and Concluded frequent use of common household cleaning sprays may be an important risk factor for adult asthma Within the follow-up of the European Community Respiratory Health Survey in 10 countries, we identified 3,503 persons doing the cleaning in their homes (Zock et al., 2007) also the same Ferrone et al., 2007 study concluded the chemical cleaning product that used in home have relation to bronchial asthma and trigger asthma (Ferrone et al., 2007).

### 5.5.5 MOLD

The researcher examined the relation between exposure to mold as a risk factors and bronchial asthma; the researcher found the exposure to mold inside the home has relation to bronchial asthma. These result are similar to previous studies that concluded that exposure to mould or dampness in indoor environments is associated with bronchial asthma for both development and exacerbation (Palaty & Shum, 2012).

Another study with objective to investigate whether reported mould or dampness exposure in early life is associated with the development of allergic disorders in children from eight European birth cohorts. The result of that study, exposure to visible mould and/or dampness during first 2 years of life was associated with an increased risk of developing asthma.These findings suggest that a mouldy home environment in early life is associated with an increased risk of asthma (Tischer et al., 2011).

### 5.5.6 Dust inside the home

The researcher examined the relation between exposure to dust as a risk factors and bronchial asthma. The researcher found that exposed to dust inside the home has relation to bronchial asthma and considered as a risk factors for bronchial asthma .the dust increase the risk for bronchial asthma about 37% as OR (CI) = 1.37(1.055 – 1.796).

These results are similar to previous studies that conclude dust that found almost everywhere indoors. It is airborne, but settles on surfaces such as open shelving, window ledges, curtains, blinds, upholstery, carpets and can be carried in many ways, including: human hair, animal dander, dead insect bodies, food scraps, chalk dust and talcum powder and trigger bronchial asthma (Ferrone et al., 2007).

Another study that demonstrated that children who are exposed to higher levels of dust are more likely to have allergies and asthma. This is because allergic triggers of dust. For people with asthma, these microscopic inhaled substances commonly cause allergic reactions in the respiratory system, which leads to nasal and airway inflammation. The more inflammation in the airways, the more noticeable your asthma symptoms (Asthma.ca, 2016).

Around 30 to 40 percent of the contaminant inside your home is brought in from outdoors. They can enter your home on your shoes, clothing, or your pets can track them in on their paws and fur. Not surprisingly, the greatest concentration of household dust is found in carpeting near the entryway surfaced walkway, such as a paved sidewalk, leading to the front door of your home(Turner et al., 2010).

### 5.5.7 Upper Respiratory Tract Infection (URTI).

The researcher examined the relation between exposed to upper respiratory tract infections as a risk factors and bronchial asthma, the researcher find the URTI has relation to bronchial asthma and considered as a risk factors for bronchial asthma by two fold OR (CI) = 2.029 (1.564 – 2.631).

This result is similar to previous studies that concluded, respiratory infections include the common cold, the flu, pneumonia and other infections. These common illnesses can affect your lungs when you have asthma. They can cause inflammation (swelling) and narrowing of your airways. These changes could trigger asthma symptoms (an asthma episode or an asthma attack) (Asthma andAllergy foundatio of America 2015).

Many studies one of them by Global Initiative for Asthma, 2017 that find upper respiratory symptoms frequently precede the onset of an asthma exacerbation, indicating the important role of viral URTI in precipitating exacerbations in many, although not all, children with asthma (Global Initiative for Asthma, 2017b).

Upper respiratory tract infections (URTI) were seen as a risk factor when at least three of the following five components are marked positive: sore throat, fever, runny/blocked nose, muscle aches and exposure to other people with a cold (Sanya, Kirenga, Worodria, & Okot-nwang, 2014)

### 5.5.8 GastroEsophageal Reflux Disease (GERD)

The reserchar examind the relation between bronchial asthma and gasrtroesophagal reflux (GERD) and the researcher found positive relatin between gasrtroesophagal reflux and bronchial asthma .

Asthma and GERD are common conditions that often coexist with 50-80% of asthmatics having GERD and up to 75% having abnormal pH testing. However, only 30% of patients who have both GERD and asthma will have GERD as the cause for their asthma. The causal relationship between asthma and GERD is difficult to establish because either condition can induce the other (GERD causing asthma as above, and asthma causing increased reflux by creating negative intrathoracic pressure and overcoming LES barrier) (Standiford et al., 2013).

Another study found 82% of adult asthmatics had abnormal gastroesophageal reflux. Compared with the controls, asthmatics had significantly lower LES pressure, greater acid exposure time, more frequency reflux episodes, and longer clearance times in both the upright and supine positions.GERD may induce or aggravate asthma. A cross-sectional international population survey in 2,661 participants showed that, compared with those without GERD, individuals with GERD had increase risk of pulmonary conditions like wheezing, breathlessness at rest and nocturnal breathlessness. In this study, association of GERD with physician-diagnosed asthma was marginally significant (Huang & Jiang, 2005).

So effective management for GERD lead to decrease exposure to this factor so can break the relation between bronchial asthma and GERD.

**5.5.9 Psychological problem (depression, grief stress)**

Regarding relation between psychological problem (depression , grief and stress) and bronchial asthma , the researcher found the psychological problem (depression , grief and stress) have relation to bronchial asthma. From the previous psychological problem, the researcher found the stress is considered as risk factor for bronchial asthma and it is increase the risk by 48% as OR(CI) = (1.480 (1.019-2.150) .

These results are similar to previous studies that one of them concluded there is growing awareness of the correlation between psychological factors, the course of asthma, and the outcomes of asthma treatment and the implications of this correlation are still poorly understood. Recent studies suggest an interaction between behavioral, neural, endocrine, and immune processes and suggest that psychological factors play an active role in the genesis of asthma, even though it is generally accepted that anxiety and depression are more common in asthmatic patients and that there is a close correlation between psychological disorders and asthma outcomes, such as poorer control of asthma symptoms, the implications and practical consequences of this link remain weak. New studies are introducing an intriguing model of the links between emotional stress, brain centers, the immune system, and the hypothalamus-pituitary-adrenal axis that is far removed from the original concept of 'asthma nervosa' (Di Marco et al., 2011).

Also another study found that current smoking, anxiety and mood disorders alone are associated with poorer asthma control (Ouellet et al., 2012) .

Next study illustrated the relationship between depression and asthma may involve more than one causal pathway and includes the possibility that depression can lead to a sense of hopelessness that erodes adherence and other health- promoting behavior, or that depression impacts asthma directly by altering the immune system. An assessment of the interplay between risk behavior, depression, adherence, and asthma can add important new information to our understanding about how to identify and treat those at greatest risk for poorly controlled disease and asthma-related death (Bender, 2006)**.**

For young people with chronic illness, risk behavior and depression are also common. There is some evidence that both depression and risk behavior are associated with non-adherence to medications, poor treatment outcomes, and death. The relationship between depression and asthma may involve more than one causal pathway and includes the possibility that depression can lead to a sense of hopelessness that erodes adherence and other health- promoting behavior, or that depression impacts asthma directly by altering the immune system (Bender, 2006)**.**

Another study even though it is generally accepted that anxiety and depression are more common in asthmatic patients and that there is a close correlation between psychological disorders and asthma outcomes, such as poorer control of asthma symptoms, the implications and practical consequences of this link remain weak. New studies are introducing an intriguing model of the links between emotional stress, brain centers, the immune system, and the hypothalamus-pituitary-adrenal axis that is far removed from the original concept of 'asthma nervosa (Di Marco et al., 2011). In conclusion, psychological problem (depression, grief and stress) should be controlled to decrease bronchial asthma exacerbations by mental health programs integration management.

### 5.5.10 Food allergy

The researcher investigated the relationship between bronchial asthma and food allergy . The study concluded that food allergy is a risk factors for bronchial asthma by three fold as OR (CI) = 3.112 (1.541- 6.286) similar to previous studies that concluded food allergy is a trigger for asthma symptoms (<2% of people with asthma). In patients with confirmed food- induced allergic reactions (anaphylaxis), co-existing asthma is a strong risk factor for more severe and even fatal reactions. Food-induced anaphylaxis often presents as life-threatening asthma (Global Initiative for Asthma, 2017b) .

Also National Asthma Conuncil Australia at 2016 demonstrated that some dietary can trigger exacerbating bronchial asthma (National Asthma Council Australia, 2016).

Children with allergy to food have a four –fold increase likelihood of having asthma compared with children without food allergy (GINA, 2015).

United States population-level association between FA and severe asthma exacerbations was also identified (Liu et al., 2010).

As there are many phenotypes of asthma one of them is allergic asthma and one is characterized by some association with is allergy to food (GINA, 2015)**.**

GINA, 2015 confirmed food allergy is a risk factor for asthma related deaths (GINA, 2015).

So counseling and health education for patient with bronchial asthma to avoid food that is confirmed to cause allergy to them is better reduce bronchial asthma exacerbation.

### 5.5.11 Drug allergy

About drug allergy the study concluded that it is risk factors for bronchial asthma by fold five folds as OR (CI) = 5.299 ( 2.338-12.009) .This result supported by many study, one of them done by Saudi thorasic scociaty at 2016 concluded ,about 10–20% of adults with asthma suffer from exacerbations in response to some drugs as baby aspirin or NSAIDs (Saudi Thorasic scociaty, 2016).

Non steroidal anti-inflammatory drugs (NSAIDs) either orally ,ingested, rectally inserted or injected NSAID received by participants was seen as a trigger/risk factor for acute asthma exacerbations (Geyser & Rheeder, 2008).

So avoidance strategies must be discussed with each patient especially with drugs

### 5.5.12 Physical activity - Exercise

Considering exercise the study concluded that there is positive relation between no exercise and bronchial asthma, as they may be avoid exercise because of (exercises induced asthma) .Similar study illustrated this result, in 2012, adults with current asthma had a significantly lower prevalence of participating in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise during the past month compared to adults who never had asthma (77.9%) and adults with former asthma (75.3%) (Forrest & Dudley, 2013).

Another study at 2012 noticed that EIA might be observed in children and adolescents of different physical conditioning backgrounds, from those not engaged in sports to competitive athletes. Physical exercise causes broncho constriction in the majority (~70%) of the children and adolescents who present asthma; nevertheless, EIA may occur in those who do not present asthma diagnosis (Oxford Health Plans, 2012).

People with asthma can participate in physical activity. For adults or children involved in competitive sport, prescribers need to check which asthma medicines are permitted in the sport.Exercise-induced broncho constriction can be managed effectively with relievers and preventers (or both) and should not stop people with asthma participating in physical activity (National Asthma Council Australia, 2015).

In conclusion, the persons with bronchial asthma need counseling and health promotion to be active and encourage them to do exercise with good preparation and special instruction before exercise to avoid exercise-induced asthma.

**5.5.13 BMI-Obesity**

The researcher investigated the relationship between bronchial asthma and obesity by use of BMI. The study concluded that there is no relation between obesity and bronchial asthma and this result can be is controversial to most studies like a study that demonstrated the prevalence of asthma is higher in obese individuals. Recently, two major phenotypes of asthma with obesity have been described: one phenotype of early-onset asthma that is aggravated by obesity, and a second phenotype of later-onset asthma that predominantly affects women. Systemic inflammation and mechanical effect, both due to the expansion of the adipose tissue, have been proposed as the main reasons for the association between obesity and asthma (Gomez-Llorente et al., 2017)**.**

This can be explained by the majority of our sample 56% are males (65.4%) so the difference between male and female relationship with bronchial asthma found in some global studies and one of them at 2004 done in New York State, USA to find relation between asthma and obesity. It concludes, men and women differ significantly in the association between BMI and asthma prevalence only with respect to the lowest weight category. While women had a monotonic association, men showed a U-shaped relationship, indicating that both extremes of weight are associated with a higher prevalence of asthma (Luder et al., 2004).

### 5.5.14 Family history

Considering, relation between family history and bronchial asthma, the researcher found that family history has a relation to bronchial asthma and considered as a risk factor for bronchial asthma. Family history increase the risk for bronchial asthma nearby fourfold as OR (CI) = 3.702 (2.071 -6.619).

These result are similar to previous studies that concluded if you have a parent with asthma, you are 3-6 times more likely to develop asthma than who doesn't (MINNESOTA DEPARTMENT OF HEALTH ASTHMA PROGRAM, 2007) .

Another study that demonstrated that genetics plays an important role in the development of asthma and allergy likely through several genes of moderate effect (i.e., genes associated with relative risks in the range of 1.2–2) Genome-wide linkage studies and case–control studies have identified 18 genomic regions and more than 100 genes associated with allergy and asthma in 11 different populations. In particular, there are consistently replicated regions on the long arms of chromosomes 2, 5, 6, 12 and 13. (Subbarao, Mandhane, and Sears 2009).

Recent genome-wide association study identified a new gene, ORMDL3, that exhibited a highly significantly association with asthma for single nucleotide polymorphism rs8067378, odds ratio 1.84, 95% confidence interval 1.43–2.42) a finding that has now been replicated in several populations. Extensive heterogeneity in the genetic basis of asthma, and in gene-by-environment interactions, is likely. Failure to identify and precisely quantify environmental exposures and their timing may account for some of the difficulty that researchers have had in replicating genetic associations (Subbarao, Mandhane, and Sears 2009).

### 5.5.15 Seasonal variation

The researcher examined the effect of seasonal variation (weather changes) on bronchial asthma, the researcher found that seasonal variation (weather changes**)** has a relation and considered  as a risk factor for bronchial asthma, but each weather have different risk .In summer the risk for bronchial asthma increase more than fourfold as OR (CI) = 4.135 (2.743-6.231), then autumn OR (CI) = 3.946 (3.065 – 5.079) after that spring OR (CI) = 3.544 (2.718 – 4.619) finally winter OR (CI) = 3217 (2.326 - 4.530).

On otherwise the most sever effect is winter (39.9%) then spring (27%) after that autumn (15.2%) and least sever summer (14.6%).

Summer is a time for outdoor activities that may trigger asthma. Mold spores may also be high in hot humid weather,  humid air is heavier and harder to breathe and carry harbors fungus, molds and dust mites that trigger asthma (Bottrell, 2011).

Outdoor moulds are usually at their peak in late summer and early fall, but are present whenever there is no snow (Ferrone et al., 2007).

Seasonal allergens. These primarily include pollens. Tree pollens are most abundant during the spring, grasses in the spring and summer, and weed pollens, such as ragweed, in late summer and fall. Seasonal allergens vary with geographic regions(Oxford Health Plans, 2012).

These results are similar to previous studies that concluded seasonal trends in asthma related hospitalizations are widely recognized; the percents of the total admissions per month were compared. peak in asthma-related hospitalizations occurred in the winter months (10.3%) and a nadir in the summer months (5.9%; $p < 0.004$) with similar trends for ICU admissions (Pendergraft et al., 2005).

Another study at 2016 find by sectional surveys of people aged ≥18 years in winter and summer in the inner-city areas of Zunyi city, Guizhou Province, China. There was significant difference in asthma prevalence among adult respondents between winter (1.8%) and summer (0.8%) in inner city Zunyi. Asthma and asthma-related symptoms occurred more frequently in winter than summer (Jie et al., 2016).

Also another to quantify the number of asthma attacks treated in the emergency room of a public hospital and to study seasonal fluctuations, taking into consideration the local climate, which is characterized by having only two seasons: a rainy/humid season and a dry season. Among the respiratory conditions treated, asthma (24.4%) was the second most common diagnosis. Most of the asthma consultations (56.6%) involved children below the age of fifteen. The correlation between climate and asthma was most significant in relation to changes in humidity, although the effect was delayed by one month the conclusion of this study visits to the emergency room for the treatment of asthma attacks were more frequent during the rainy season, increasing at one to two months after the annual increase in humidity and decreasing in the dry season (VALENÇA et al., 2006).

Also, National Asthma Conuncil Australia at 2016 found thunderstorms are associated with epidemics of asthma flare-ups (National Asthma Council Australia, 2016).

That means our community in Gaza Strip specially who have bronchial asthma need special health education programs for all seasonal variations (weathers) specially at winter as it has the highest effect on bronchial asthma because it has many risks such as recurrent upper respiratory infections, heating type (coal and wool combustion) increase, increase mould in the home and bad sanitation and ventilation, that means increase indoor air pollution increase especially in winter so in conclusion health education before winter is mandatory as it has multiple risks factors .

Considering summer and spring our result supported by many studies, one of them by NationalAsthmaConuncilAustralia, 2016 that demonstrated that exposure to pollen can worsen asthma symptoms during the pollen seasons. Pollen counts are generally highest on calm, hot, sunny days in spring, early summer or during the dry season in tropical regions (National Asthma Council Australia, 2016).

Another study in New York City study was done and concluded that tree pollen peaking in mid-spring exhibit substantive impacts on allergy, and asthma exacerbations, particularly in children (Ito et al., 2015).

## Chapter (6)

## Conclusion and Recommendations

### 6.1 Conclusion

As some patients, continue to experience exacerbations even with maximal doses of current treatment. Having even one exacerbation increases the risk that a patient will have another within the next 12 months. There is increasing research interest in identifying at-risk patients, and in investigating new strategies to further reduce exacerbation risk. In clinical practice, exacerbation risk can be reduced both by optimizing asthma medications, and by identifying and treating modifiable risk factors. Not all risk factors require or respond to a step up in controller treatment. Both pharmacological and non-pharmacological therapies and strategies are important in asthma management (Global Initiative for Asthma, 2017).

This study is carried out in an attempt to identify the risk factors for bronchial asthma demographic, socioeconomic, and environmental and others in the Gaza Strip. The researcher used case-control study and was conducted in sixth UNRWA clinics, and included 214 cases and 214 controls.

Through understanding bronchial asthma risk factors, the study seek to suggest developing preventive strategies towards reducing burden of asthma.

The study demonstrated that many grateful results and important things is determination of risk factors for bronchial asthma in Gaza Strip as follows, low level of education, not use things for heating in winter, using nothing for cooling home at summer, tobacco smoking, Arrjila smoking, upper respiratory tract infection, exposure to stress, perfume and dust inside home, food allergy, drug allergy, family history and seasonal variation.

The researcher also found many positive and important relation between bronchial asthma and the following, family size more than 8 members, low income less than1000 INS, not working, passive smoker, use of mat to cover land, painting home with polygad, home size less than 140m2, house consist of 2 rooms or less, living near trash container or corrals and crusher, also exposure for soil, cement, exhausts burn of wood and plastic, derivatives of petroleum, chemical cleaning substance, frying of oil, entrance of kitchen at time of cooking, mold inside home, gastro esophageal reflux, no exercises psychological condition as depression and grief.

Through understanding bronchial asthma risk factors and other relations that explored in the study, the researcher suggests developing preventive strategies to reduce the burden of bronchial asthma and to improve the health situation in Gaza Strip.

## 6.2 Recommendation

**Recommendations for decision makers and management in order to standardize bronchial asthma care**

1-Put bronchial asthma as priority in front of health decisions maker as other chronic diseases as hypertension and diabetes mellitus.

2-Designated special file for bronchial-asthma follow up.

3-Palestinian health care system need to evaluate the offered bronchial -asthma services and to identify gap in services.

4-Put standardized protocols for follow up bronchial asthma and identify risk factors for every patient.

5-CONTROL-BASED ASTHMA MANAGEMENT; In control-based asthma management, pharmacological and non-pharmacological treatment is adjusted in a continuous cycle that involves assessment, treatment and review (Global Initiative for Asthma, 2017).

6- Initiated an annual World Asthma Day to raising awareness about the burden of asthma, and becoming a focus for local and national activities to educate families and health care professionals about effective methods to manage and control asthma.

**General recommendations to reduce risk factors and factors that have relation to bronchial asthma:**

1- Stop smoking
2- Improve housing condition
3- Adopting health promotion as general strategy
4- There is need to develop community-based activities to increase health awareness about bronchial asthma
5- Vaccination for influenza virus
6- Treat co-morbid conditions

7- Avoid exposures to allergens and pollutants or irritants that make asthma worse

**Recommendations at the level of health centre**

The long-term goals of asthma management are to achieve good symptom control, and to minimize future risk of exacerbations, and side-effects of treatment. The patient's own goals regarding their asthma and its treatment should also be identified.

1-Effective asthma management requires a partnership between the person with asthma and their health care providers.

2-Teaching communication skills to health care providers may lead to increased patient satisfaction, better health outcomes, and reduced use of health care resources.

3 –Increase patient's ability to obtain, process and understand basic health information to make appropriate health decisions.

4-Recommend measures to control exposures to allergens and pollutants or irritants that make asthma worse.

**Recommendations for patient with bronchial-asthma to improve health promotion and health massages:**

**Control Environmental Factors and Co morbid Conditions**:

**Tips to Controlling Dust in your Home**:

1-Place a mat right outside the front door to brush off heavy dirt from your shoes and/or place a washable mat just inside the door and wipe shoes on that mat (wash weekly)

2-Make sure to remove shoes and other footwear at the door

3-Remove outdoor clothing and put on 'at home' wear as soon as you get home; make sure to leave outdoor clothing in closet with door closed or launder them immediately

4-If possible, remove all wall-to-wall carpeting and replace with tile, linoleum or wood flooring

5-Remove all heavy fabric drapery and replace with washable curtains or, better still, a washable roll-down blind

6-Change furnace filters regularly and, if you have a forced gas furnace, place a filter over the bedroom vents

7-Remove all clutter (e.g. ornaments, mobiles, silk flowers, etc) to reduce the number of places that dust can collect

8-Store books in cabinets with doors closed(Asthma.ca, 2016)

9-Choose leather or vinyl furniture over fabric pieces.

10-Wash throw blankets weekly in 130°F (about 55 C°) hot water.

11-If you cannot remove the carpet; cleaned by vacuum daily if possible.

**Smoking**

1-Quit tobacco Smoking (cigarette, arrjila, environmental tobacco smoke (Secondhand Smoke)

2- At every visit, strongly encourage people with asthma who smoke to quit. .

3-Build access for smoking, ETS counseling, and smoking cessation programs.

**Reducing your family's exposure to secondhand smoke**

1-you can quit smoking yourself, if you are a smoker.

2-Ask guests to not smoke in or near your house or car.

3-Until you quit, do not smoke around your families, in your home or in your car.

4-Strongly encourage people with asthma to avoid environmental smoke exposure

**2- URTI**

1-Get plenty of rest, eat healthily, and exercise regularly.

2-Avoid being around people who are sick, and wash your hands often.

3-Get flu shot once a year in the fall,

4-Keep your breathing equipment clean. This includes your asthma inhaler, asthma nebulizer, and nebulizer tubing and mouthpiece.

5-Do not share your breathing equipment or medicines with others.

6-Wash hands often with soap and warm water, particularly after coughing or sneezing. If you don't have access to running water, use an alcohol-based hand cleanser.

7-Don't touch your eyes, nose or mouth. This is how germs often spread.

8-Cough or sneeze into your inner elbow when you don't have a tissue.

9-If you get the flu, stay home from work or school.

10-There are antiviral drugs approved to treat flu.

**3-Mold**

Measures that may reduce mould exposure include:

1 - Removing visible mould by cleaning with bleach or other mould reduction cleaners. If you are sensitive to strong odors, wear a mask or ask someone else to do this for you.

2 -using high-efficiency air filters – these may be integrated in air-conditioning, heat-recovery ventilation systems, or in stand-alone air purifiers

3- Ensuring adequate natural ventilation including the use of extractor fans

4- Sealing leaks in bathrooms and roofs

5- Clearing overflowing gutters and blocked under floor vents

6-Removing indoor pot plants (which promote mould growth)

7-Drying or removing wet carpets

8-Treating rising damp as soon as it is detected

**Heating**

1-Use modern air conditioning.

2-Don't use wood burning stoves, fireplaces, or kerosene heaters at home.

3-Encourage people with asthma to use non-polluting heating.

**Weather**

A sudden blast of cold air, excess heat and humidity, and dry climates can all trigger asthma symptoms.

*In the cold:

1-Dress appropriately.

2-Wear a scarf (or a special asthma mask) over your nose and mouth to keep your breath warm and moist.

3- Breathe in through your nose to warm the air.

* In heat and humidity:

1-Use air conditioning in your home and in your car.

2-Drink plenty of fluid.

**Emotional stress**

1-Try to stay calm and relaxed.

2- Take deep breaths.

3- Consciously slow your breathing by counting while you breathe in and out.

4- Do any activity that distracts and relaxes you.

5- Relaxations techniques and breathing exercises may be helpful.

**Exercise**

Exercise — a common trigger — can bring on coughing, wheezing, or shortness of breath. Still, when your symptoms are well controlled, exercise is good for your lungs. It's one trigger you should NOT avoid.

A-patient with bronchial asthma should be able to be active without symptoms.

Recommend that reduce EIA following strategies:

1- Take medication 5 to 10 minutes before you begin exercise.

2-Warm up for 10 minutes before exercise.

3- Get some aerobic exercise every day.

4- Encourage people with asthma to engage in regular physical activity for its general health benefits

5-Provide advice about prevention and management of exercise induced broncho constriction

6- Regular physical activity improves cardiopulmonary fitness, but confers no other specific benefit on lung function or asthma symptoms, with the exception of swimming in young people with asthma

7-With proper management, you can enjoy exercise and achieve your full potential.

**Foods and food additives**

1- Food avoidance should not be recommended unless an allergy or food chemical sensitivity has been clearly demonstrated.

2- For confirmed food allergy, food allergen avoidance may reduce asthma exacerbations

3- Encourage patients with asthma to consume a diet high in fruit and vegetables for its general health benefits

**Drug allergy**

1-Consult with your healthcare provider or pharmacist before you takes medications other than the ones that have been prescribed for your asthma.
2-Avoidance of medications that may make asthma worse
3- Always ask about asthma before prescribing NSAIDs, and advise patients to stop using them if asthma worsens
4- Always ask people with asthma about concomitant medications
5-Aspirin and NSAIDs (non-steroidal anti-inflammatory drugs) are not generally contraindicated unless there is a history of previous reactions to these agents (Global Initiative for Asthma, 2017a)

**GERD**

Be questioned about symptoms of GERD. If symptoms are present, a trial of anti-GERD measures (including a proton pump inhibitor).

## References

Abed, Y., Mumcuoglu, K. Y., Armenios, B., Shaheen, S., Jacobs, J., Bar-Sela, S., & Richter, E. (1994). Asthma in Gaza refugee camp children and its relationship with house dust mites. *Annals of Allergy*, *72*(2), 163–166. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/8109806

Ahmed, H. O., & Abdullah, A. A. (2012). Dust Exposure and Respiratory Symptoms among Cement Factory Workers in the United Arab Emirates. Retrieved May 17, 2017, from https://www.ncbi.nlm.nih.gov/pubmed/22453209

AIHA. (2011). Facts about Mold. Retrieved September 3, 2017, from https://www.aiha.org/get-involved/VolunteerGroups/Documents/BiosafetyVG-FactsAbout MoldDecember2011.pdf

Akinbami, L. J., Moorman, J. E., & Liu, X. (2011). Asthma prevalence, health care use, and mortality: United States, 2005-2009. Retrieved December 26, 2017, from http://www.cdc.gov/nchs

Alavinezhad, A., & Boskabady, M. H. (2017). The prevalence of asthma and related symptoms in Middle East countries. *The Clinical Respiratory Journal*, (April). Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/crj.12655/epdf

Al Zabadi, H., & El Sharif, N. (2009). Risk factors for asthma severity among emergency rooms attendees, Palestine. *Pulmonary Pharmacology and Therapeutics*, *22*(3), 208–213. https://doi.org/10.1016/j.pupt.2008.12.016

Asthma.ca. (2016). Dust control in your home. Retrieved December 29, 2017, from https://asthma.ca/wp-content/uploads/2017/08/asthma_dust_eng.pdf

Asthma and Allergy foundatio of America. (2015a). Breathe Easier Improving Indoor Air Quality in Your Living Room _ Asthma and Allergy Foundation of America. Retrieved September 12, 2017, from https://community.aafa.org/blog/breathe-easier-improving-indoor-air-quality-in-your-living-room

Asthma and Allergy foundatio of America. (2015b). Tobacco Smoke and Asthma. Retrieved October 1, 2017, from http://www.aafa.org/page/secondhand-smoke-environmental-tobacco-asthma.aspx

Astma- og Allergiforbundet. (2007). Useful facts on asthma (astma) - NAAF. Retrieved October 9, 2017, from http://www.naaf.no/astma/fakta_om_astma/

Beasley, R., Semprini, A., & Mitchell, E. A. (2015). Risk factors for asthma: is prevention possible? *The Lancet*, *386*, 1075–1085. https://doi.org/10.1016/S0140-6736(15)00156-7

Beasley, R., Semprrin, A., & Mitchell, E. (2015). Risk factors for asthma: is prevention possible؟. *Lancet*, *386*, 1075–85. https://doi.org/10.1016/S0140-6736(15)00156-7

Belanger, K., & Triche, E. W. (2008). Indoor Combustion and Asthma. *Immunology and Allergy Clinics of North America*, *28*(3), 507–519. https://doi.org/https://doi.org/doi:10.1016/j.iac.2008.03.011

Ben Abdallah, F. C., Taktak, S., Chtourou, A., Mahouachi, R., & Kheder, A. Ben. (2011). Burden of Chronic Respiratory Diseases (CRD) in Middle East and North Africa (MENA). *World Allergy Organization Journal.*, *4*(1), S6-8. https://doi.org/10.1097/WOX.0b013e3182093cbb

Bender, B. G. (2006). Risk Taking , Depression , Adherence , and Symptom Control in Adolescents and Young Adults with Asthma. *AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE*, *173*(9), 953–957. https://doi.org/10.1164/rccm.200511-1706PP

beyondblue and Asthma Australia. (2015). anxiety & depression Asthma ,. Retrieved July 16, 2017, from https://www.google.ps/search?q=Asthma%2C+anxiety+%26+depression+Anxiety&oq=Asthma%2C+anxiety+%26+depression+Anxiety&aqs=chrome..69i57.2601j0j8&sourceid=chrome&ie=UTF-8

Billings, P. G., Nolen, J. E., Tran, T. A. V., Alexander, L. M., Bender, L. K., Edelman, N., … Tubbs, G. (2016). STATE OF THE AIR 2016. Retrieved August 26, 2017, from http://www.lung.org/local-content/california/documents/state-of-the-air/2016/sota-2016-full-report.pdf

Bottrell, J. (2011). Here's Why Humidity and Cold Air Trigger Asthma. Retrieved November 13, 2017, from https://www.healthcentral.com/article/heres-why-humidity-and-cold-air-trigger-asthma

Braman, S. S. (2006). The burden of asthma. *Chest*, *130*(1), 326–328. https://doi.org/10.1378/chest.130.1

Brauer, M., Hystad, P., & Reynolds, C. (2012). Environmental Guidelines for Urban and Rural Land Development in British Columbia. Supporting Information -- Air Quality. Retrieved March 15, 2017, from
http://www.env.gov.bc.ca/wld/documents/bmp/devwithcare2012/DWC-Air-Quality.pdf

Busse, W. W., Lemanske Jr., R. F., & Gern, J. E. (2010). The Role of Viral Respiratory Infections in Asthma and Asthma Exacerbations. *Lancet*, *376*(9743), 826–834. https://doi.org/10.1016/S0140-6736(10)61380-3.

D'Amato, G., Liccardi, G., D'Amato, M., & Cazzola, M. (2002). Outdoor air pollution, climatic changes and allergic bronchial asthma. *European Respiratory Journal*, *20*(3), 763–776. https://doi.org/10.1183/09031936.02.00401402

Di Marco, F., Santus, P., & Centanni, S. (2011). Anxiety and depression in asthma. *Current Opinion in Pulmonary Medicine*, *7*(39–44), 13–18. https://doi.org/10.1097/MCP.0b013e328341005f.

El-Sharif, N., Abdeen, Z., Qasrawi, R., Moens, G., & Nemery, B. (2002). Asthma prevalence in children living in villages, cities and refugee camps in Palestine. *European Respiratory Journal*, *19*(6), 1026–1034. https://doi.org/10.1183/09031936.02.01832001

Ferrone, M., MacPherson, A., Wilton, Mas. P., & Woychyshyn, J. (2007). All About Asthma Triggers. Retrieved July 22, 2017, from https://www.saswh.ca/.../All_About_Asthma_Triggers_singlesFinal2A...%0A

Fiori, N. S., Gonçalves, H., Dumith, S. C., Cesar, M. A. D. C., Menezes, A. M. B., Macedo, S. E. C., & 1. (2012). Ten-year trends in prevalence of asthma in adults in southern Brazil : comparison of two population-based studies. *Cadernos de Saude Publica*, *28*(1), 135–144. https://doi.org/http://dx.doi.org/10.1590/S0102-311X2012000100014

Forrest, J., & Dudley, J. (2013). Burden of Asthma in Florida. Retrieved February 6, 2017, from http://www.floridahealth.gov/diseases-and-conditions/asthma/_documents/asthma-burden2013.pdf

Garshick, E., Laden, F., Hart, J. E., & Caron, A. (2003). Residence Near a Major Road and Respiratory Symptoms in U.S. Veterans. *Epidemiology*, *2814*(6), 728–736. https://doi.org/10.1037/a0013262.Open

Geyser, M., & Rheeder, P. (2008). Risk factors precipitating exacerbations in adult asthma patients presenting at Kalafong Hospital , Pretoria. *South African Family Practice Journal*, *50*(4), 67. https://doi.org/https://doi.org/10.1080/20786204.2008.10873743

GINA. (2015). Global Strategy for Asthma management and prevention. *Global Initiative for Asthma*. Retrieved from http://www.ginasthma.org/local/uploads/files/GINA_Report_2015.pdf

Global Asthma Network. (2014). The Global Asthma Report 2014. Retrieved March 20, 2017, from www.globalasthmanetwork.org

Global Initiative for Asthma. (2017a). Global Strategy For Asthma Management and Prevention. https://doi.org/10.1183/09031936.00138707

Global Initiative for Asthma. (2017b). GLOBAL STRATEGY FOR ASTHMA MANAGEMENT AND PREVENTION. *Global Initiative for Asthma*.

GlobalAsthmaNetwork. (2014). The Global Asthma Report Asthma Repotr. Retrieved March 20, 2017, from www.globalasthmanetwork.org

Gomez-Llorente, M., Romero, R., Chueca, N., Martinez-Cañavate, A., & Gomez-Llorente, C. (2017). Obesity and Asthma: A Missing Link. *International Journal of Molecular Sciences*, *18*(7), 1490. https://doi.org/10.3390/ijms18071490

Grant, E. N., Lyttle, C. S., & Weiss, K. B. (2000). The relation of socioeconomic factors and racial/ethnic differences in US asthma mortality. *American Journal of Public Health*, *90*(12), 1923–1925. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1446442/

Green Leaf A/C and Heating. (2017). How to Control Asthma at Night with Air Conditioning. Retrieved October 3, 2017, from https://www.greenleafhvac.net/how-to-control-asthma-at-night-with-air-conditioning/

Hassan, M. R., Kabir, L., Mahmud, A. M., Rahman, F., & Hossain, M. A. (2002). Self-reported asthma symptoms in children and adults of Bangladesh : findings of the National Asthma Prevalence Study. *International Epidemiological Association*, *31*, 483–488. Retrieved from http://www.japi.org/march_2014_special_issue/07_effects_of_smoking.pdf

Hjern, A., Rasmussen, F., & Bra, L. (2005). Social class in asthma and allergic rhinitis: a national cohort study over three decades. *EUROPEAN RESPIRATORY JOURNAL*, *26*(6), 1064–1068. https://doi.org/10.1183/09031936.05.00022105

Hroob, A. M., Nawafleh, H., Altaif, K. I., & Elfaqir, S. (2015). Population Prevalence of Asthma in Ma ' an Governorate , Jordan. *Health Science Journal*, *10*(21), 1–4. Retrieved from http://www.hsj.gr/medicine/population-prevalence-of-asthma-in-maan-governorate-jordan.php?aid=8704

Huang, W., & Jiang, P. (2005). Role of gastroesophageal reflux disease in asthmatic patients. Retrieved February 20, 2017, from www.europeanreview.org/wp/wp-content/uploads/308.pdf

Ito, K., Weinberger, K. R., Robinson, G. S., Sheffield, P. E., Lall, R., Mathes, R., … Matte, T. D. (2015). The associations between daily spring pollen counts , over-the-counter allergy medication sales , and asthma syndrome emergency department visits in New York City , 2002-2012. *Environmental Health*, 1–12. https://doi.org/10.1186/s12940-015-0057-0

J., W., K., E., G., S., & D., N. (2016). Exacerbation of asthma among adults in relation to the home environment in multi-family buildings in Sweden. Retrieved from http://ovidsp.ovid.com/ovidweb.cgi?T=JS&CSC=Y&NEWS=N&PAGE=fulltext&D=emex&AN=614843724

Jie, Y., Kebing, L., Yin, T., & Jie, X. (2016). Prevalence of Asthma and Asthma-Related Symptoms Among Adults Exposed to Indoor Environmental Risk Factors : a Comparison between Winter and Summer in Zunyi , China. *Pol. J. Environ. Stud*, *25*(2), 621–633. https://doi.org/10.15244/pjoes/60491

Jindal, S. K. (2014). Effects of Smoking on Asthma. *JOURNAL OF THE ASSOCIATION OF PHYSICIANS OF INDIA*, *62*(march), 32–37. Retrieved from http://www.japi.org/march_2014_special_issue/07_effects_of_smoking.pdf

Jindal, S. K., & Gupta, D. (2004). The relationship between tobacco smoke & bronchial asthma. - *Indian Journal of Medical Research*, *120*(5), 443–453. Retrieved from http://medind.nic.in/iby/t04/i11/ibyt04i11p443.pdf

Kaelin, M. A., & Bayona, M. (2004). Case – Control Study. *The Young Epidemiology Scholars Program (YES)*. Retrieved from www.collegeboard.com/prod_downloads/.../4297_MODULE-06.pdf%0A

Karmaus, W., & Botezan, C. (2002). Does a higher number of siblings protect against the development of allergy and asthma? A review. *Epidemiol Community Health*, *56*(3), 209–217. https://doi.org/10.1136/jech.56.3.209

Kim, H. Y., Kwon, E. B., Baek, J. H., Shin, Y. H., Yum, H. Y., Jee, H. M., … Han, M. Y. (2013). Prevalence and comorbidity of allergic diseases in preschool children. *Korean J Pediatr*, *56*(8), 338–342. https://doi.org/10.3345/kjp.2013.56.8.338

Kirkhorn, S. R., Clinical, A., & Resources, O. H. (2000). AGRICULTURAL RESPIRATORY HAZARDS AND DISEASE. Retrieved December 28, 2016, from http://worh.org/files/AgHealth/resp.pdf

Lara, J., Akinbami, M. D., Cheryl, D., & Fryar, M. S. P. H. (2016). Current Asthma Prevalence by Weight Status Among Adults: United States, 2001–2014. *NCHS Data Brief.*, *239*, 1–8. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/27019018

Lewallen, S., & Courtright, P. (1998). Epidemiology in practice: Case-control studies. *Community Eye Health Journal*, *11*(28), 57–58. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1706071/

Leynaert, B., Neukirch, C., Jarvis, D., Chinn, S., & Burney, P. (2001). Does Living on a Farm during Childhood Protect against Asthma , Allergic Rhinitis , and Atopy in Adulthood ?, *164*, 1829–1834. https://doi.org/10.1164/rccm2103137

Lieshout, R. J. Van, & Macqueen, G. (2008). Psychological Factors in Asthma. *Allergy, Asthma, and Clinical Immunology*, *4*(1), 12–28. https://doi.org/10.2310/7480.2008.00002

Lin, W., Brunekreef, B., & Gehring, U. (2013). Meta-analysis of the effects of indoor nitrogen dioxide and gas cooking on asthma and wheeze in children, (2), 1724–1737. https://doi.org/10.1093/ije/dyt150

Litonjua, A. A., Carey, V. J., Weiss, S. T., & Gold, D. R. (1999). Race , Socioeconomic Factors , and Area of Residence Are Associated With Asthma Prevalence. *Pediatric Pulmonology*, *28*(6), 394–401. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/10587412

Litonjua, A. A., & Weiss, S. T. (2016). Risk factors for asthma. *UpToDate*. Retrieved from https://www.uptodate.com/contents/risk-factors-for-asthma

Liu, A. H., Jaramillo, R., Sicherer, S. H., Wood, R. A., Bock, S. A., Burks, A. W., … Zeldin, D. C. (2010). National prevalence and risk factors for food allergy and relationship to asthma: Results from the National Health and Nutrition Examination Survey 2005-2006. *Journal of Allergy and Clinical Immunology*, *126*(4), 798–806.e14. https://doi.org/10.1016/j.jaci.2010.07.026

Luder, E., Ehrlich, R. I., Lou, W. Y. W., Melnik, T. A., & Kattan, M. (2004). Body mass index and the risk of asthma in adults. *Elsevier Ltd.*, 29–37. https://doi.org/10.1016/j.rmed.2003.08.004

Mahboub, B., Shendi, F., Safarini, B., AbdulAziz, M., Mustafa, G., & Prakash, V. (2013). Cost of Asthma in Dubai, United Arab Emirates (UAE). *Journal of Pulmonary & Respiratory Medicine*, *3*(2), 2–5. https://doi.org/10.4172/2161-105X.1000146

Maziak, W., Ward, K. D., Afifi Soweid, R. A., & Eissenberg, T. E. (2004). Tobacco smoking using a waterpipe: A re-emerging strain in a global epidemic. *Tobacco Control*, *13*(4), 327–333. https://doi.org/10.1136/tc.2004.008169

McNeill, A. (2011). TOBACCO USE AND EFFECTS ON HEALTH. *Basic Cardiac Electrophysilogy*, *2*, 4–8. https://doi.org/10.1007/b11990

Michailopoulos, P., Almaliotis, D., Georgiadou, I., Papakosta, D., Gougoulias, K., Giouleka, P., … Karampatakis, V. (2017). Allergic Conjunctivitis in Patients with

Respiratory Allergic Symptoms; a Retrospective Study in Greece. *Medical Hypothesis, Discovery and Innovation in Ophthalmology*, *6*(1), 3–9.

Minawi, W. (2005). *Prevalence of Asthma and Allergy and Their Risk Factors Among An – Najah National University Students - Nablus - Palestine*. Najah National University Students, Nablus - Palestine.

Ministry of Communications. (2016). Number of vehicl in Gaza Strip. Retrieved April 4, 2017, from http://www.palsawa.com/news/2016/03/27/reports/63628.html

MINNESOTA DEPARTMENT OF HEALTH ASTHMA PROGRAM. (2007). Asthma and family health history. Retrieved December 20, 2017, from http://www.health.state.mn.us/asthma/documents/familyhistory.pdf

Modig, L., Torén, K., Janson, C., Jarvholm, B., & Forsberg, B. (2009). Vehicle exhaust outside the home and onset of asthma among adults. *European Respiratory Journal*, *33*(6), 1261–1267. https://doi.org/10.1183/09031936.00101108

Mohammad, Y., ShaabanR1, Hassan, M., Yassine, F., Mohammad, S., Tessier, J., & Ellwood, P. (2015). Respiratory effects in children from passive smoking of cigarettes and narghile ISAAC Phase Three in Syria. *The International Journal of Tuberculosis and Lung Disease*, *18*(11), 1279–84. https://doi.org/doi: 10.5588/ijtld.13.0912.

National Asthma Council Australia. (2015). Australian Asthma Handbook – Quick Reference Guide. *Australian Asthma Handbook*, 44. https://doi.org/10.1016/B978-155860916-7/50017-8

National Asthma Council Australia. (2016). *AUSTRALIAN ASTHMA HANDBOOK CLINICAL ISSUES TRIGGES,Version 1.2*. Retrieved from https://www.asthmahandbook.org.au/uploads/5804261b0ca75.pdf

NGUYEN, Kimber., ZAHRAN, H., IQBAL, S., PENG, J., & BOULAy, E. (2011). Factors Associated with Asthma Control among Adults in Five New England States , 2006 – 2007. *Journal of Asthma, Early Online*, (13), 1–8. https://doi.org/10.3109/02770903.2011.576744

Nunes, C., Pereira, A. M., & Morais-Almeida, M. (2017). Asthma costs and social impact. *Asthma Research and Practice*, *3*(1), 1. https://doi.org/10.1186/s40733-016-0029-3

Nutrition and Health Department Nestlé Research Center Lausanne Switzerland. (2015). Atopic dermatitis: Global epidemiology and risk factors. Retrieved September 13, 2017, from http://www.karger.ch/journals/anm/anm_jh.htm%5Cnhttp://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=emed18a&NEWS=N&AN=604238349

OCHA. (2016). The Gaza Strip: The humanitarian impact of the blockade. Retrieved from https://www.ochaopt.org/documents/ocha_opt_gaza_blockade_factsheet_july_2015_english.pdf

Oliveira, D., Boff, R., Luiz, J., & Schaefer, A. (2015). Allergology International Indicators of violence and asthma : An ecological study. *Allergology International*, *64*, 344–350. https://doi.org/10.1016/j.alit.2015.04.003

OSHA. (2006). Preventing Mold-Related Problems in the Indoor Workplace. Retrieved January 17, 2017, from https://www.osha.gov/Publications/preventing_mold.pdf

Ouellet, K., Boudreau, M., Plourde, A., Bacon, S. L., & Lavoie, K. L. (2012). Individual and combined impact of cigarette smoking, anxiety, and mood disorders on asthma control. *Nicotine & Tobacco Research, 14*(8), 961–9. https://doi.org/10.1093/ntr/ntr315.

Oxford Health Plans. (2012). Understanding asthma. Retrieved January 2, 2017, from https://www.oxhp.com/secure/materials/member/asthma.pdf

Palaty, C., & Shum, M. (2012). Health Effects from Mould Exposure or Dampness in Indoor Environments. Retrieved September 10, 2017, from www.ncceh.ca/sites/.../files/Mould_and_Health_Effects_Jul_2012.pdf%0A

Pendergraft, T., Stanford, R., Beasley, R., Stempel, D., & McLaughlin, T. (2005). Seasonal variation in asthma-related hospital and intensive care unit admissions. Retrieved March 6, 2017, from https://www.ncbi.nlm.nih.gov/pubmed/16032935

Peter, A., Anderson, J., Menkedick R., J., & Wooton A., M. (2005). Healthy Homes Issues : Carbon Monoxide. Retrieved August 17, 2017, from https://www.hud.gov/sites/documents/DOC_12481.PDF

Piipari, R., Jaakkola, J. J. K., Jaakkola, N., & Jaakkola, M. S. (2004). Smoking and asthma in adults. *European Respiratory Journal*, 734–739. https://doi.org/10.1183/09031936.04.00116903

Pinto Pereira, L. M., Jackman, J., Figaro, N., Babootee, N., Cudjoe, G., Farrell, S., … Bekele, I. (2010). Health burden of co-morbid asthma and allergic rhinitis in West Indian children. *Allergologia et Immunopathologia*, *38*(3), 129–134. https://doi.org/10.1016/j.aller.2009.09.002

Pritsos, C. A., & Muthumalage, T. (2015). The impact of commonly used air filters in eliminating the exposure to secondhand smoke constituents. *Environ Sci Process Impacts.*, *17*(3), 543–51. https://doi.org/10.1039/c4em00479e.

QDAIS, H. A. (2008). Global warming potential of solid waste landfills. In *The 3rd* *centeal and Estren Europian Conferance on Health and Environment* (Vol. 14, pp. 5–116). Cluj-Napoca,Romania. Retrieved from www.omfi.hu/cejoem/Volume14/Vol14No1/CE08_1.pdf

Radon, K., Büsching, K., Heinrich, J., Wichmann, H., Jörres, R. A., Magnussen, H., & Nowak, D. (2002). Passive Smoking Exposure : A Risk Factor for Chronic Bronchitis and Asthma in Adults ? *Chest*, *122*(3), 1086–1090. https://doi.org/10.1378/chest.122.3.1086

Roberge, B. (2008). Animal health prevention fact sheet. Retrieved March 20, 2017, from https://www.irsst.qc.ca/media/documents/PubIRSST/RF-536.pdf

Sahni, S., Khanijo, S., Talwar, B., & Talwar, A. (2017). Socioeconomic status and its relationship to chronic respiratory disease. *Socioeconomic Status and Its Relationship to Chronic Respiratory Disease*. https://doi.org/10.5603/ARM.2017.0016

Sanya, R. E., Kirenga, B. J., Worodria, W., & Okot-nwang, M. (2014). Risk factors for asthma exacerbation in patients presenting to an emergency unit of a national referral hospital in Kampala , Uganda . *African Journals Online*, *14*(3), 707–715. Retrieved from doi:http://dx.doi.org/10.4314/ahs.v14i3.29

Saudi Thorasic scociaty. (2016). The Saudi Initiative for Asthma : Guidelines for the diagnosis and management of asthma in adults and children. Retrieved July 7, 2017, from http://saudithoracic.com/download/SINA Guidelines 2016.pdf

Sauerhoff, M. W. (2008). Carpet , Asthma and Allergies – Myth or Reality. Retrieved January 1, 2017, from http://referralfw.com/resource/pdf/Carpet, Asthma and Allergies Myth or Reality  - Mitchell W Sauerhoff Ph.D..pdf

SHARP. (2017). Marijuana and Work-Related Asthma. https://doi.org/http://dx.doi.org/10.1016/j.anai.2015.01.004.

Standiford, C. J., Grant M Greenberg, M., & Harrison, R. Van. (2013). Gastroesophageal Reflux Disease ( GERD ) Patient population. Retrieved from http://www.med.umich.edu/1info/fhp/practiceguides/gerd/gerd.12.pdf

Strachan, D. P. (2000). Family size , infection and atopy : the first decade of the “ hygiene hypothesis .” *Thorax*, *55*(Suppl 1), 2–11. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1765943/pdf/v055p000S2.pdf

Subbarao, P., Mandhane, P., & Sears, M. (2009a). Asthma : epidemiology , etiology and risk factors. *Journal, Canadian Medical Association*, *181*(9). https://doi.org/10.1503/cmaj.080612

Subbarao, P., Mandhane, P., & Sears, M. (2009b). Asthma : epidemiology , etiology and risk factors. *Canadian Medical Association Journal*, *181*(9). https://doi.org/10.1503/cmaj.080612

Thomas, M., & Griffith, C. (2005). Editorials Asthma and Panic Scope for Intervention ? *AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE*, *171*, 1197–1198. https://doi.org/10.1164/rccm.2503005

Thomson, N. C., Chaudhuri, R., & Livingston, E. (2004). Asthma and cigarette smoking. *European Respiratory Journal*, *24*(i), 822–833. https://doi.org/10.1183/09031936.04.00039004

Tischer, C. G., Hohmann, C., Thiering, E., Herbarth, O., Mu, A., Henderson, J., … Kuehni, C. E. (2011). Meta-analysis of mould and dampness exposure on asthma and allergy in eight European birth cohorts : an ENRIECO initiative. *Allergy*, *12*, 1570–1579. https://doi.org/10.1111/j.1398-9995.2011.02712.x

Toyoshima, M., Chida, K., Suda, T., & Sato, M. (2011). Possible relationship between asbestos exposure and bronchial asthma. https://doi.org/10.1164/ajrccm.183.11.1571a

Trembath, F. (2015). Animal Exposure , Asthma and Allergies. Retrieved March 3, 2017, from https://habricentral.org/resources/45851

Turner, P. R., Gibson, S. M. S., & Reed, A. L. (2010). *leave it at the Door*. *The University of Georgia, Cooperative Extension, Colleges of Agricultural and Environmental Sciences and Family and Consumer Sciences*. Retrieved from http://www.fcs.uga.edu/docs/Leave_it_at_door_HACE-E-81.pdf

UNDP. (2014). The Gaza Strip - Facts , Figures and UNDP 's Response to the Ongoing Crisis. Retrieved April 4, 2017, from http://www.ps.undp.org/content/dam/papp/docs/Publications/UNDP-papp-focus-crisis-GazaFF082014.pdf

VALENÇA, L., RESTIVO, P., & NUNES, M. (2006). Seasonal variations in emergency room visits for asthma attacks in Gama, Brazil. Retrieved August 9, 2017, from https://www.ncbi.nlm.nih.gov/pubmed/17268726

Venn, A. J., Lewis, S. A., Cooper, M., Hubbard, R., & Britton, J. (2002). Living near a main road and the risk of wheezing illness in children. *American Journal of Respiratory and Critical Care Medicine*, *164*(12), 2177–2180. https://doi.org/10.1164/ajrccm.164.12.2106126

Wark, P. A. B., Bucchieri, F., Johnston, S. L., Gibson, P. G., Hamilton, L., Mimica, J., … Davies, D. E. (2007). IFN-γ-induced protein 10 is a novel biomarker of rhinovirus-induced asthma exacerbations. *Journal of Allergy and Clinical Immunology*, *120*(3), 586–593. https://doi.org/10.1016/j.jaci.2007.04.046

Watson, S., Henline, D., & Sullivan. (2016). How to Limit Cold Weather Induced Asthma. Retrieved October 10, 2017, from https://www.healthline.com/health/asthma/cold-induced-asthma#3

WHO. (2000). Air Quality Guidelines - Second Edition.Chapter 7.1 Nitrogen dioxide. Retrieved May 26, 2017, from http://www.euro.who.int/__data/assets/pdf_file/0017/123083/AQG2ndEd_7_1nitrogendioxide.pdf

Yasuda, H., Suzuki, T., Zayasu, K., Ishizuka, S., Kubo, H., Sasaki, T., … Yamaya, M. (2005). Inflammatory and bronchospastic factors in asthma exacerbations caused by upper respiratory tract infections. Retrieved April 20, 2017, from https://www.ncbi.nlm.nih.gov/pubmed/16141679

Yousser, M. (2015). Passive smoking interference with wheezing and asthma : short review of current knowledge. *Herbert Open Access Journals*, *3*. https://doi.org/10.7243/2053-6739-3-2

Zheng, T., Niu, S., Lu, B., Fan, X., Sun, F., Wang, J., … Leaderer, B. (2002). Childhood Asthma in Beijing , China : A Population-based Case-Control Study. *American Journal of Epidemiology*, *156*(10), 977–983. https://doi.org/10.1093/aje/kwf127

Ziad M El-Zaatari, Hassan A Chami, G. S. Z. (2015). Health effects associated with waterpipe smoking. *Tobacco Control*, *24*, 131–143. https://doi.org/10.1136/tobaccocontrol-2014-051908

Zock, J. P., Plana, E., Jarvis, D., Ant, J. M., Kromhout, H., Kennedy, S. M., … Seaton, D. (2007). The use of household cleaning sprays and adult asthma: An international longitudinal study. *American Journal of Respiratory and Critical Care Medicine*, *176*(8), 735–741. https://doi.org/10.1164/rccm.200612-1793OC

# Annexes

## Annex (1) sample size calculation

### Sample size results

Assumptions:

| | | |
|---|---|---|
| Odds ratio | = | 2 |
| Exposed controls | = | 15% |
| Alpha risk | = | 5% |
| Power | = | 80% |
| Controls / Case ratio | = | 1 |

Total exposed = 20.5435%

Estimated sample size:

| | | |
|---|---|---|
| Number of cases | = | 208 |
| Number of controls | = | 208 |

Total = 416

Annex (2) questionnaire in Arabic language:

بسم الله الرحمن الرحيم

سعياً إلى مجتمع صحي ومعافى نضع بين يديك استبانه تتضمن عددا من العبارات التي تصف العوامل التي تساهم في ظهور مرض ( الربو). فأرجو منك الإجابة عليها بدقة

| | |
|---|---|
| **Control ( ) 1** | **Case ( )** |

| | | |
|---|---|---|
| **2- العمر....................** | **3-الجنس :** □ذكر □ أنثى | **4-عدد أفراد العائلة في المنزل: ..............** |
| **5-المنطقة : □ الشمال□ غزة □ الوسطى□ خان يونس□ رفح** | | **6-الحالة الاجتماعية: □غير متزوج □ متزوج** |
| **7-الدخل الشهري......................** | | |

## السكن بالقرب

| **8-السكن بالقرب في محيط** | **لا** | **نعم** | **200م** | **200l م-500م** | **500م -1000م** | **أكثر من 1000م** |
|---|---|---|---|---|---|---|
| 1. محطة كهرباء | | | | | | |
| 2. مولدات كهرباء-كبيرة | | | | | | |
| 3. مولدات كهرباء صغيرة | | | | | | |
| 4. مزارع (بيارات) حمضيات | | | | | | |
| 5. (بيارات) مزارع - زيتون | | | | | | |
| 6. مزارع- مزروعات موسمية | | | | | | |
| 7. كروم - عنب | | | | | | |
| 8. مكب نفايات | | | | | | |
| 9. شوارع رئيسة | | | | | | |
| 10. مصانع البطون ولبللوك | | | | | | |
| 11. كسارة | | | | | | |
| 12. حظائر لتربية الدواجن | | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 13. حظائر لتربية-حمام | | | | | | |
| 14. حظائر لتربية-بط | | | | | | |
| 15. حظائر لتربية-أغنام | | | | | | |
| 16. حظائر لتربية الأبقار | | | | | | |
| 17حضائر لتربية حيوانات | | | | | | |
| 18. منجرة | | | | | | |
| 19. محل للحدادة | | | | | | |
| 20. كراج سيارات | | | | | | |
| 21. تصنيع عطور | | | | | | |
| 22. فرن | | | | | | |

## نوع السكن

**9. نـــوع السكـــــــــن:** □بطون□الأسبست□كونتينر□زينكو

**10. سقف المنــــزل:** □بطون□اسبست□اسبست + بطانة□زينكو□زينكو + بطانة □كرميد□كرميد + بطانة

**11. الأرضيـــــــــــة:** □بلاط□كراميكا□مدة باطون□رمل□أخرى حدد.................

**12. غطاء الأرضية:** □ سجاد□موكيت□حصيرة□لا شيء□أخرى حدد.................

**13. دهان المنـــــزل:** □زيت□عادي□جبس□أخرى حدد.................

| | | |
|---|---|---|
| **14. مساحة المنزل: ................** | **15. عدد الغرف: ....................** | **16. عدد الأفراد في المنزل:.........** |

**17. مساحة غرفة النوم:** □2X2□3X2 □4X3 □أخرى حدد.................

**18. عدد الأفراد داخل الغرفة التي تنام فيها:** □1-2□3-4 □5-6□أخرى حدد.................

**19. المستوى التعليمي:** □أمي□ابتدائي□إعدادي□ثانوي □دبلوم □جامعي فما فوق

**20العمل:** □يعمل□لا يعمل

| | |
|---|---|
| **21. مكان الطبخ:**□داخل المنزل □خارج المنزل | **22. نوع وقود الطبخ:** □كهرباء □غاز□حطب □فحم |

**23. نوع التدفئـــة:** □كهرباء □غاز□حرق حطب □حرق فحم

**24. التبريد:** □مكيف □مروحة □لاشيء

| | |
|---|---|
| **25. هل يوجد طيور داخل المنزل:** □نعم □لا | **26. هل يوجد حيوانات داخل المنزل:** □نعم □لا |

## التدخين

| | |
|---|---|
| **27. هل أنت مدخن: □نعم□لا** | **إذا كنت مدخن سجائر كم عدد السجائر يوميا: .........................** |
| **28.هل أنت مدخن أرجيلة: □نعم□لا** | **إذا كانت الإجابة بنعم: برجاء حدد عدد المرات يومياً: ..................** |

| | | |
|---|---|---|
| **29. عدد سنوات التدخين: ......** | **30. هل يوجد مدخن في البيت:□نعم□ لا** | **31. عدد المدخنين في البيت:.......** |

## التعرض للحساسية

32- هل يوجد تعرض للعوامل التالية المختلفة وفي حالة التعرض و التأثير اختر نوع التأثير وضع رقمه في المربع الأخير=نوع التأثير :**حساسية جلدية( 1)ضيق النفس (2)حساسية للأنف-عاطس ( 3)حساسية للعيون ( 4)**

| | **يوجد تعرض وتأثر شديد** | **يوجد تعرض و تأثر متوسط** | **يوجد تعرض و تأثر خفيف** | **تعرض ولا يوجد تأثر** | **لا يوجد تعرض** | **نوع التأثير محدد بالرقم** |
|---|---|---|---|---|---|---|
| 1-التربة | | | | | | |
| 2-الاسمنت | | | | | | |
| 3-العطور | | | | | | |
| 4-عوادم المركبات التي تعمل بالوقود | | | | | | |
| 5-أبخرة حرق المواد الخشبية | | | | | | |
| 6- أبخرة حرق المواد البلاستيكية | | | | | | |
| 7-المنظفات –الكيميائية | | | | | | |
| 8- الغبار داخل المنزل | | | | | | |
| 9- العفن علي جدران المنزل | | | | | | |
| 10- مشتقات الوقود – البترول | | | | | | |
| 11-غليان زيت الطبخ- القلي | | | | | | |
| 12-دخولك مكان إعداد | | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| الطعام | | | | | | |
| 13-فصل الشتاء | | | | | | |
| 14- فصل الصيف | | | | | | |
| 15- فصل الخريف | | | | | | |
| 16- فصل الربيع | | | | | | |
| 17-الإحباط - لم تحقق أحلامك أو ما تصبو إليه | | | | | | |
| 18- الحزن والكآبة | | | | | | |
| 19- التوتر-ضغوط الحياة | | | | | | |
| 20- التهابات الجهاز التنفسي العلوي | | | | | | |
| 21-الارتجاع | | | | | | |

| |
|---|
| **33. هل يوجد حساسية للطعام :** □نعم□لا |
| **إذا كان نعم فما هي الأنواع:** □الشكولاته□البندورة □المانجا□المعلبات□فواكه□خضار □مجمدات□اللحوم |
| □البهارات□المشروبات الغازية □أخرى : .................... |
| **34. هل يوجد حساسية للأدوية:** □نعم□لا |
| **إذا كان نعم فما هي الأنواع:** □NSAID □Aspirin□ Paracetmol □أخرى: ........................... |

| | |
|---|---|
| **35. هل تمارس الرياضة:** □نعم□لا | |
| **إذا كان نعم فما مدة النشاط: .............** | **عدد مرات ممارسة الرياضة أسبوعياً: .................** |
| **نوع النشاط الرياضي:** □المشي□الجري □السباحة □كرة القدم □كمال أجسام □لياقة بدينة□أخرى : ............. | |
| □البهارات□المشروبات الغازية □أخرى : .................... | |

| | |
|---|---|
| 37.0 الوزن: ................ | 36. الطول: ............ |
| 38- هل يوجد من العائلة احد لديه حالة الربو :□نعم□لا | |

**Annex (3) questionnaire in English language:**

**Part( 1)**

| | |
|---|---|
| **1-Control ( )** | **Case ( )** |
| **2-Age** | **3-gender** |
| **4-Number of family** | **5-Governorate north** □ North □ Gaza □Middle □ Khanuonis □ Rafah |
| **6-Marital status** □married □ not married | **7-Income** |

**Part (2)**

| 8-Residency near by | yes | no | 200m | 201M 500M | 501M-1000M | More than 1000M |
|---|---|---|---|---|---|---|
| 1-Electricity station | | | | | | |
| 2- Big generators | | | | | | |
| 3-Small generators | | | | | | |
| 4-Farmer citrus | | | | | | |
| 5- Farmer olive | | | | | | |
| 6- Seasonal crops | | | | | | |
| 7 Farmer grape | | | | | | |
| 8-Trash container | | | | | | |
| 9-Main street | | | | | | |
| 10-Concrete | | | | | | |
| 11-Crusher | | | | | | |
| 12-Corrals-chicken | | | | | | |
| 13-Corrals-dove | | | | | | |
| 14 -Corrals-ducks | | | | | | |
| 15-Corrals-sheep | | | | | | |
| 16-Corrals-cow | | | | | | |
| 17corrals-other animals | | | | | | |
| 18-Carpentry | | | | | | |
| 19-Metal work | | | | | | |
| 20-Mechanical for car | | | | | | |
| 21-Manufacturing perfume | | | | | | |
| 22-oven | | | | | | |

**Part (3) House character**

| | | |
|---|---|---|
| 9-Type of house : ☐ cement☐ Aspast ☐container ☐ zinco | | |
| 10- House roof : ☐cement ☐aspast ☐ aspast + linear ☐zinco<br>☐ zinco+ linear ☐ carmaid ☐ carmid + linear | | |
| 11- House land (floor ): ☐ flagstone ☐kramica ☐batoon ☐sand ☐ Others | | |
| 12- land cover: ☐carpet ☐mokait ☐mat ☐others | | |
| 13- house painting: ☐oil ☐ polygad ☐gypsum ☐others | | |
| 16- Number of persons inside house:------- | 15 Number of room:<br>-------------------- | 14-House size:<br>------------------- |
| Size for bed room ☐2X2 ☐2*3 ☐3X 4 ☐ others | | |
| 18- Number of persons inside bed room:<br>☐1 -2 ☐ 3-4 ☐5 – 6 ☐others ---------------- | | |
| 19-level of education ☐Illiteracy ☐Primary ☐Preparatory ☐secondary ☐Diploma ☐University and above | | |
| 20- Work ☐Yes ☐ No | | |
| 22-Kind of fuel for cooking<br>☐Electricity ☐ Gaze ☐ wood | 21-Site of cooking<br>☐ inside home ☐outside home | |
| 23- type of heating ☐ Electricity ☐ Gaze ☐ Wood ☐ Coal | | |
| 24 - type of cooling ☐ Air condition ☐ Fan ☐ Nothing | | |
| 26-Animal inside home ☐ NO ☐ Yes | 25- Bird inside home ☐NO ☐Yes | |

## Part (4)Smoking

| | |
|---|---|
| 27-Are you smoking ☐ Yes☐ No<br>Cigarettes smoking<br>☐ Yes☐ No if yes<br>Number of cigarettes per day:<br>Number of years of smoking: | |
| 28-Are you \|Arjilla smoking<br>☐ Yes☐ No if yes Number o arjilla smoking per week | |
| 30-Are there any body smoke in home<br>☐ Yes☐ No | 31-Number of smoker inside home<br>--------------------------- |

**Part ( 5 ) exposure and degree of effect with type of effects to some elements**

**If you exposed to following , what will happen, then determine type of effect if present**

**1-shortness of breath 2 allergic rhinitis 3 dermatitis 4 conjunctivitis**

| | **Not exposed** | **Exposed with NO Effect** | **Exposed with mild effect** | **Exposed With moderate effect** | **Exposed with sever effect** | **Type of effect** |
|---|---|---|---|---|---|---|
| 1-soil | | | | | | |
| 2-Cement | | | | | | |
| 3-Perfume | | | | | | |
| 4-Exhaust of vehicles | | | | | | |
| 5-Exhaust of wood | | | | | | |
| 6-Exhaust of plastic | | | | | | |
| 7-Chemical cleaning substances | | | | | | |
| 7-Dust inside home | | | | | | |
| 8-Mold | | | | | | |
| 9-Petroleum | | | | | | |
| 10-Oil | | | | | | |
| 11-Entrance site of cooking | | | | | | |
| 12-Winter | | | | | | |
| 13- Summer | | | | | | |
| 14-Autumn | | | | | | |
| 15-Spring | | | | | | |
| 16-depression | | | | | | |
| 17-Grief | | | | | | |
| 18-Stress | | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 19-Upper respiratory tract infection | | | | | | |
| 20-Gastro oesophageal reflux | | | | | | |

**Part (6)**

| | |
|---|---|
| 33 - Food allergy □Yes □No | |
| If yes , what types :□chocolate □ tomato □mango □canned food □fruit□ vegetable □freezer □meat□spices □soft drink □others | |
| 34 Drug allergy □Yes □ No<br>Kind of drug allergy: □Paracetmool □Aspirin □ NSAID □ Others | |
| 35- Exercise □Yes □No<br>If yes -- duration of exercise<br>- Type of exercise<br>□Walking □ Running □Swimming □Football □Body building □ Fitnes | |
| -37 Weight | 36 Tall |
| 38--Family history □ Yes □ No | |

**Annex ( 4 ) Names of experts**

Dr Basssam Abo Hamad, Dr Yahya Abed, Dr Kitam Abo Hamad, Dr Ahmad Al shargawi, Dr Mohamad Allolo, Dr Maher Al-Biomee, Dr Yosef Awad and others

## Annex (5) Palestinian Health Research Council

المجلس الفلسطيني للبحث الصحي

**Palestinian Health Research Council**

تعزيز النظام الصحي الفلسطيني من خلال مأسسة استخدام المعلومات البحثية في صنع القرار

**Developing the Palestinian health system through institutionalizing the use of information in decision making**

### Helsinki Committee

**For Ethical Approval**

**Date: 01/08/2016** **Number:** PHRC/HC/152/16

**Name:** WAEL OM MATAR **الاسم:** وائل عمر مطر

We would like to inform you that the committee had discussed the proposal of your study about:

نفيدكم علماً بأن اللجنة قد ناقشت مقترح دراستكم حول:

**Risk Factors for Bronchial Asthma among adults in Gaza: Case Control Study**

The committee has decided to approve the above mentioned research. Approval number PHRC/HC/152/16 in its meeting on 01/08/2016

و قد قررت الموافقة على البحث المذكور عاليه بالرقم والتاريخ المذكوران عاليه

**Signature**

**Member** **Member**

1/8/2016

**Chairman**

2016

**Genral Conditions:-**

1. Valid for 2 years from the date of approval.
2. It is necessary to notify the committee of any change in the approved study protocol.
3. The committee appreciates receiving a copy of your final research when completed.

**Specific Conditions:-**

1/8/16

**E-Mail:pal.phrc@gmail.com**

**Gaza - Palestine** غزة - فلسطين

شارع النصر - مفترق العيون

**Annex(6) Relationship between cases and controls by percentage (percentage) according to distance between residence and electricity Generators and Ovens**.

| Distance By Meter | Control | | | | Case | | | | X2 | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|
| | **200 and less** | **200-500** | **501-1000** | **>1000** | **200 and less** | **200-500** | **501-1000** | **>1000** | | |
| Electricity Station | 16 | 12.0 | 28.0 | 44.0 | 11.1 | 16.7 | 27.8 | 44.4 | 0.343 | 0.952 |
| Big Generators | 40.3 | 35.8 | 11.9 | 11.9 | 40.8 | 42.9 | 6.1 | 10.2 | 1.449 | 0.694 |
| Small Generator | 63.5 | 31.8 | 4.7 | 0.0 | 65.1 | 27.0 | 6.3 | 1.6 | 1.822 | 0.610 |
| Oven | 35.9 | 35.9 | 15.4 | 12.8 | 36.8 | 39.5 | 15.8 | 7.9 | 0.522 | 0.914 |

**Annex (7) Relationship between cases and controls by percentage (%) according to distance between residence and main street**

| Distance By Meter | Control | | | | Case | | | | X2 | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|
| | **200 and less** | **200-500** | **501-1000** | **>1000** | **200 and less** | **200-500** | **501-1000** | **>1000** | | |
| **Main Streets** | 56.7 | 28.4 | 8.5 | 6.4 | 54.0 | 34.1 | 5.6 | 6.3 | 1.618 | 0.655 |

**Annex (8) Relationship between cases and controls by percentage (%) according to distance between residence and trash containers**

| Distance By Meter | Control | | | | Case | | | | X2 | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|
| | 200 and less | 201-500 | 501-1000 | >1000 | 200 and less | 200-500 | 501-1000 | >1000 | | |
| **Trash-container** | 44.0 | 18.0 | 22.0 | 16.0 | 53.1 | 23.5 | 6.2 | 17.3 | 7.319 | 0.062 |

**Annex (9) Relationship between cases and controls by percentage (%) according to distance between residence and farms**

| Distance By Meter | Control | | | | Case | | | | X2 | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|
| | 200 and less | 200-500 | 501-1000 | >1000 | 200 and less | 200-500 | 501-1000 | >1000 | | |
| **Citrus** | 45.3 | 32.1 | 17.0 | 5.7 | 50.0 | 21.7 | 13.0 | 15.2 | 3.559 | 0.313 |
| **Olive** | 51.5 | 29.4 | 14.7 | 4.4 | 50.9 | 29.8 | 14.0 | 5.3 | 0.060 | 0.996 |
| **Seasonal crops** | 38.5 | 38.5 | 15.4 | 7.7 | 50.0 | 26.5 | 14.7 | 8.8 | 1.380 | 0.710 |
| **Grape** | 35.7 | 12.5 | 25.0 | 25.0 | 58.3 | 25.0 | 4.2 | 12.5 | 6.658 | 0.084 |

**Annex (10) Relationship between cases and controls by percentage (%) according to distance between residence and Corrals**

| Animals | Control | | | | Case | | | | X2 | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|
| | 200 and less | 200-500 | 501-1000 | >1000 | 200 and less | 200-500 | 501-1000 | >1000 | | |
| **Chicken** | 43.3 | 40.3 | 13.4 | 3.0 | 52.6 | 33.3 | 9.0 | 5.1 | 2.171 | 0.538 |
| **Dove** | 50.0 | 41.1 | 8.9 | 0.0 | 64.7 | 30.9 | 2.9 | 1.5 | 4.816 | 0.186 |
| **Ducks** | 57.1 | 39.3 | 3.6 | 0.0 | 70.0 | 27.5 | 2.5 | 0.0 | 1.192 | 0.551 |
| **Sheep** | 38.2 | 44.1 | 14.7 | 2.9 | 65.7 | 17.1 | 11.4 | 5.7 | 7.066 | 0.070 |
| **Cows** | 29.4 | 41.2 | 5.9 | 23.5 | 41.2 | 29.4 | 23.5 | 5.9 | 4.267 | 0.234 |
| **Other** | 44.4 | 50.0 | 0.0 | 5.6 | 60.0 | 20.0 | 0.0 | 0.0 | 6.643 | 0.077 |

**Annex (11) Relationship between cases and controls by percentage (percentage) according to distance between residence and professions and factories.**

| Distance By Meter | Control | | | | Case | | | | X2 | Sig. |
|---|---|---|---|---|---|---|---|---|---|---|
| | 200 and less | 200-500 | 501-1000 | >0001 | 200 and less | 200-500 | 501-1000 | >1000 | | |
| **Concrete** | 34.7 | 28.6 | 14.3 | 22.4 | 25.9 | 33.3 | 25.9 | 14.8 | 2.349 | 0.503 |
| **Crusher** | 11.8 | 58.8 | 5.9 | 23.5 | 36.4 | 22.7 | 13.6 | 27.3 | 6.126 | 0.106 |
| **Carpentry** | 44.1 | 28.8 | 11.9 | 15.3 | 50.0 | 32.5 | 7.5 | 10.0 | 1.238 | 0.744 |
| **Metalwork's** | 43.6 | 43.6 | 7.3 | 5.5 | 43.1 | 25.5 | 27.5 | 3.9 | 8.975 | 0.030 |
| **Mechanical for cars** | 39.6 | 39.6 | 14.6 | 6.3 | 59.0 | 20.5 | 15.4 | 5.1 | 4.254 | 0.235 |
| **Manufacturing Perfume** | 70.6 | 17.6 | 5.9 | 5.6 | 38.5 | 15.4 | 0.0 | 46.2 | 7.249 | 0.064 |

**Annex (12) Relationship between cases and controls according to kind of effect of exposure to soil**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Soil** | | | | | | | | |
| Skin dermatitis | 2 | 5.4 | 8 | 6.3 | 10 | 6.1 | 0.036 | 0.604 |
| Shortness of breath | 11 | 29.7 | 91 | 71.1 | 102 | 61.8 | 20.807 | 0.000 |
| Allergic rhinitis | 21 | 56.8 | 54 | 42.2 | 75 | 45.5 | 2.457 | 0.084 |
| Eye allergy- conjunctivitis | 10 | 27.1 | 14 | 10.9 | 24 | 14.5 | 5.978 | 0.018 |

**Annex (13) Relationship between cases and controls according to kind of effect of exposure to cement**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Cement** | | | | | | | | |
| Skin dermatitis | 3 | 8.6 | 9 | 7.6 | 12 | 7.8 | 0.033 | 0.547 |
| Shortness of breath | 11 | 31.4 | 95 | 80.5 | 106 | 69.3 | 30.522 | 0.001 |
| Allergic rhinitis | 18 | 51.4 | 42 | 35.6 | 60 | 39.2 | 2.840 | 0.069 |
| Eye allergy- conjunctivitis | 11 | 31.4 | 13 | 11.0 | 24 | 15.7 | 8.503 | 0.006 |

**Annex (14) Relationship between cases and controls according to kind of effect of exposure to perfume**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Perfume** | | | | | | | | |
| Skin dermatitis | 4 | 11.4 | 9 | 5.7 | 13 | 6.8 | 1.471 | 0.194 |
| Shortness of breath | 8 | 22.9 | 116 | 73.9 | 124 | 64.6 | 32.581 | 0.001 |
| Allergic rhinitis | 23 | 65.7 | 66 | 42.0 | 89 | 46.4 | 6.452 | 0.009 |
| Eye allergy- conjunctivitis | 4 | 11.4 | 12 | 7.6 | 16 | 8.3 | 0.537 | 0.325 |

**Annex (15 )Relationship between cases and controls according to kind of effect of exposures to exhausts fuels from vehicles , burn of wood , plastic and derivatives of petroleum**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **Exhausts' fuels from cars** | | | | | | | | |
| Skin dermatitis | 1 | 1.6 | 5 | 3.1 | 6 | 2.7 | 0.373 | 0.472 |
| Shortness of breath | 26 | 41.9 | 135 | 83.3 | 161 | 71.9 | 38.014 | 0.000 |
| Allergic rhinitis | 29 | 46.8 | 49 | 30.2 | 78 | 34.8 | 5.369 | 0.016 |
| Eye Allergy- conjunctivitis | 10 | 16.1 | 12 | 7.4 | 22 | 9.8 | 3.851 | 0.047 |
| **Exhausts burn of wool** | | | | | | | | |
| Skin dermatitis | 2 | 4.2 | 8 | 5.4 | 10 | 5.1 | 0.121 | 0.535 |
| Shortness of breath | 25 | 52.1 | 122 | 83.0 | 147 | 75.4 | 18.631 | 0.000 |
| Allergic rhinitis | 14 | 29.2 | 43 | 29.3 | 57 | 29.2 | 0.000 | 0.573 |
| Eye allergy conjunctivitis | 13 | 27.1 | 17 | 11.6 | 30 | 15.4 | 6.694 | 0.012 |
| | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 5 | 3.7 | 5 | 2.7 | 1.739 | 0.229 |
| Shortness of breath | 28 | 60.9 | 121 | 89.0 | 149 | 81.9 | 18.286 | 0.001 |
| Allergic rhinitis | 12 | 26.1 | 35 | 25.7 | 47 | 25.8 | 0.002 | 0.553 |
| Eye Allergy- conjunctivitis | 9 | 19.6 | 11 | 8.1 | 20 | 11.0 | 4.629 | 0.035 |
| **Derivatives of petroleum** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 5 | 4.8 | 5 | 3.6 | 1.528 | 0.258 |
| Shortness of breath | 21 | 65.6 | 89 | 84.8 | 110 | 80.3 | 5..676 | 0.020 |
| Allergic rhinitis | 9 | 28.1 | 31 | 29.5 | 40 | 29.2 | 0.023 | 0.534 |
| Eye allergy- conjunctivitis | 4 | 12.5 | 10 | 9.5 | 14 | 10.2 | 0.237 | 0.421 |

**Annex (16) Relationship between cases and controls according to kind of effect of exposure to Chemical cleaning substance and some kitchen activities**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **Chemical cleaning substance** | | | | | | | | |
| Skin dermatitis | 7 | 11.7 | 8 | 5.8 | 15 | 7.5 | 2.101 | 0.125 |
| Shortness of breath | 25 | 41.7 | 116 | 83.5 | 141 | 70.9 | 35.436 | 0.001 |
| Allergic rhinitis | 28 | 46.7 | 39 | 28.1 | 67 | 33.7 | 6.499 | 0.009 |
| Eye allergy- conjunctivitis | 8 | 13.3 | 13 | 9.4 | 21 | 10.6 | 0.704 | 0.273 |
| **Frying of oil** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 3 | 2.6 | 3 | 2.2 | 0.608 | 0.579 |
| Shortness of breath | 12 | 52.2 | 103 | 88.8 | 115 | 82.7 | 18.018 | 0.001 |
| Allergic rhinitis | 8 | 34.8 | 25 | 21.6 | 33 | 23.7 | 1.856 | 0.173 |
| Eye Allergy- conjunctivitis | 5 | 21.7 | 10 | 8.6 | 15 | 10.8 | 3.431 | 0.073 |
| **Entrance of kitchen at time of cooking** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 5 | 4.9 | 5 | 4.1 | 1.073 | 0.385 |
| Shortness of breath | 13 | 61.9 | 91 | 89.2 | 104 | 84.6 | 9.945 | 0.004 |
| Allergic rhinitis | 6 | 28.6 | 23 | 22.5 | 29 | 23.6 | 0.351 | 0.367 |
| Eye Allergy- conjunctivitis | 4 | 19.0 | 11 | 10.8 | 15 | 12.2 | 1.110 | 0.236 |

**Annex (17) Relationship between cases and controls according to kind of effect of exposure to mold**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **Mold inside home** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 5 | 6.4 | 5 | 2.2 | 1.284 | 0.328 |
| Shortness of breath | 9 | 47.4 | 66 | 84.3 | 75 | 77.3 | 12.087 | 0.001 |
| Allergic rhinitis | 9 | 47.4 | 19 | 24.4 | 28 | 28.9 | 3.939 | 0.047 |
| Eye allergy-conjunctivitis | 4 | 21.1 | 8 | 10.3 | 12 | 12.4 | 1.643 | 0.182 |

**Annex (18) Relationship between cases and controls according to kind of effect of exposure to dust inside home**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % | | |
| **Dusts inside home** | | | | | | | | |
| Skin dermatitis | 2 | 3.4 | 6 | 3.5 | 8 | 3.5 | 0.001 | 0.669 |
| Shortness of breath | 25 | 43.1 | 136 | 80.0 | 161 | 70.6 | 28.372 | 0.001 |
| Allergic rhinitis | 33 | 56.9 | 63 | 37.1 | 96 | 42.1 | 6.982 | 0.007 |
| Eye allergy- conjunctivtis | 9 | 15.5 | 14 | 8.2 | 23 | 10.1 | 2.528 | 0.094 |

**Annex (19) Relationship between cases and controls according to kind of effect of exposure URTI**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **URTI** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 4 | 2.2 | 4 | 1.7 | 1.192 | 0.355 |
| Shortness of breath | 27 | 50.9 | 161 | 89.0 | 188 | 80.3 | 37.495 | 0.001 |
| Allergic rhinitis | 25 | 47.2 | 53 | 29.3 | 78 | 33.3 | 5.903 | 0.013 |
| Eye-allergy conjunctivitis | 8 | 15.1 | 8 | 4.4 | 16 | 6.8 | 7.333 | 0.012 |

**Annex (20) Relationship between cases and controls according to kind of effect of exposure GERD**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **GERD** | | | | | | | | |
| Skin dermatitis | 1 | 5.0 | 4 | 3.9 | 5 | 4.1 | 0.054 | 0.595 |
| Shortness of breath | 16 | 80.0 | 94 | 91.3 | 110 | 89.4 | 2.247 | 0.136 |
| Eye allergy conjunctivitis | 2 | 10.0 | 7 | 6.7 | 9 | 7.3 | 0.245 | 0.448 |
| Allergic rhinitis | 2 | 10.0 | 12 | 11.7 | 14 | 11.4 | 0.045 | 0.594 |

**Annex (21) Relationship between cases and controls according to kind of effect of exposure psychological changes**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | | |
| **depression** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 3 | 2.2 | 3 | 1.7 | 0.911 | 0.454 |
| Shortness of breath | 30 | 75.0 | 118 | 88.1 | 148 | 85.1 | 4.134 | 0.042 |
| Allergic rhinitis | 6 | 15.0 | 24 | 17.9 | 30 | 17.2 | 0.183 | 0.436 |
| Eye allergy conjunctivitis | 4 | 10.0 | 9 | 6.7 | 13 | 7.5 | 0.480 | 0.346 |
| Grief | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 4 | 2.6 | 4 | 2.0 | 1.145 | 0.368 |
| Shortness of breath | 35 | 81.4 | 134 | 93.5 | 178 | 90.8 | 5.862 | 0.022 |
| Allergic rhinitis | 5 | 11.6 | 15 | 9.8 | 20 | 10.2 | 0.122 | 0.457 |
| Eye allergy- conjunctivitis | 4 | 9.3 | 8 | 5.2 | 12 | 6.1 | 0.964 | 0.255 |
| **Stress** | | | | | | | | |
| Skin dermatitis | 0 | 0.0 | 6. | 3.7 | 6 | 2.9 | 1.727 | 0.283 |
| Shortness of breath | 35 | 77.8 | 149 | 92.5 | 184 | 89.3 | 8.042 | 0.008 |
| Allergic rhinitis | 11 | 24.4 | 22 | 13.7 | 33 | 16.0 | 3.038 | 0.068 |
| Eye allergy- conjunctivitis | 4 | 8.9 | 8 | 5.0 | 12 | 5.8 | 0.985 | 0.252 |

**Annex (22) Relationship between cases and controls according to kind of effect of exposure to seasonal variation (weather)**

| | Control | | Case | | Total | | $X^2$ | Sig. |
|---|---|---|---|---|---|---|---|---|
| | **No** | **%** | **No** | **%** | **No** | **%** | | |
| **Winter** | | | | | | | | |
| Skin dermatitis | 2 | 6.1 | 10 | 6.1 | 12 | 6.1 | 0.000 | 0.626 |
| Shortness of breath | 13 | 39.4 | 138 | 83.6 | 151 | 76.3 | 29.735 | 0.000 |
| Allergic rhinitis | 20 | 60.6 | 53 | 32.1 | 73 | 36.9 | 9.586 | 0.002 |
| Eye allergy- conjunctivitis | 4 | 12.1 | 12 | 7.3 | 16 | 8.1 | 0.870 | 0.266 |
| **Summer** | | | | | | | | |
| Skin | 1 | 9.1 | 3 | 3.1 | 4 | 3.7 | 1.017 | 0.351 |
| Shortness of breath | 4 | 36.4 | 78 | 79.6 | 82 | 75.2 | 9.917 | 0.005 |
| Allergic rhinitis | 4 | 36.4 | 30 | 30.6 | 34 | 31.2 | 0.152 | 0.467 |
| Eye allergy- conjunctivitis | 3 | 27.3 | 10 | 10.2 | 13 | 11.9 | 2.743 | 0.124 |
| **Autumn** | | | | | | | | |
| Skin dermatitis | 1 | 5.6 | 4 | 4.0 | 5 | 4.3 | 0.085 | 0.573 |
| Shortness of breath | 6 | 33.3 | 78 | 78.8 | 84 | 71.8 | 15.540 | 0.000 |
| Allergic rhinitis | 11 | 6.1 | 27 | 27.3 | 38 | 32.5 | 7.952 | 0.007 |
| Eye Allergy conjunctivitis | 2 | 11.1 | 12 | 12.1 | 14 | 12.0 | 0.015 | 0.632 |
| **Spring** | | | | | | | | |
| Skin dermatitis | 2 | 7.7 | 5 | 3.6 | 7 | 4.2 | 0.922 | 0.301 |
| Shortness of breath | 5 | 19.2 | 112 | 80.0 | 117 | 70.5 | 38.922 | 0.000 |
| Allergic rhinitis | 15 | 57.7 | 39 | 27.9 | 54 | 32.5 | 8.893 | 0.004 |
| Eye allergy-conjunctivitis | 6 | 23.1 | 13 | 9.3 | 19 | 11.4 | 4.115 | 0.053 |

**ملخص الدراسة**

## عوامل الخطر لمرضى الربو بين البالغين في قطاع غزة

لا يزال الربو واحدا من الحالات الشائعة والأمراض المزمنة الخطيرة التي تفرض عبئا كبيرا على المرضى وأسرهم والمجتمع. وهو يسبب أعراض الجهاز التنفسي والحد من النشاط و في حالات التهيج الشديد يتطلب الرعاية الصحية العاجلة ويمكن أن يكون هذا التهيج مهددا للحياة.

الهدف العام من هذه الدراسة هو تحديد واكتشاف عوامل الخطر الرئيسية المرتبطة بالربو مثل العوامل الديمغرافية والاجتماعية والاقتصادية والبيئية والنفسية وغيرها من العوامل التي تؤثر في الربو في قطاع غزة وهذا بدوره سيساعد في تطوير قاعدة البيانات الأساسية(حيث إن هذه الدراسة تعتبر الأولي من نوعها في قطاع غزة) عن هذا المرض والتي يمكن أن تساعد في تطوير برنامج وقائي يهدف إلى الحد من عبء الربو .

وأجريت الدراسة في عيادات الأونروا في قطاع غزة على 214( حالة مصابة بالمرض) و 214(شخص غير مصاب بالمرض) وتم مقارنتها مع بعضهم وتم تثبيت عناصر (العمر والجنس والعنوان) وفق خصائص معينة و تم جمع البيانات من خلال استبيان منظم خاص تم تصميمه من قبل الباحث وتم اختيار العيادات و الحالات بطريقة عشوائية .

وقد أظهرت نتائج هذه الدراسة عوامل الخطر التالية التي تؤثر في مرض الربو في قطاع غزة ولها (معامل ترجيح وذات دلاله إحصائية معتبرة) تشمل التالي :

نقص المستوى التعليمي (أقل من المستوى الجامعي) و التدخين وخصوصا الأرجيلة وعدم وجود مصادر للتدفئة في الشتاء وأيضا عدم وجود مصادر لتبريد الهواء في الصيف بالإضافة إلى ما سبق تعتبر العوامل الوراثية والحساسية سواء للدواء أو الطعام عوامل مهمة جدا.وأظهرت الدراسة أيضا بأن التعرض للعطور والغبار المنزلي والتهابات الجهاز التنفسي العلوي و التغيرات المناخية وأيضا التعرض لضغوط الحياة (التوتر) جميعهم عوامل خطر لمرض الربو.

وأظهرت النتائج في هذه الدراسة وجود علاقة إيجابية بين الربو(مع دلاله إحصائية معتبرة) وكل من حجم الأسرة أكثر من 8 أفراد ،الدخل المنخفض (أقل من 1000 شيكل في الشهر)، عدم العمل ،عدم ممارسة الرياضة،التدخين السلبي والعيش في مكان قريب من حاويات القمامة، و استخدام حصيرة لتغطية الأرض وطلاء المنزل بالبوليجاد وعندما يكون بلاط المنزل حجري وحجم المنزل أقل من 140م وعندما يكون المنزل مكون من غرفتين أو أقل، وكذلك التعرض للتربة، الاسمنت، وقود العادم، ومشتقات الوقود، وحرق الأخشاب والمواد البلاستيكية و ومواد التنظيف الكيميائية وقلي الزيت ، دخول المطبخ في وقت الطهي ، والارتجاع وأخيرا الحالة النفسية (الاكتئاب والحزن)

وأيضا هناك علاقة بين الربو(من غير دلاله إحصائية معتبرة) وكل من السكن في المنازل المبنية من الاسبست أو الزينكو و النوم في الغرف الضيقة (م2*م2)او عدد الأفراد داخل غرفة النوم أكثر من 4 أشخاص الطبخ خارج المنزل واستخدام الأخشاب كوقود للطهي والسكن بالقرب من الحظائر والكسارات.

وأظهرت الدراسة أنه لا توجد علاقة بين الربو و الإقامة بالقرب من محطة الكهرباء، المولدات، الأفران ، المزارع أو الشوارع الرئيسية .

وهذا بدوره سيساعد في تطوير قاعدة البيانات الأساسية (حيث إن هذه الدراسة تعتبر الأولى من نوعها في قطاع غزة) عن هذا المرض والتي يمكن أن تساعد في تطوير برنامج وقائي يهدف إلى الحد من عبء الربو .

وقد خرجت هذه الدراسة ببعض التوصيات الهامة من بينها:

**توصيات لصناع القرار والإدارة من أجل توحيد الرعاية للربو القصبي**

*وضع استراتيجيات وقائية للحد من عبء الربو وتحسين الوضع الصحي في قطاع غزة

*وضع الربو كأولوية أمام صناع القرارات الصحية كالأمراض المزمنة الأخرى مثل ارتفاع ضغط الدم وداء السكري.

*عمل ملفات خاصة للربو من اجل المتابعة

* يحتاج نظام الرعاية الصحية الفلسطيني إلى تقييم الخدمات المقدمة لمرضي الربو، وتحديد الفجوة في هذه الخدمات

*وضع بروتوكولات موحدة لمتابعة الربو وتحديد عوامل الخطر لكل مريض

* إدارة الربو القائمة على السيطرة، وتعديل العلاج الدوائي وغير الدوائي في دورة مستمرة تتضمن التقييم والعلاج والمراجعة (المبادرة العالمية للربو، 2017)
*إيقاف التدخين بمختلف أشكاله وأنواعه

**توصيات عامة للحد من عوامل الخطر والعوامل التي لها علاقة بالربو**

- التوقف عن التدخين بمختلف أشكاله وأنواعه
- تحسين حالة السكن
- تبني تعزيز الصحة كإستراتيجية عامة
- هناك حاجة لتطوير أنشطة مجتمعية لزيادة الوعي الصحي بالربو القصبي
- تطعيم ضد فيروس الأنفلونزا
- علاج الحالات المرضية المشتركة
- تجنب التعرض للمواد المسببة للحساسية والملوثات أو المهيجات التي تجعل الربو أكثر سوا كما وردت في الدراسة .

**توصيات على مستوى المركز الصحي**

الأهداف طويلة الأجل لإدارة الربو تتحقق من خلال مراقبة جيدة للأعراض، وتقليل المخاطر المستقبلية للتفاقم، والحد من الآثار الجانبية للعلاج. وينبغي أيضا تحديد أهداف المريض الخاصة بشأن الربو وعلاجه.
- تتطلب الإدارة الفعالة للربو شراكة بين الشخص المصاب بالربو ومقدمي الرعاية الصحية.
- قد يؤدي تعليم مهارات الاتصال لمقدمي الرعاية الصحية إلى زيادة رضا المرضى، وتحسين النتائج الصحية، وتقليل استخدام موارد الرعاية الصحية.
- زيادة قدرة المريض على الحصول على المعلومات الصحية الأساسية ومعالجتها وفهمها لاتخاذ القرارات الصحية المناسبة.
- يوصى باتخاذ التدابير اللازمة للسيطرة على التعرض للمواد المسببة للتهيج لمرض الربو.