ENVIRONMENTAL RISK FACTORS WITH POTENTIALS FOR INFLUENCING THE OCCURRENCE OF ASTHMA AMONG CHILDREN IN IBADAN, NIGERIA

BY

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A DISSERTATION SUBMITTED TO THE UNIVERSITY OF IBADAN
IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE
AWARD OF MASTERS OF PUBLIC HEALTH
(ENVIRONMENTAL HEALTH) DEGREE

DEPARTMENT OF EPIDEMIOLOGY, MEDICAL STATISTICS
AND ENVIRONMENTAL HEALTH, FACULTY OF PUBLIC HEALTH,
COLLEGE OF MEDICINE,
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JULY, 2012

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CERTIFICATION

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DEDICATION

This research work is dedicated to GOD, THE ALMIGHTY who has made it possible for me to complete it successfully, unto HIM be praise for ever.

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ACKNOWLEDGEMENT

To God be the glory great things He hath done. I give God Almighty the Glory great things He hath done. I express my profound gratitude to God for giving me life, grace and strength to complete this work. May His name be praised for ever.

My sincere appreciation goes to my wonderful supervisor, Dr. G..R.E.E Ana, your constructive criticism, counsel, guidance and advice and your unrelenting effort in making this work a reality is overwhelming. The lord will richly reward you.

My special thanks goes to Professor M.K.C.Sridhar a father indeed, who supervised and thoroughly guided me throughout the period of carrying out this research. God bless you sir.

I cannot but remember Professor Falade of the Paediatrics Department, UCH for his fatherly advice and contribution to the successful completion of this project.

My special appreciation goes to the Head of Department of EMSEH, Dr Fawole for her motherly advice, encouragement and support. I also appreciate Dr. Oshiname of The Health Promotion and Education Department who painstakingly went through my abstract. I cannot but remember a mother indeed, Dr. Oloruntoba for her constant encouragement and assistance throughout the course of this work.

To all my lecturers, Professor Bamgboye, Dr. Okareh, Dr. Fatiregun, Dr. Yusuf, Dr. Bolaji who have contributed in one way or the other to the success of this work, I say a big THANK YOU. You have all contributed to make this work what it is. God bless you all.

I appreciate all my colleagues, Hammed Taiwo, Pastor Awe, Wale, Tolu, Tope, Bimbo, Buki, Andrew, Nike, Rachael, Femi, Feyi, Yemi, Mrs. Ajayi, Mrs. Omotayo and all others for their encouragement, love , care and advice.

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I am particularly very grateful to Mr Akinyemi, Mr Nathaniel, Wale Morakinyo, Mrs Titi Badru(Family Health International, Abuja) and all those that assisted in the analysis of my data.

My deepest gratitude goes to Mummy and Daddy Aluko for their prayers, care, encouragement and assistance given to me in the course of this work. God will richly reward you, you will live long enough to enjoy the rewards of your labour.

My sincere thanks to my parents Mr and Mrs Akindele Daramola who stood by me throughout the period of this programme. Your love, care, encouragement, prayers and assistance has brought me thus far, I pray that your labour will not be in vain in Jesus name. I cannot but appreciate my siblings Buki, Seun, Yemi and Tosin for their assistance, prayers and encouragement, may the Lord bless you all.

To MINE, Mr. Ijakwu Adah Odeh, my inestimable jewel, the love of my life, my Sweet heart, you are indeed one in a million, I say kudos to you. Your support and assistance financially, morally and spiritually was indeed wonderful, I say thank you.

To my lovely jewels, the wonderful gifts of God to me Victor Oluwatobi Adehi Odeh and Vera Oluwanifemi Ene Odeh, I say thank you for your understanding, cooperation and endurance throughout the period of doing this project.

I also appreciate Mr Oyeyemi of computer room, health promotion for all his assistance throughout the period of writing this thesis. I cannot but appreciate all the administrative members of staff of EMSEH, Mr Tony, Mr. Dotun, Mr. Akin, Mrs. Ashipa, Aunty Yinka for all their contributions in one way or the other. GOD BLESS YOU ALL.

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ABSTRACT

The prevalence of asthma has increased in recent years. Although most asthma-related studies in Nigeria focused on the burden of the disease among children, the risk factors that may favour its occurrence have not been well investigated. This study was therefore designed to determine the environmental factors which have potential for precipitating the occurrence of asthma among children in Ibadan.

A case-control study design was employed with 32 asthmatic children serving as Cases and 32 non- asthmatic children but with non- life threatening health conditions constituting the Controls. The study participants aged 4-15 years were consecutively selected from the patients attending the children outpatient clinic of the University College Hospital and Oni Memorial Children Hospital, Ibadan from January-July, 2008. A validated questionnaire was used to collect data from their parents. The participants and their parents were traced to their homes where both indoor and outdoor environmental monitoring was done using dust track for Particulate Matter (PM) and digital gas meter for Carbon-monoxide (CO), Nitrogen-dioxide (NO_2) and Sulphur-dioxide (SO_2). Data were analysed using descriptive statistics, Chi-square and Mann Whitney U tests. The results were compared with the National Guideline Limit (NGL).

The mean ages of the cases and controls were 8.9±3.6 years and 8.8±3.2 years respectively. There were more male cases (62.5%) and controls (75.0%). Significantly more cases (56.3%) had a family history of asthma compared with the controls (15.6%) ($p<0.05$). The prevalence rates of parental smoking among cases and controls were 12.5% and 6.3% respectively. Parents' rates of reporting mold growth on the walls of their houses among cases and controls were 40.6% and 34.4% respectively. A higher proportion of the houses of the cases (40.6%) compared with the controls (21.9%) were near industries. More cases (34.4%) had pets in their houses than the controls (25.0%). Significantly more cases (87.5%) compared with the control (21.9%) reported allergic reaction to dust ($p<0.05$). Episodes of asthma were significantly higher during the rainy season (65.6%) than the dry season (9.4%)

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($p<0.05$). There was no significant difference in the median concentration of indoor CO in the living room of the cases (0.2ppm) (range: 0.04ppm -0.67ppm) compared with the controls (0.2ppm) (range: 0.03ppm - 0.3ppm). The median concentration of indoor NO_2 in the cases' kitchen was 0.02ppm (range: 0.001ppm to 0.2ppm) compared with 0.01ppm among the controls (range: 0.01ppm to 0.04ppm). The median concentration of the outdoor particulate matter among the cases was 28.75μg/m^3 (range: 0.14 μg/m^3 – 186.70 μg/m^3) compared with the controls (34.7 μg/m^3) (range: 10.6 μg/m^3-360.2 μg/m^3) and the difference was not statistically significant. The median concentrations for PM, CO and NO_2 among the cases and controls were PM (Cases-28.75μg/m^3; Controls-34.7 μg/m^3), CO (Cases- 0.2ppm; Controls- 0.2ppm), NO_2 (Cs-0.02ppm; Ct- 0.01ppm) and these fell within the NGL of 250 μg/m^3, 10ppm and 0.04-0.06ppm respectively.

Smoking cigarettes, dust and rainy conditions were the main environmental factors that were linked with asthma in children. Public enlightenment interventions are needed to sensitise parents about these risk factors.

Keywords: Asthma, children, Environmental risk factors, Seasonal variation, Ibadan.

Word counts: 492

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CHAPTER ONE

INTRODUCTION

1.1 Background information

Bronchial asthma is a chronic disorder characterized by airway inflammation, reversible airway obstruction, and airway hyperresponsiveness. Eosinophils are believed to play important roles in the pathogenesis of asthma through the release of inflammatory mediators (Nakagome et al, 2011). Even though the pathogenesis of bronchial asthma is not completely understood, it is evident that this clinical condition has a multifactorial etiology.

There has been a sharp increase in the global prevalence, morbidity, mortality, and economic burden associated with asthma over the last 40 years, particularly in children. Approximately 300 million people worldwide currently have asthma, and its prevalence increases by 50% every decade. In North America, 10% of the population have asthma. Asthma is underdiagnosed and undertreated, although the use of inhaled corticosteroids has made a positive impact on outcomes. 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 (Braman et al, 2006).

Most asthma deaths occur in those greater than or equal to 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,

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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 et al, 2006).

People with asthma can suffer from symptoms ranging from wheezing, cough and a sensation of tightness in the chest, to a severe inability to expel air from the lungs, suffocation and death. Although asthma can begin at any age, it most commonly occurs in childhood. In some cases, as children grow older, their asthma becomes less severe or resolves altogether. People who had asthma as children sometimes experience a recurrence of the disease later in life. Asthma is treated with bronchodilators to help in the event of an acute attack. Up to 80% of children with asthma develop symptoms before age five years. Asthma symptoms are often under-recognized and under-treated by both parents and healthcare providers. Common symptoms of childhood asthma may include cough, wheeze, breathlessness or chest tightness, frequently associated with colds, sleep or exercise.

Air pollution is associated convincingly with many signs of asthma exacerbation, e.g. increased bronchial hyper responsiveness, visits to emergency departments, hospital admissions, and increased medication use (Sunyer et al, 1993; Atkinson et al, 1999;Kunzli et al, 2000). Moreover, time-series data clearly show that traffic-related air pollution in urban areas has adverse effects on mortality from respiratory and cardiovascular disease (Scwartz et al, 2000; Schenker et al, 1993; Abbey et al, 1999; Pope et al, 1999; Brunekreef et al, 2000). In a study of six USA cities, after adjusting for smoking, the mortality rate ratio increased in the most polluted areas compared with the least polluted city. This observation, which initially met with some skepticism, has been confirmed in a wide range of settings and in different countries. Although the nature and concentration of outdoor pollutants vary from one area to another, the most abundant pollutants in the atmosphere of urban areas are NO_2, O_3 and respirable PM. Sulphur dioxide (SO2) is an additional concern in industrial areas.

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1.2 Problem Statement

The morbidity and mortality of asthma continues to increase in spite of awareness of its pathophysiology and the availability of some potent drugs for its management. (Falade et al, 1998). Studies have shown that the incidence of asthma in children is about two to five times than in young adults (Dodge and Burrow 1980). Epidemiological surveys across the globe have shown that the prevalence of asthma is on the increase among children1-14 years (Donohue, 1995). The increase in prevalence is a major source of concern because of the attendant anguish and the socio economic burden. The incidence of asthma accounts for a lot of absenteeism among school children. It has been reported that disruption of sleep by asthma accounts for why asthmatics perform poorly academically.

Both the prevalence and severity of respiratory allergic diseases such as bronchial asthma have increased in recent years. Urbanization with its high levels of vehicular emissions and Westernized lifestyle parallels the increase in respiratory allergy in most industrialized countries.

A wealth of evidence suggests that allergic respiratory diseases such as bronchial asthma have become more common worldwide in recent years (European Community Respiratory Health Survey, 1996; Woolcock 1997; The International Study of Asthma and Allergy in Childhood (ISAAC) Steering Committee, 1998; United Nations Environment Programme and World Health Organisation Report, 1994). In parallel, much aetiological and pathogenic research has been carried out in the attempt to determine the underlying reasons. While the interplay between genetic and environmental factors in the development of allergic respiratory diseases remains a subject of investigation, it appears there is a link between the increase in the prevalence of allergic airway diseases and the increase in air pollution.

Several studies have shown the adverse effects of ambient air pollution on respiratory health (Gielen, 1997, Viegi et al, 1998; Lebowitz 1996; Holgate 1999; American Thoracic Society, 1996) and exposure to components of air pollution enhances the airway response to inhaled allergens in susceptible subjects. World wide prevalence rates vary from one community to

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1.2 Problem Statement

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another with lower rates being found generally in rural areas. In Nigeria for example , Falade et al using ISAAC (International Study of Asthma and Allergy in Childhood) found 10.7% among 13-14 years old(Falade et al, 1998), and 5.1 % among 6-7 years old(Falade et al, 2004) in Ibadan community. Faniran et al, 1999 also reported 10.2% among 8-11 years old in lagos. Okoromah(2000) reported a rate of 3.0% among 6-13 year old children in Enugu.

1.3 Justification of the study

This study which is aimed at identifying various environmental factors which have potentials for influencing the occurrence of asthma is of great importance because most studies carried out in Nigeria on asthma have focused more on the burden of the diseases among children but the risk factors that may favour its occurrence have not been well investigated. This study being the first in Ibadan will generate data that will serve as good reference material for other researchers in Nigeria and other developing countries.

1.4 Research Questions

1.What are the various environmental risk factors associated with the development of or exacerbation of asthma among children?

2. What are the routes of exposure to these environmental factors?

3. What is the level of exposure of these children to the environmental factors?

Null Hypothesis

There is no association between occurrence of asthma and exposure to environmental pollutants among the children in the selected health institutions.

Alternative Hypothesis

There is association between the occurrence of asthma and exposure to environmental pollutants among the children in the selected communities.

Research Hypothesis

The prevalence rate of asthma is higher among children exposed to some environmental factors than in those not exposed.

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1.5 Objectives of the Study

1.5.1 Broad Objective

To identify the potential linkages between some environmental factors and occurrence of asthma among children in Ibadan.

1.5.2 Specific Objectives

The specific objectives were to:

1. characterize the indoor housing conditions where the subjects reside.
2. assess the knowledge and attitude of the parents/wards of the study participants in relation to asthma.
3. determine the risk factors associated with asthma among the study populations
4. measure the air quality in the vicinity of the selected subjects
5. assess prevalence of perceived symptoms associated with asthma among the study populations.
6. identify possible linkages between the environmental risk factors and the prevalence of asthma.

1.6 Limitations of the study

The parents and the wards of the target population restricted access into their homes to carry out the environmental assessment. Recruiting the asthmatic patients was also problematic due to the low attendance of asthmatic patients at the clinic. It was very difficult recruiting the controls since many of them refused to give their consents. Location of the houses of the respondents using the addresses given was not very easy. In fact, some of the respondents could not be located, hence inability to carry out environmental assessment in their homes. Unavailability of the instruments for environmental monitoring was one of the challenges faced in this study.

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CHAPTER TWO

LITERATURE REVIEW

2.1 Definition of asthma

Asthma is a chronic respiratory disease characterized by bronchial hyper responsiveness, intermittent reversible airway obstruction, and airway inflammation (Busse et al.2001).
Genetic and environmental factor contributes to the development of asthma, and early life exposure may play an important role. It is likely that recent changes in children's environment have contributed to the increasing prevalence of asthma.

Recent studies suggest that the neonatal immune system tends to favor an allergic (IgE promoting) response to certain environmental allergens (eg, dust mite). This response is mediated in part through infant T helper cells that tends to release a series of cytokines that promotes the development of an "allergic (IgE promoting)" B cell response to certain environmental allergens. The T cell cytokine profile that favors an IgE response is termed a TH2 response. In contrast, as the immune system matures, naive T cells tend to release a different mix of cytokines in response to environmental allergen that favor an IgG promoting (TH1) response.

It is postulated that exposure to environment stimuli during early childhood could either further enhance the Th2 response depending on the stimuli and/or genetic predisposition(Gern et al. 1999; National Institutes of Health,1997). Proposed hypotheses on the environmental factors that may favor the TH2 response include improved hygiene, changes in diet, changes in intestinal flora due to increased use of antibiotics, increased exposure to allergens due to changes in housing and lifestyle, obesity and reduced physical activity, and changes in the prenatal environmental (National Institute of Health, 1997).

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The "hygiene hypothesis" postulates that early childhood infection (which promotes a TH1 response) are becoming less frequent, favoring a persistent TH2 imbalance (Busse et al, 2001; Strachan et al, 2000). Observation that the presence of an older sibling and early child care attendance are associated with a reduced incidence of asthma support this hypothesis. The development of a TH2 (IgE) response to common environme ntal contaminants in the air (eg, house-dust mites, cockroach antigens, and cat allergens) is strongly correlated with the development of childhood asthma (Gern et al, 1999; Platts –Mills et al, 2000).

The relationship between early life exposure and allergic sensitization is not completely understood. As an example of the complexity, exposure to high levels of cat allergen during infancy may protect children developing asthma, except if the mother has a history of asthma (Platts-Mills et al, 2000; Celedon et al, 2002). Once sensitized to aero allergens, optimal management of asthma includes control of exposure to environmental triggers (National Institutes of Health, 1997).

2.2 Prevalence of asthma

An increased understanding of the causes of asthma is coming from the international comparisons of asthma prevalence, particularly those from the European Community Respiratory Health Survey of asthma prevalence in adults and the International Study of Asthma and Allergies in Childhood. From these and other studies of asthma prevalence, it is possible to draw some tentative conclusions as to the patterns of asthma prevalence worldwide. There are five striking patterns: first, asthma prevalence is increasing worldwide; second, asthma is generally more common in Western countries and less common in developing countries; third, asthma is more prevalent in English-speaking countries; fourth, asthma prevalence is increasing in developing countries as they become more Westernized or communities become urbanized; and fifth, the prevalence of other allergic disorders may also be increasing worldwide. These five key features of the international patterns of asthma prevalence raise major questions about the role of "established" risk factors for the development of asthma. As a result, recent research has expanded to include the study of novel factors that may "program" the initial susceptibility to sensitization or contribute to the

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development of asthma independent of atopic sensitization. These include various exposures in utero, which are reflected in various perinatal factors measured at birth, and exposures (or lack of exposures) in the early years of life that may make the infant m ore susceptible to the subsequent development of asthma. These issues are now the focus of an intensive research effort worldwide, and the next few years are likely to see exciting advances in our understanding of the causes of asthma (Beasly et al 2000).

Until recently, most studies reported that asthma prevalence has increased in recent decades. The best indication of what is now happening globally will be provided by Phase III of the International Study of Asthma and Allergies in Childhood (ISAAC) study. Some individual ISAAC centres in Western countries, as well as several studies in adults, have already reported no increase or even a decrease in asthma prevalence over the last 10 years. 'Established' risk factors for asthma cannot account for the global prevalence increases, the international patterns or the recent declines in prevalence in some Western countries. It seems that the 'package' of changes in the intrauterine and infant environment occurring with 'Westernisation' is causing increased susceptibility to the development of asthma and/or allergy. The 'package' includes changes in maternal diet, increased foetal growth, smaller family size, reduced infant infections, increased use of antibiotics and paracetamol and immunisation, all of which have been (inconsistently) associated with an increased risk of childhood asthma, but none of which can alone explain the increases in prevalence. It is likely that the 'package' is more than the sum of its parts, and that these social and environmental changes are all pushing our immune systems in the same direction. To know what that direction is requires that better aetiological theories of asthma are developed to replace the allergen theory, or to incorporate it as a special case. Global comparisons of asthma prevalence and assessment of time trends will continue to play a major role in this process (Pearse et al, 2006).

2.3 Global burden of asthma

There has been a sharp increase in the global prevalence, morbidity, mortality, and economic burden associated with asthma over the last 40 years, particularly in children. Approximately

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300 million people worldwide currently have asthma, and its prevalence increases by 50% every decade. In North America, 10% of the population have asthma. Asthma is underdiagnosed and undertreated, although the use of inhaled corticosteroids has made a positive impact on outcomes. 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 greater than or equal to 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 et al, 2006).

2.4 Symptoms of asthma

People with asthma can suffer from symptoms ranging from wheezing, cough and a sensation of tightness in the chest, to a severe inability to expel air from the lungs, suffocation and death. Although asthma can begin at any age, it most commonly occurs in childhood. In some cases, as children grow older, their asthma becomes less severe or resolves altogether. People who had asthma as children sometimes experience a recurrence of the disease later in life. Asthma is treated with bronchodilators to help in the event of an acute attack.Up to 80% of children with asthma develop symptoms before age five years. Asthma symptoms are often under-recognized and under-treated by both parents and healthcare providers.Common symptoms of childhood asthma may include cough, wheeze, breathlessness or chest

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tightness, frequently associated with colds, sleep or exercise. Frequently multiple factors are involved in the triggering of an asthma exacerbation (Rasmussen et al, 2002).Triggers of asthma symptoms include allergens, exercise, cold air, emotional stress, viruses, air pollution, foods and food additives, medications and chemical and irritant exposures.

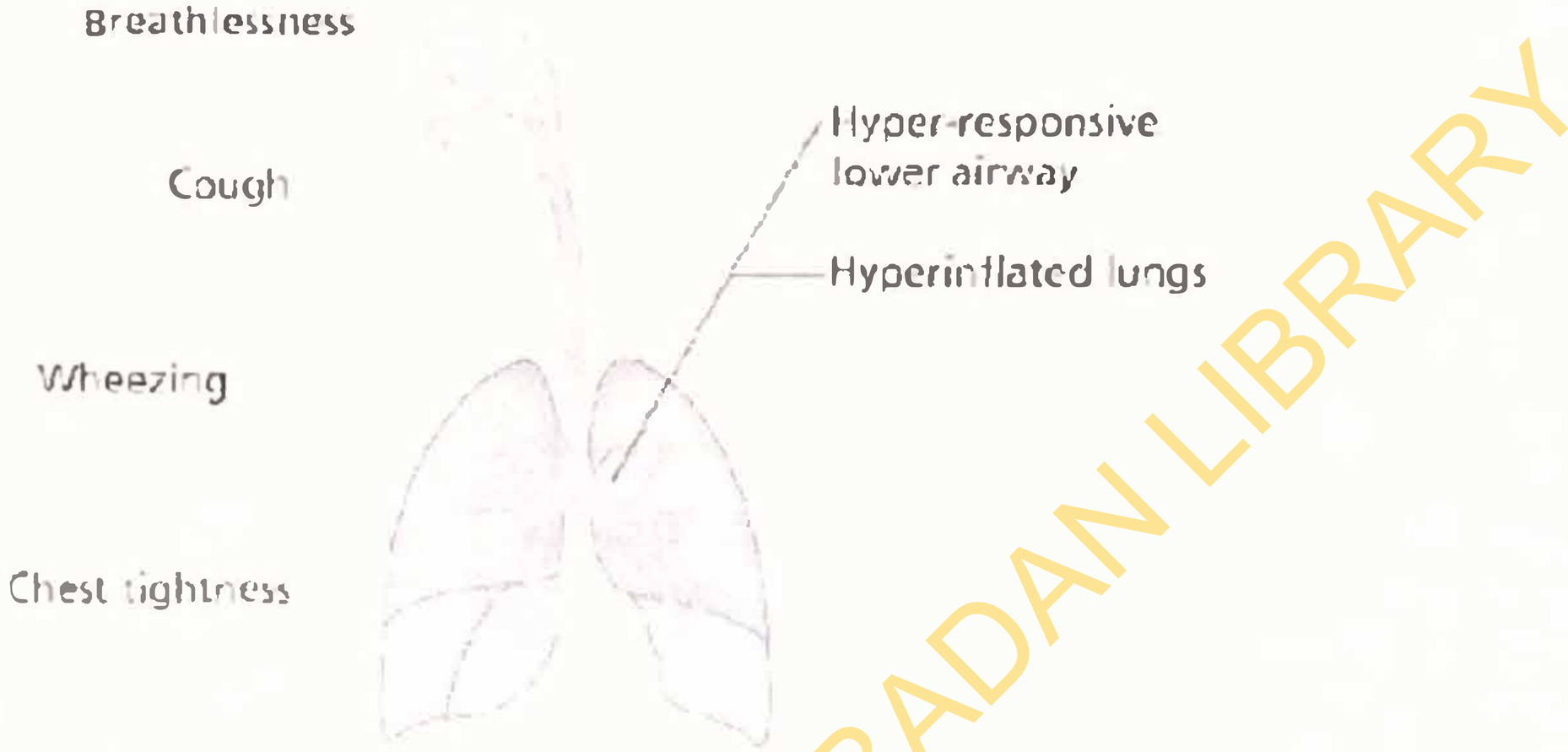


Figure 2.1: Showing common symptoms of asthma

Source: Atlas of Allergic Diseases

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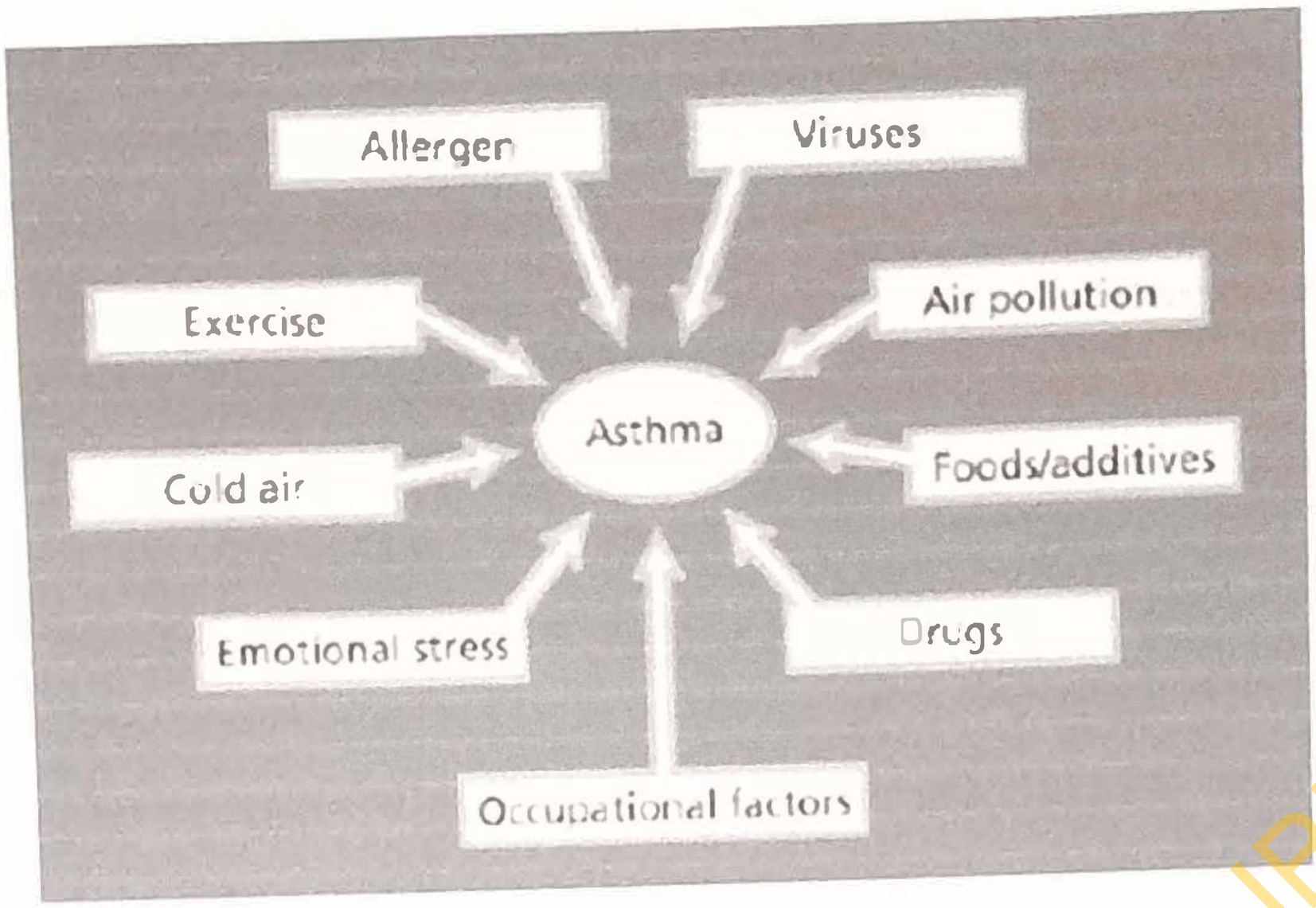


Figure 2.2: Triggers of asthma symptoms

Source: Atlas of Allergic Diseases (Lieberman et al, 2002)

2.5 Classification of asthma

Asthma can be classified using different methods. Asthma is a chronic illness affecting more than 20 million people in the US alone, of which 9 million of them are children, and the numbers are growing daily. Diagnosing asthma is a fairly straightforward process, including a personal and family medical history, lung function testing, and a physical examination by a doctor.

However, there are a number of approaches to figuring out asthma types in different people. This include the following methods:

- Types of asthma related to underlying cause or disease process
- Asthma classifications by severity
- Asthma types by degree of control

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2.5.1 Asthma Classification by Underlying Cause or Disease Process

One way to classify asthma is by separating asthma into different types or causes, though that is not truly an asthma classifications scheme. The most common cause of asthma is allergies, but there are several other types of asthma as well. Here is a list of asthma types;

- **Allergic asthma.** In this type of asthma, symptoms are triggered by environmental triggers called allergens, such as pollen, dust mites, and pet dander.
- **Non-allergic asthma.** This type of asthma is triggered by factors other than allergens, such as irritants like smoke.
- **Occupational asthma.** This type of asthma is triggered by irritants in the workplace, such as strong fumes or chemicals.
- **Exercise-induced asthma.** When asthma symptoms are triggered by exercise or vigorous exertion, it is called exercise-induced asthma.
- **Cough-variant asthma.** With this kind of asthma, the main symptom is continuous coughing. There may be shortness of breath, but generally there is no wheezing.
- **Medication-induced asthma.** Sometimes, medications such as aspirin or other non-steroidal anti-inflammatory drugs (NSAIDs for short) can trigger asthma symptoms.

2.5.2 Asthma Classifications by Severity

The National Asthma Education Program, which produces the US-based asthma treatment guidelines, classifies asthma by its severity, a more commonly used scheme by most health professionals. It's a useful way to look at asthma, because it guides asthma treatment. Classification of asthma by severity is based on frequency and severity of asthma symptoms, along with peak flow readings. Levels are referred to as steps, as follows:

- **Step 1: Mild Intermittent.** At this level, asthma symptoms occur less than 2 times a week during waking hours and less than twice a month during the night. In between asthma attacks, no symptoms occur at all, and the attacks themselves are generally brief, though their intensity can vary. *Peak flow variability is less than 20 percent.*
- **Step 2: Mild Persistent.** At this level, asthma symptoms are occurring more than twice a week, but not as often as daily. They may occasionally wake the victim up at night,

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but that happens less than 2 times a month. Asthma attacks may interfere with activity temporarily. *Peak flow tends to be more than 20—but less than 30—percent.*

- **Step 3: Moderate Persistent.** At this level, asthma is starting to interfere more with daily living. Symptoms crop up every single day, and the victim needs to use a quick-relief inhaler daily (if there is one). Asthma attacks are occurring at least twice a week and often interfere with activity. They may last for days at a time. The person wakes up 1 or more times a week with symptoms. Peak flow rate varies by more than 30 percent.
- **Step 4: Severe Persistent.** This is the most severe form of asthma and at this level, symptoms are basically continuous. Activity is severely limited and asthma attacks and night symptoms are frequent. Peak flow varies by more than 30 percent.

The National Asthma Education Program (NAEPP) advocates a step-wise approach to treating asthma in adults and children older than age 5, based on the types of asthma severity described above. For instance, mild intermittent asthma is usually treated only with quick-relief medicines, while severe persistent asthma is treated with one or more daily controller medicines and frequent use of quick-relief medicines.

2.5.3 Asthma Types by Degree of Control

The Global Initiative for Asthma (known as GINA) released new guidelines for asthma management in late 2006 that emphasized looking at asthma not only in terms of severity, but also in terms of response to treatment, which is another way of describing the level of asthma control. In addition, the new GINA guidelines use this approach with children under the age of 5, which is a major difference from the NAEPP guidelines. GINA recommends using the NAEPP types of asthma only for research purposes. Instead, they suggest these 3 asthma types, which are more a variation in mindset than anything else, these are:

- **Controlled:** at this level, there are no daytime or nighttime symptoms, no activity restrictions, and infrequent need for quick-relief medicines (no more than twice a week). Peak flow is normal and there are no asthma attacks.
- **Partly controlled:** with this asthma classification, daytime symptoms are occurring more than twice a week, sometimes at night and occasionally they limit activity. Quick-relief

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medicine is needed more than twice a week. Peak flow rate is less than 80 percent of your personal best, and asthma attacks occur at least once a year.

- **Uncontrolled:** your asthma type is classified as uncontrolled if you have 3 or more of the features of partially controlled asthma at least 3 times a week, and asthma attacks are occurring weekly.

GINA maintains that complete control of asthma is possible with the right treatment for the right types of asthma.

2.6 Diagnosis of asthma

Asthma is diagnosed by identification of the presence of a classic pattern of symptoms of recurrent airway hyper-reactivity including cough, wheeze, shortness of breath and chest tightness after the exclusion of any underlying conditions that could masquerade as asthma (National Asthma Education and Prevention Program expert Panel Report,1997).

The American Thoracic Society defined asthma as the presence of four out of five of the following clinical parameters shown to be highly sensitive and specific in making the diagnosis of asthma. These parameters include:

- ✓ Wheezing with colds
- ✓ Wheezing apart from colds
- ✓ Difficulty in breathing associated with wheeze
- ✓ Wheezing after exertion
- ✓ Persistent cough

Physical examination may be remarkable for prolonged exhalation phase, increased effort of breathing, wheezing or diminished lung sounds and in severe cases cyanosis.

Due to the variable nature and fluctuating course of asthma, all clinical examination features may be normal. The diagnosis of asthma may be confirmed in children older than age four years through pulmonary function tests that demonstrate reversible airflow limitation.

Chest x-ray may show hyperinflation of the lungs when airway inflammation and reactivity are severe.

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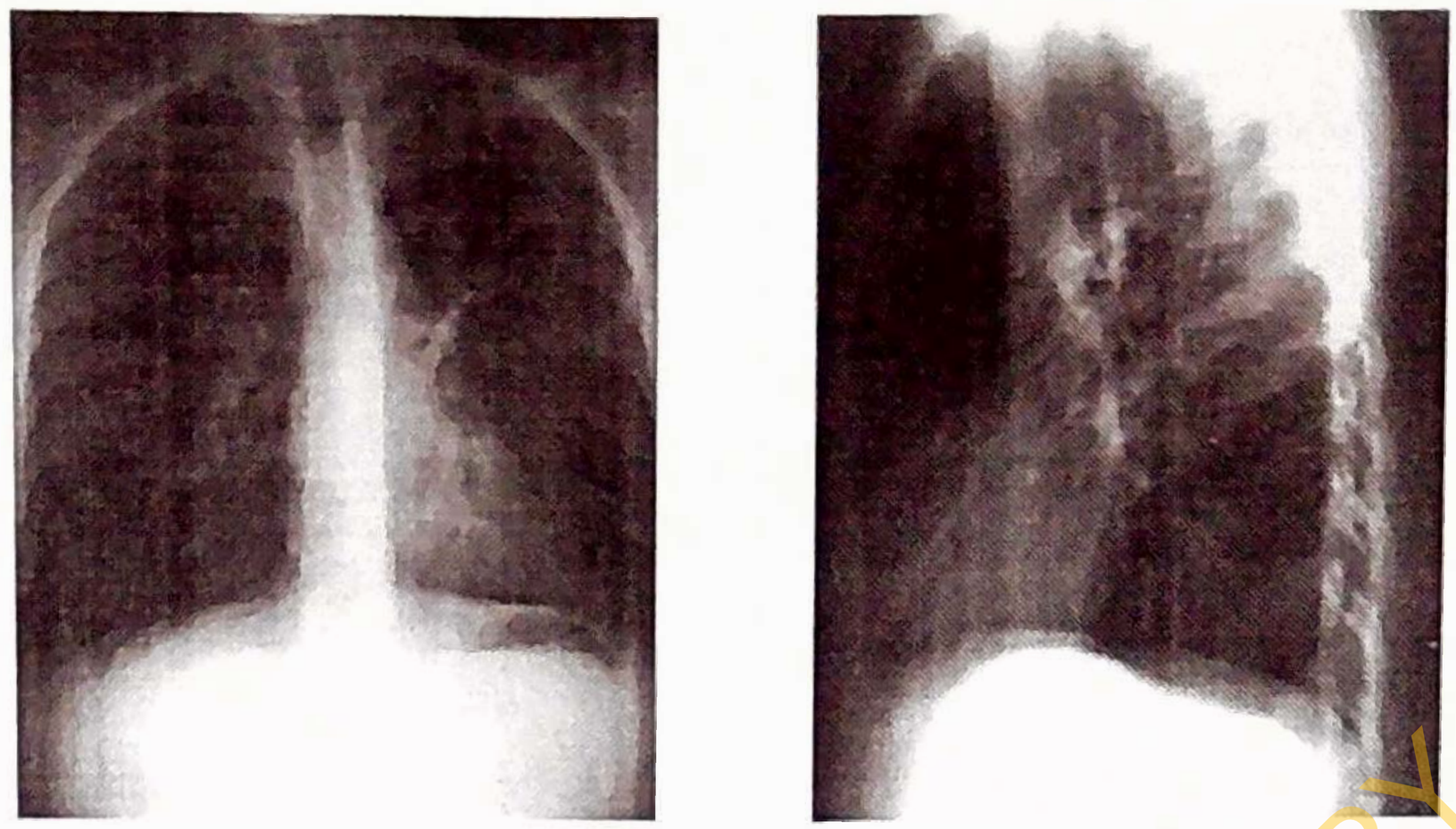


Figure 2.3 : Air Trapping with Hyperinflation in an 8 year old Asthmatic

Source: Atlas of Allergic Diseases

When dealing with children under 5 years of age, doctors rarely conduct lung function tests because young children usually have trouble following the instructions. Instead, a hit and trial method is used. When a child's symptoms, medical history and physical examination suggest asthma, the doctor may prescribe a bronchodilator — a drug that opens the airways. If the child's signs and symptoms improve after using the bronchodilator, an asthma diagnosis is likely.

It is often difficult to immediately certify that asthma is the diagnosis. To reach a conclusion, the doctor will need information on:

- The symptoms, such as wheezing, coughing, shortness of breath.
- What triggers the symptoms or when do the symptoms get worse.
- Medications that were taken prior and whether they helped.
- Any family history of breathing problem, allergies or asthma.

Acute episodes are usually straightforward to interpret but chronic, low-grade symptoms can be difficult.

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2.6.1 Steps and Procedures in Asthma diagnosis

Symptoms alone aren't enough for a doctor to arrive at a diagnosis of asthma. He will also take note of the following:

1. Evaluation based on patient's history and the symptoms being displayed (such as sudden, severe episode or recurrent episodes of wheezing, coughing or shortness of breath) .
2. Patient's family history should not be neglected, as it has a strong chance of influencing the patient - whether parents, brothers, sisters or children ever have been diagnosed with asthma, allergies, sinusitis or nasal growths?
3. A physical examination of the upper respiratory tract, chest and skin generally follows the history. Using a nasal mirror, the doctor may look inside the nose for signs of allergic disease such as increased nasal secretions, swelling or polyps. These signs may suggest that allergies are responsible for triggering suspected asthma.
4. Next, the doctor may use a stethoscope to listen to the sounds the lungs make while breathing. Wheezing sounds indicate one of the main signs of asthma which is as a result of obstructed airways.
5. Finally, the doctor may examine the skin for signs of allergic conditions such as eczema or hives, which are often associated with asthma.
6. Breathing tests called **spirometry** - this test measures the amount exhaled from the lungs over time. Spirometry measures three values that are important in diagnosing asthma, these are:
    - Vital capacity, which is the maximum amount of air that one can inhale and exhale.
    - Peak expiratory flow rate, also known as the peak flow rate, which is the maximum flow rate one can generate during a forced exhalation.
    - Forced expiratory volume, which is the maximum amount of air one exhale in one second.

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7. **Challenge test** - During this test, a deliberate attempt is made to trigger airway obstruction and asthma symptoms by inhaling an airway-constricting chemical or taking several breaths of cold air.
8. Chest and sinus X-rays.
9. The doctor may also test a person's pulmonary function after administering him some asthma medication. This helps confirm that the blockage in the air passages that shows up on pulmonary function tests goes away with treatment.

2.6.2 Different tests used to diagnose asthma

Different lung pulmonary tests that may be conducted in diagnosing asthma. Some of them include:

- Bronchial provocation test
- Routine pulmonary function test
- Exercise induced bronchoconstriction test
- Allergy intradermal skin test - although allergy tests aren't used to diagnose asthma, they can help identify substances that may be causing or worsening asthma.
- Allergy prick skin test – to identify the sources of sneezing
- Bone density test
- Bone scan
- CT scan of sinuses
- Exercise tolerance test

Asthma is a complicated and difficult disease to diagnose, and the results of airway function testing may be normal even if a person has asthma.

2.6.3 When to undergo diagnosis test for asthma

If symptoms which are similar to asthma persist, the doctor may recommend an asthma test to get to the root of the problem. This is usually advised if any one of the following occur repeatedly:

- Severe bouts of asthma

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- Conditions that complicate asthma such as chronic sinusitis, rhinitis, hayfever, nasal polyps.
- Frequent treatment with steroid tablets which are not curing the problem
- Confusion with the diagnosis
- Allergies are popping up
- Asthma seems to be getting worse

Asthma is a chronic condition, where symptoms and the condition change in time. If you feel that you are not making progress with your current treatment, talk to your doctor about alternative treatment.

2.6.4 Current Tools in Asthma Diagnosis

The use of ex-haled nitric-oxide has been found to be a more robust or sensitive method for diagnosing asthma. As spirometry diagnosis tests aren't always accurate, doctors are looking for better ways to diagnose asthma. One new approach is to measure a chemical marker of asthma — nitric oxide — in exhaled air. In general, higher levels of nitric oxide corresponds with higher degrees of asthma severity.

2.7 Factors precipitating asthma

Major indoor triggers of asthma include environmental tobacco smoke (ETS), respiratory irritants such as volatile organic compounds (VOCs) and fragrances, animal and insect allergens (such as dander and cockroach antigen), and molds (Table 2.1). Although most agents that exacerbate asthma in children are inhaled, asthma may be exacerbated in some atopic individuals who touch e.g. latex or ingest certain products e.g.peanuts.Outdoor air pollution also has been associated with asthma exacerbation (Etzel et al, 2003).

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Table 2.1 Common Indoor and outdoor Agents Precipitating Asthma

AGENT	MAJOR SOURCES
Indoor	
Environmental tobacco smoke	Cigarettes, cigar, other tobacco products
Combustion pollutants	Fireplaces and wood-burning stoves
Molds	Floods, roof leaks, plumbing leaks, wet basements, air-conditioning units
Nitrogen oxides	Space heaters, gas-fueled cooking stoves
Odors or fragrances	Sprays, deodorizer, cosmetics, household cleaning products, pesticides
Volatile organic compounds	Building and insulation material, cleaning agents, solvents, pesticides, sealants, adhesive, combustion products, molds
Allergens	
Dust mites	Beddings,(pillow, mattress, box springs, bed linens), carpets, soft upholstered furniture, draperies, stuffed toys.
Animal allergens(dander, saliva, urine)	Cats, dogs, rodents
Cockroaches	Kitchen, bathrooms (near food and water sources)
Outdoor	
Pollens	Seasonal release from flowering plant
Molds	Ubiquitous in soil, increase in wet environments and decaying organic matter (eg, wood chips)
Ozone (O_3)	Motor vehicle exhaust, power plants
Particulate matter	Combustion sources (eg, diesel engines, industry, wood, wood burning)
Sulfur dioxide (SO_2)	Burning of coal (coal-fired power plants, other industrial sources)

Source: Etzel etal, 2003

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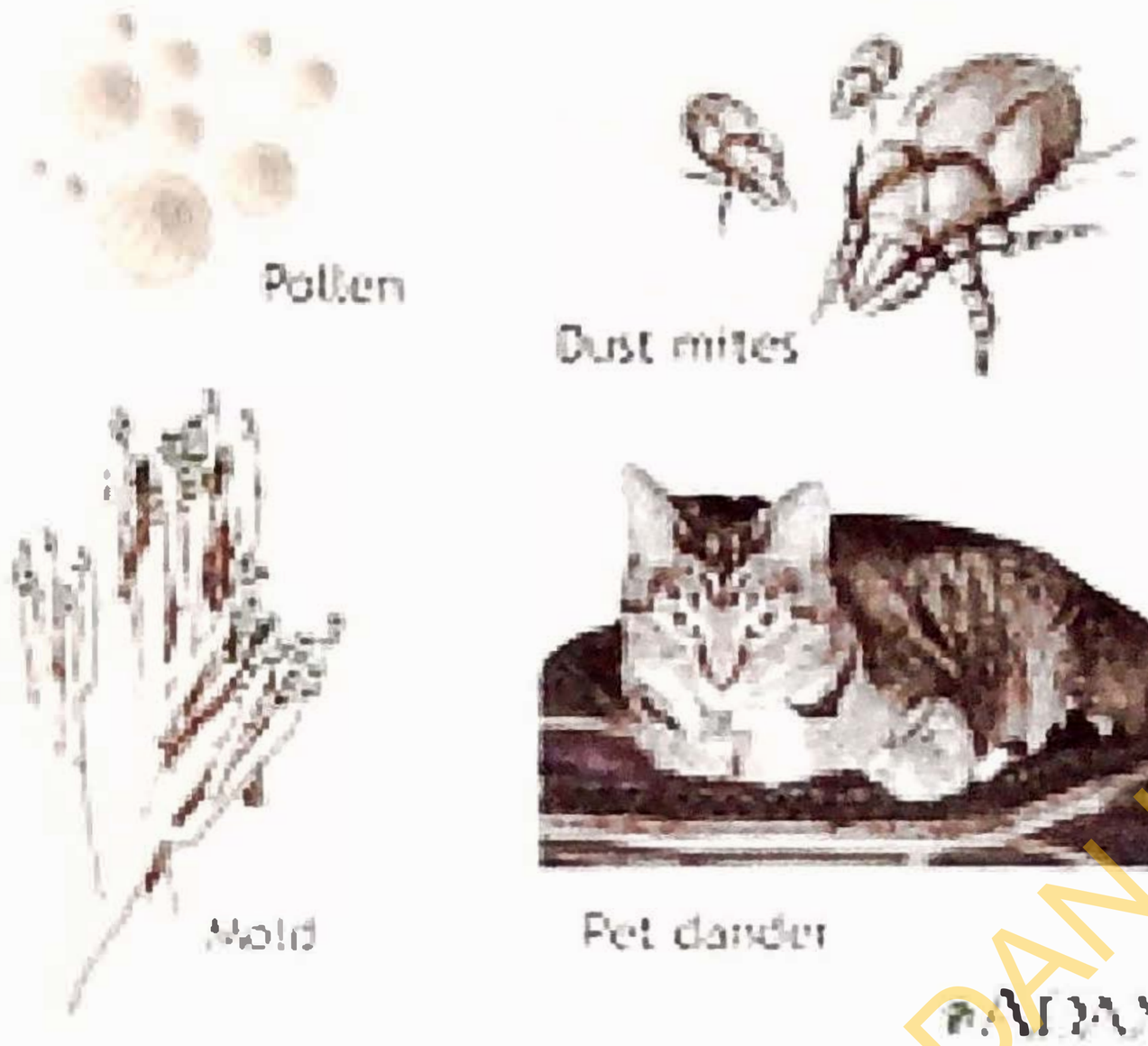


Figure 2.4: Typical indoor and outdoor precipitants

2.7.1 Indoor environmental precipitants of asthma

2.7.1.1 Environmental Tobacco Smoke

Forty-three percent of children in the United State live with at least one smoking parent (Pirlke et al, 1996). Children whose mothers smoke have more wheezing symptoms and a higher incidence of lower respiratory tract illness compared with those with mother who do not smoke(Ehrlich et al, 1996). The greatest effect seems to be related to maternal smoking during pregnancy and/or early infancy(Martinez et al,1992),\ perhaps due to inflammatory stimuli on lung parenchyma during a period of rapid lungs development and prolonged close exposure to the mother. Exposure to ETS is associated with increase in asthma attacks, earlier symptom onset, increased medication use, and a more prolonged recovery from acute attack.

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Maternal smoking of half a pack or more per day has been associated with an increased risk of developing asthma in children (Martinez et al,1992). Acute short-term ETS exposure has been demonstrated to increase bronchial hyper reactivity, requiring as long as 3 weeks to recover baseline pulmonary function following exposure (Menon et al, 1992; Committee of the environmental and occupational health assembly of the American thoracic society,1996). Environmental tobacco smoke (ETS) is probably the most important indoor pollutant, especially around young children. Doctor-diagnosed asthma is more common among non-smoking adults exposed to ETS than those not exposed. Among people with asthma, higher ETS exposure is associated with a greater risk of severe attacks (Taylor et al,1992).

2.7.1.2 Combustion related pollutants

Combustion pollutants in the home arise primarily from gas ranges, particularly when they malfunction or are used as space heaters, and from improperly vented wood stoves and fire places. Combustion of natural gas result in the emission of nitrogen dioxide(NO_2) and carbon monoxide(CO). Levels of NO_2 in the homes generally are increased during winters when ventilation s reduced to conserve energy. During the winter, average indoor concentration of NO_2 in homes with gas cooking stoves are as much as twice as high as outdoor levels. Some of the highest indoor NO_2 levels have been measured in homes where ovens were used as space heaters.

Residential levels of CO generally are low.Cooking or heating with wood results in the emission of liquids(suspended droplets), solids(suspended particles), and gases such as NO2 and sulphur dioxide(SO_2). The aerosol mixture of very fine solids and liquid particles or "smoke" contains particles in the inhalable range, less than 10μm in diameter. In several studies, concentration of inhalable particles were higher in homes with wood stoves, compared with homes without wood stoves. Depending on the frequency and duration of cooking or heating with wood and the adequacy of ventilation, the concentration of inhalable particles may exceed outdoor air standards. Epidemiologic evidence for the role of these indoor air pollutants in exacerbating asthma is limited but suggestive of associations between exposures to these pollutants and asthma exacerbation(Institute of medine 2000;Delfino et al, 2002).

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2.7.1.3 Indoor fungi (moulds).

Molds are most prominent in climates with increases ambient humidity, although some can grow in relatively dry areas. Species of common indoor molds (e.g Aspergillus, Penicillium, Cladosporium) require sufficient moisture for growth, and places where indoor mold growth is commonly found include household areas with high humidity (e.g basements, crawl spaces, ground floors, bathrooms, and areas with standing waters such as air conditioner condensers) and areas with recent moisture damage. Carpeting, ceilings and paneled or hollow walls also are common reservoirs.

Dampness or presence of mold should be suspected when there is visible mold or mildew in the home, a moldy or musty smell, evidence of water condensation on window sills(excluding immediately after shower or cooking in the kitchen), or the use of a humidifier. Epidemiologic studies have suggested an association between dampness and mold in the homes and asthma symptoms (Institute of medicine ,2000; Peat et al, 1998; Bornehag et al, 2001). Because dampness and visible mold growth could be indicators for dust mite allergen exposure, the relative contribution of fungi versus other allergens (e.g house dust mite) is not entirely clear. However several studies have found decreased respiratory symptoms in the presence of dampness after adjusting for levels of dust mite allergen (Nafstad et al, 1998; Dales et al, 1999).

2.7.1.3.1 Routes and Sources of Exposure

Exposure to molds occurs via inhalation of contaminated air and dermal contact with surfaces where they are deposited. Molds are ubiquitous in the outdoor environment and can enter the home through doorways, windows, heating, ventilation systems, and air conditioning systems. Molds proliferate in environments that contain excessive moisture, such as from pet urine or leaks in roofs, walls and plant pots. The major systems affected are the mucous membranes of the eyes, nose, throat, and respiratory tracts.

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2.7.1.3.2 Clinical Effects

The clinical effects of exposure to molds may be allergic or toxic. Some children who are exposed to molds have persistent upper respiratory tract symptoms such as rhinitis, sneezing, and eye irritation, as well as lower respiratory tract symptoms such as coughing and wheezing. Toxic effects of molds may be due to inhalation of mycotoxins, lipid-soluble toxins readily absorbed by the airways. Species of mycotoxin-producing molds include *Fusarium, Trichoderma,* and *Stachybotrys*. Exposure to *Stachybotrys* and other molds has been associated with acute pulmonary hemorrhage among young infants in Cleveland, Kansas City, MO, and Delaware. Exposure to *Trichoderma* and other molds has been associated with acute pulmonary hemorrhage in a North Carolina infant.

2.7.1.3.3 Diagnostic Methods

Several questions may help to identify exposure to molds. They include: Has the home been flooded? Is there any water-damaged wood or cardboard in the house? Has there been a roof or plumbing leak?Have occupants seen any mold or noticed a musty smell? Testing the environment for specific molds usually is not necessary. There currently is no diagnostic test for mycotoxins in human tissue.

2.7.3.1.4 Prevention of Exposure

Prevention strategies include cleaning up water and removing all water-damaged item (including carpets) within 24 hours of a flood or leak. If this is done, toxic mold will not have the opportunity to grow. If some mold already is present, the affected area needs to be washed with soap and water, followed with a solution of 1 part bleach to 10 parts water. Protective gloves should be worn during cleanup.

2.7.1.4 Formaldehyde and volatile organic compounds

Formaldehyde and VOCs—which can arise from sources such as new linoleum flooring, synthetic carpeting, particleboard, wall coverings, furniture, and recent painting—have been implicated as potential risk factors for the onset of asthma and wheezing (Garrett et al. 1999;

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Jaakkola et al. 2004; Rumchev et al. 2004). Clinicians should advise patients to be aware of the potential irritating effects of newly installed furnishings and finishes.

2.7.1.4.1 Routes and Sources of Exposure

The route of exposure to VOCs occurs through inhalation and dermal contact with surfaces where they are deposited. Many household furnishings and products of gas VOCs (US environmental protection agency, 1999, WHO,1999). These include chemicals such as aliphatic and aromatic hydrocarbons (including chlorinated hydrocarbons), alcohols, and ketones in products such as finishing rug and oven cleaners, paints and lacquers, and paint strippers. Formaldehyde is found primarily in building materials and home furnishings (US Environmental Protection Agency, 1999; WHO,1999; Diaz sanchez et al,1999).

Because product labels may not always specify the presence of organic compounds, the specific chemical (s) to which a product user may be exposed may be difficult to discern. Over the normal range of room temperatures, VOCs are released as gases or vapors from furnishings or consumer products. Volatile organic compound measurements in residential and nonresidential buildings show that exposure to VOCs is widespread and highly variable (Shusterman 2001). In general, VOCs are likely to be higher in recently constructed or renovated buildings compared with older buildings. Once building-related emissions decrease, consumer products are likely to remain the predominant source of exposure to VOC's. Studies have shown that indoor concentrations of VOCs (measured using a personal monitor) are greater than outdoor concentrations, breath levels correlate better with air exposures in a person's breathing zone than with outdoor air levels, and inhalation accounts for more than 99% of exposure for many VOCs (Bates et al, 1995).

Formaldehyde is one of the most ubiquitous indoor air contaminants. It is used in hundreds of products such as urea-formaldehyde and phenol-formaldehyde resin, used to bond laminated wood products and to bind wood chips in particle board; as a carrier solvent in dyeing textiles and paper products; and as a stiffener, wrinkle resistant, and water repellent in floor coverings eg, rugs and linoleums (US environmental protection agency, 1999; WHO,1999;

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Schwartz et al. 2000). Urea-formaldehyde foam insulation, one source of formaldehyde used in home construction until the early 1980s, is no longer used. The addition of new furniture to a home increase formaldehyde concentrations. Mobile homes, which have small enclosed spaces, low air exchanges rates, and many particle board furnishings, may have much higher concentrations of formaldehyde than other types of homes.

2.7.1.4.2 Clinical Effects

Exposures to VOCs may result in dermal, mucocutaneous, and non-specific effects(Gilliland et al,) . Depending on the dominant compounds and route and level of exposure, signs and symptoms may include upper respiratory tract and eye irritation, rhinitis, nasal congestion, rash, pruritus, headache, nausea, and vomiting (Committee of the Environmental and Occupational health Assembly of the American Thoracic Society, 1996).

Volatile organic compounds have only recently attracted some investigations, and studies are ongoing to determine which VOCs are associated with specific health effects(White et al, 1994). The effects of exposure to formaldehyde have received more attention. Formaldehyde may exacerbate asthma in some infants and children (US Environmental Protection Agency 1999), and it has been linked to nasopharyngeal cancer in occupationally exposed adults(McConnell et al, 2002).

2.7.1.4.3 Diagnostic Methods

Several questions may help to identify potential exposure. These include: Does the family live in a new home with large amounts of pressed wood products? Is there new pressed wood furniture? Have household members recently worked on craft or graphic materials? Are chemical cleaners used extensively? Has remodeling recetly been done? Has anyone recently used paints, solvents, or sprays in the home? Do the chils' symptoms clear when he is removed from the home and reappear when he returns?

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2.7.1.4.4 Prevention of Exposure

Prevention strategies include increasing ventilation and avoiding storage of opened containers of unused paints and similar materials within the home. If formaldehyde is thought to be the cause of the problem, the source should be identified and, if possible, removed. Measuring levels of formaldehyde in the air usually is not necessary. If it is not possible to remove the source, the exposure can be reduced by coating cabinets, paneling, and other furnishings with polyurethane or other nontoxic sealants and by increasing the amount of ventilation in the home. Formaldehyde concentrations decrease rapidly over the first year after a product is manufactured.

2.7.1.5 Dust mites

House-dust mites are universal in areas of high humidity (most areas of the United States) but are usually not present at high altitudes or in arid areas unless moisture is added to the indoor air (Platts-Mills et al. 1997). Mites depend on atmospheric moisture and human dander for survival. High levels of mites can be found in dust from mattresses, pillows, carpets, upholstered furniture, bed covers, clothes, and soft toys. The patient's bed is the most important source of dust mites to control. Washing bedding is advised, preferably in hot water, but cold water, detergent, and bleach can also be effective (Arlian et al. 2003; McDonald and Tovey 1992). Several recent studies support the efficacy of allergen avoidance in the treatment of asthma (Carter et al. 2001; Halken et al. 2003; Htut et al. 2001; Morgan et al. 2004; Peroni et al. 2002; Rijssenbeek-Nouwens et al. 2003; van der Heide et al. 1997). Other studies provide important insight into the details of allergen avoidance. For example, three studies reported that mattress covers without other measures were not effective (Luczynska et al, 2003; Terreehorst et al, 2003; Woodcock et al, 2003). Likewise, two well-conducted studies failed to show an effect of HEPA filters alone (Francis et al. 2003; Wood et al, 1998). Thus, the conclusion remains that effective allergen avoidance requires a comprehensive approach, and that individual steps alone are generally ineffective (Platts-Mills et al, 2000). Chemical agents are available for killing mites and denaturing the antigen; however, the effects are not dramatic and do not appear to be maintained for long periods. Therefore, use of these agents in the homes of persons who have asthma and are

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sensitive to house-dust mites should not be recommended routinely (Woodfolk et al. 1995). Vacuuming removes mite allergen from carpets but is inefficient at removing live mites.

2.7.1.6 Odors

The chemical identity of odors sometimes can be difficult to determine. Some common sources of odorous air pollution include sewage treatment plants, landfills, livestock feed lots, composts, pulp mills, geothermal plants, waste lagoons, tanneries, and petroleum refineries, among others. The odor of some compounds can be detected at level below those generally recognized as posing a significant health risk.

However, odors can negatively impact a person's quality of life and can exacerbate health problems among persons who are particularly sensitive to odors(Shusterman et al, 2001). Hydrogen sulfide (H_2S), a fairly prevalent odorous air pollutant, is emitted as part of a variety of industrial processes, including oil refining, wood pulp production, and wastewater treatment, as well as from concentrated animal feeding operations, geothermal plants, and landfills. Hydrogen sulfide, also known as sewer gas, has an odor similar to that of rotten eggs.

Routes of Exposure: The primary route of exposure is through inhalation. Substances released into the atmosphere, however, can enter the hydrologic cycle as a result of atmosphere dispersion and precipitation and thus contaminate aquatic ecosystems. Similarly, deposition of suspended particulate matter occurs in soil. Thus material that was originally released into the atmosphere can be ingested as a result of the sub sequent contamination of water, soil, or vegetation or the consumption of fish from contaminated waters. Some toxic air pollutants (such as mercury, lead, polychlorinated biphenyls, and dioxins) degrade very slowly or not at all and thus persist or accumulate in soil and the sediments of lakes and streams.

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2.7.1.7 Nitrogen dioxide

Use of unvented gas stoves and appliances results in increased indoor levels of NO_2. Use of gas stoves for cooking has been associated with increased respiratory symptoms, including wheezing in school children (Garrett et al, 1998; Withers et al, 1998) and increased prevalence of bronchial hyperresponsiveness in atopic adults (Kerkhof et al, 1999). However, data from the National Health and Nutrition Examination Survey III (NHANES III) did not suggest any impact of gas-stove use on pulmonary function or respiratory symptoms in adults who have asthma (Eisner and Blanc 2003). Infants at high risk for asthma who were exposed to higher levels of NO_2 but levels which currently are not considered to be harmful had increased days of wheezing and shortness of breath (Van Strien et al, 2004). In school-aged children, increased levels of NO_2 were associated with increased bronchitis, wheeze, and asthma in girls but not boys (Shima and Adachi, 2000). When unflued gas heaters in schools were replaced, NO_2 levels decreased by two-thirds, accompanied by significant reduction in both daytime and nighttime asthma symptoms (Pilotto et al, 2004). Exposure to gas heaters and appliances in infancy has been found to be a risk for wheezing, asthma, and bronchial hyperresponsiveness as well as sensitization to house-dust mites in school-aged children (Phoa et al, 2004; Ponsonby et al, 2000, 2001). Current use of gas appliances also was found to be a risk for decreased FEV1 in children sensitized to house-dust mites (Glasgow et al, 2001).

NO_2, a precursor of photochemical smog, is found in outdoor air in urban and industrial regions and, in conjunction with sunlight and hydrocarbons, results in the production of O_3. Automobile exhaust is the most significant source of outdoor NO_2, although power plants and other sources that burn fossil fuels also release NO_2 into the environment. Indoors, the most significant exposure to NO_2 occurs in conjunction with the use of gas cooking stoves and kerosene space heaters. Most ambient NO_2 is generated by the burning of fossil-derived fuels. Like O_3, NO_2 is an oxidant pollutant, although it is less chemically reactive and, thus, probably less potent.

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Outdoor levels of NO_2 are not usually associated with notable changes in bronchial function in asthmatic patients. Controlled exposure studies of subjects with asthma have produced inconsistent results regarding the ability of NO_2 to enhance nonspecific airway responsiveness, with some evidence of a subgroup with increased sensitivity (Hazucha et al, 1983; Bauer et al, 1986; Linn et al, 1986; Moshenin et al, 1987; Roger et al, 1990). Limited data from epidemiological studies suggest that exposure to high levels of NO_2 may be associated with acute decrements in lung function in subjects with asthma (Lebowitz et al, 1985; Goldstein et al, 1988). Two, controlled, human exposure studies, one with NO_2 alone and the other with a combination of NO_2 and SO_2, used pollutant *versus* sham pre-exposure followed by allergen challenge to investigate whether allergen-induced bronchoconstriction would be enhanced. All eight asthmatic subjects studied by Tunnicliffe et al,(1994) had a greater allergen-induced early bronchoconstrictor response after exposure to 0.4 ppm NO_2 for 1 h, while at rest, than after sham exposure. Seven of eight asthmatic subjects in a study by Devalia et al.(1998) had a lower PD_{20} after exposure to 0.4 ppm NO_2 or to 0.2 ppm SO_2, or to the combination of both pollutants for 6 h than after sham exposure. However, only the combined exposure caused a significant decrease in PD_{20} compared with the sham control. Neither study looked at the effects of pollutants on the late inflammatory response.

2.7.2 Outdoor precipitants

2.7.2.1 Outdoor allergens (tree, grass, and weed pollen; seasonal mold spores).

The strongest associations between mold-spore exposure and asthma have been with the outdoor fungi, such as *Alternaria* (Halonen et al, 1997; O'Hollaren et al, 1991; Targonski et al, 1995). Patients can reduce exposure during peak pollen season by staying indoors with windows closed in an air-conditioned environment (Solomon et al, 1980), particularly during the midday and afternoon when pollen and some spore counts are highest (Mullins et al, 1986). Conducting outdoor activities shortly after sunrise will result in less exposure to pollen. These actions may not be realistic for some patients, especially children.

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2.7.2.2 Outdoor air pollutants

Increased pollution levels, especially of particulate matter ≤10 micrometers (PM10) (Abbey et al. 1993) and ozone (Cody et al., 1992; Thurston et al, 1992;), but also of SO_2 (Moseholm et al, 1993) and nitric oxide (NO_2) (Kesten et al, 1995; Moseholm et al., 1993) have been reported to precipitate symptoms of asthma (Abbey et al, 1993; Moseholm et al, 1993;), and hospitalizations for asthma (Cody et al. 1992; Thurston et al, 1992).

High exposure to NO2 in the week before the start of a respiratory viral infection, at levels within current air quality standards, may increase the severity of virus-induced asthma exacerbations (Chauhan et al, 2003). Exposure to pollutants may increase airway inflammation (Hiltermann et al, 1999) and enhance the risk of allergic sensitization through simultaneous exposure to aeroallergens (Diaz-Sanchez et al. 1999; Fujieda et al. 1998; Jenkins et al. 1999). The propensity for particulate pollution to enhance allergic sensitization may be genetically regulated (Gilliland et al, 2004; Peden, 2005).

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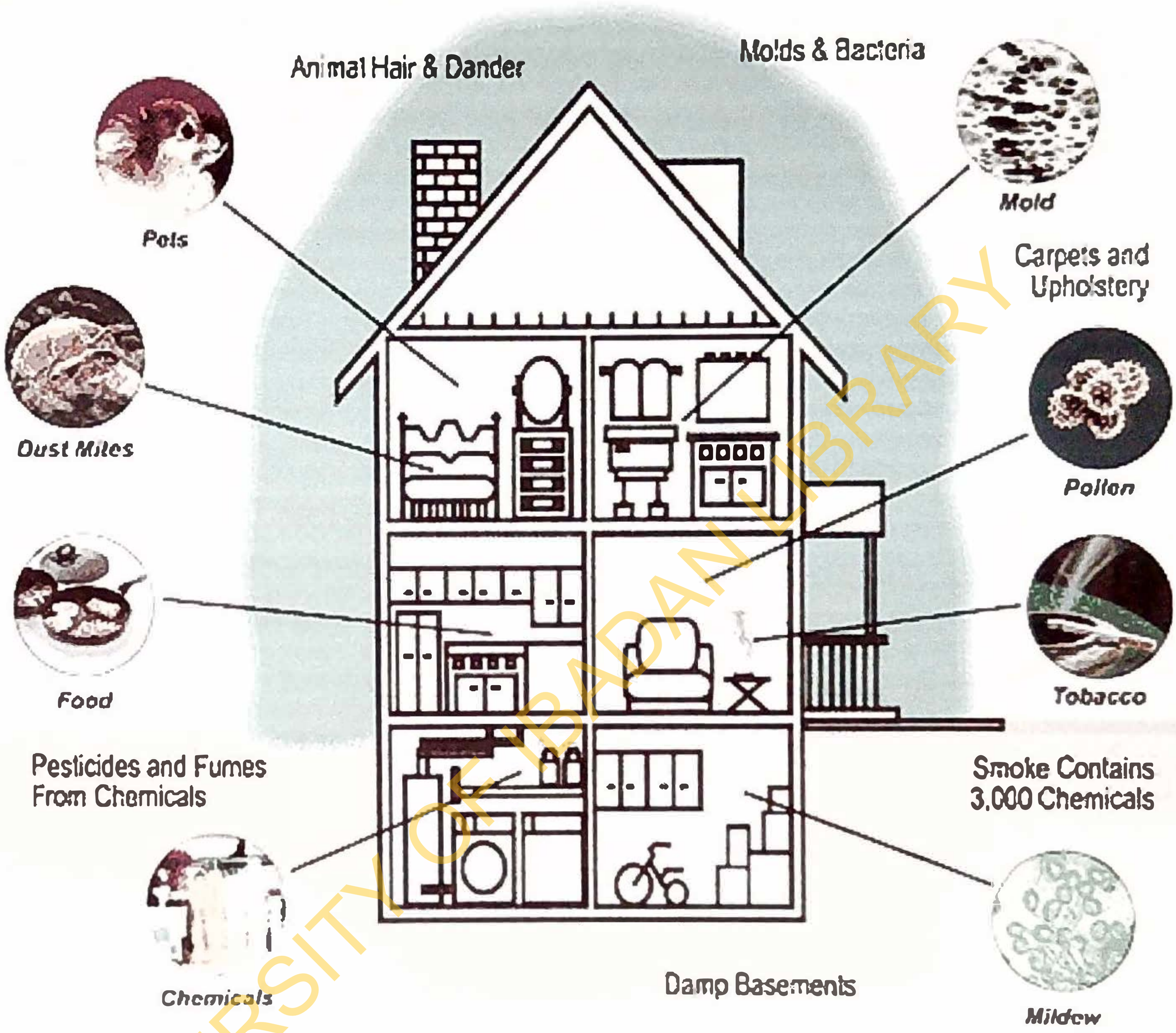


Figure 2.5: Examples of Indoor and Outdoor air pollutants

2.7.2.3 Particulate Matter

Particulate matter is a general term that refers to a mixture of solid particles and liquid droplets. The term is applied to pollutants of varying chemical composition and physical properties. Particles size is the primary determinant of where the particles will be deposited

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in the respiratory system. Particles larger than 10 um in aerodynamic diameter are too large to be inhaled beyond the nasal passages. Children, however, frequently breathe through their mouths, thus bypassing the nasal clearance mechanism.

All particulate matter less than 10 μm is aerodynamic diameter is known as PM 10. Particles of this size cannot be seen with the naked eye, but their presence in the atmosphere can be seen as sooty clouds or general haze that impairs visibility. Particulates smaller than 2.5 μm in aerodynamic diameter ($PM_{2.5}$) are referred to as "fine" particles.

Fine particles result from the combustion of fuels used in motor-vehicles, power plants, and industrial operations, as well as the combustion of organic material such as fires and woodstoves. Particles between 2.5 um and 10 um in aerodynamic diameter (or even larger) are referred to as "coarse" particles. Coarse particles can include dusts that have been generated from the mechanical breakdown of solid matter (such as rocks, soil, and dust) and windblown dust. Compared with coarse longer periods and be transported over longer distances. As a result, the concentration of fine particles tends to be more uniformly distributed over large urban areas, whereas the concentration of coarse particles tends to be more localized near particular sources. While fine and coarse particles have been linked with adverse health effects, recent evidence suggest that fine particles have stronger respiratory effects in children than coarse particles(WHO, 1999).

Airborne PM, which is a major component of urban air pollution, is a mixture of solid and liquid particles of different origin, size and composition, among which pollen grains and other vegetable particles carrying allergens and mould spores are included. Inhalable PM that can reach the lower airways is measured as PM_{10} and $PM_{2.5}$ (particles with an aerodynamic diameter <2.5 μm) (Churg et al, 1997; Brain et al, 1979; Anderson et al, 1994). Human lung parenchyma retains $PM_{2.5}$, while particles >5 μm and <10 μm only reach the proximal airways, where they are eliminated by mucociliary clearance if the airway mucosa is intact (Churg et al, 1997; Brain et al, 1979; Anderson et al, 1994).. PM is the most serious air pollution problem in many cities and towns and it appears to be the component of air

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pollution associated most consistently with adverse health effects. Particulate air pollution is significantly associated with enhanced mortality from respiratory and cardiovascular diseases, exacerbation of allergies, asthma, chronic bronchitis, respiratory tract infection and hospital admissions in many geographical areas (Pope et al, 1995; Katsouyanni et al, 1997; Salvi et al, 1999). Moreover, the World Health Organisation (WHO) estimates that inhalation of PM is responsible for 500,000 excess deaths each year worldwide 5. Adverse health events have also been observed in a range of air concentrations considered safe according to WHO guidelines.

Seaton *et al.*.(1995) hypothesised that fine PM found in urban areas is able to induce alveolar inflammation by penetrating deep into airways, which is responsible for variation in blood coagulability and release of mediators that induce acute episodes of respiratory and cardiovascular diseases. To explain the acute respiratory effects associated with inhalable PM, the same authors (Seaton et al, 1995) suggested that transition metals in the particles damage the airways, thereby, generating free radicals. In particular, iron, which generates hydroxyl radicals, seems to be responsible for the adverse respiratory effects (Smith et al, 1997; Ghio et al, 1993). Other transition metals (chromium, cobalt, copper, manganese, nickel, titanium, vanadium and zinc) derived from various urban or combustion source samples were also correlated to radical activation and lung injury in animal experiments (Donaldson et al, 1997; Costa et al, 1997; Dreher et al, 1997; Costa et al, 1998).

2.7.2.4 Ozone

O_3 is the most important factor in the so-called "summer smog", since it is the main component of photochemical oxidants and probably accounts for up to 90% of total oxidant levels in cities that enjoy a mild sunny climate, such as those of the Mediterranean area and California, USA, *etc.* O_3 is generated at ground level by photochemical reactions involving ultraviolet radiations on atmospheric mixtures of NO_2 and hydrocarbons derived from vehicle emissions. O_3 trends depend not only on substrate supply (NO_2 emitted by cars), but also on sunny weather that favours the transformation of NO_2 into O_3, thereby, producing photochemical smog. Current safety standards for O_3 levels are exceeded frequently in most

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Mediterranean countries. Approximately 40–60% of inhaled O_3 is absorbed in the nasal airways, the remainder reaching the lower airways.

Exposure to increased atmospheric levels of O_3 causes decrements in lung function, increased airway reactivity to nonspecific and specific bronchoconstrictor agents and is related to an increased risk of asthma exacerbation in susceptible asthmatic patients (Wjithermore et al, 1980; Holguin et al, 1985; Bates et al, 1983; Balmes et al, 1993; White et al, 1994; Thurston et al, 1997). Atmospheric levels of O_3 and NO_2 have been linked to increases in respiratory morbidity and in hospital admissions for asthma in children and adults (Atkinson etal, 1999; Peters et al, 1999). Bayram *et al.* (2001) demonstrated that O_3 and NO_2 modulate the airway inflammation of diseases, such as bronchial asthma, by increasing the release of inflammatory mediators from bronchial epithelial cells, and that the cells of asthmatic subjects may be more susceptible to the adverse effects of these pollutants. It has also been observed that O_3 exposure has a priming effect on allergen-induced responses as well as an intrinsic inflammatory effect in the airways of allergic asthmatics (Molfino et al, 1991; Peden et al, 1995; Jorres et al, 1996; Kerhl et al, 1999). Indeed, O_3 produces an immediate, dose-dependent increase in intracellular reactive oxygen species and in epithelial cell permeability, which could facilitate entry of inhaled allergens and toxins causing an increase in the release of inflammatory cells and their products (interleukin (IL)-1, -6, -8, tumour necrosis factor, *etc*). Moreover, in an animal study mucociliary clearance times decreased as O_3 increased(Schlesinger et al, 1987).

Since inhalation of O_3 by healthy subjects increases airway responsiveness and airway inflammation, asthmatic subjects were expected to be more sensitive to the acute effects of O_3(McDonnell et al, 1987;). Neurologically mediated inhibition of inspiratory effort involving C-fibres rather than bronchoconstriction has been proposed as the primary mechanism for O_3-induced decrements in forced expiratory volume in one second (FEV_1)(White et al, 1994;Coleridge et al, 1976;Coleridge et al, 1993). In other words, stimulation of bronchial C-fibres by arachidonic acid products, such as prostaglandin E_2, may account for changes in lung function due to O_3(Coleridge et al, 1976). Other pharmacological

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studies suggest mechanisms by which O_3 acts by altering lung function. Atropine inhibits O_3-induced decreases in airway resistance, suggesting a cholinergic action for this mechanism (Beckett et al, 1985). O_3 also increases sensitivity to inhaled methacholine (Holtzman et al, 1983;Folinsbee et al, 1994).

A study involving a high concentration of O_3 (0.4 parts per million (ppm)) and relatively heavy exercise (Kreit et al., 1989) and another involving prolonged exposure (7.6 h) to a lower O_3 concentration (0.16 ppm) (Horstman et al, 1995) showed enhanced responses in asthmatic subjects compared with healthy controls. Epidemiological studies have provided evidence that high ambient O_3 concentrations are associated with an increased rate of asthma attacksHolguin et al, 1985;White et al, 1994). Other studies have documented increased hospital admissions or emergency department visits for respiratory disease, including asthma, after days in which there have been high O_3 concentrations(Bates et al, 1983; White et al, 1994).

It has long been speculated that O_3 and other pollutants may render allergic individuals more susceptible to antigens to which they are sensitised, and animal data support such an effect(Yanai et al, 1990). Recently it has been observed that the incidence of new diagnoses of asthma are associated with heavy exercise in communities with high concentrations of O_3, thus, air pollution and outdoor exercise could contribute to the development of asthma in children (McConnell et al, 2002). However, O_3 can affect both the upper and lower respiratory tract and it induces more adverse effects in asthmatic individuals than in healthy subjects (Devalia et al, 1998).

The outcome of two, controlled, human exposure studies supports investigations in which O_3 exposure enhanced responses to inhaled antigens in animals. Thus, exposure to O_3 may increase the risk of allergic sensitisation in predisposed subjects. Indeed, by lowering the threshold concentration of allergen able to induce clinical symptoms, O_3 can enhance the airway responsiveness of sensitised subjects.

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2.7.2.5 Sulphur dioxide

SO_2 is generated primarily from the burning of sulphur-containing fossil fuel and is released into the atmosphere primarily as a result of industrial combustion of high sulphur-containing coal and oil. SO_2 has clearly been shown to induce acute bronchoconstriction in asthmatic subjects at concentrations well below those required to induce this response in healthy subjects (Sheppard et al, 1980;Linn et al, 1987). In contrast to O_3, the bronchoconstrictor effect of inhaled SO_2 in individuals with asthma occurs after extremely brief periods of exposure, especially with oral breathing and high ventilatory rates, as in exercise. While the data on responses of subjects with asthma to NO_2 exposure are inconsistent, there is no question that brief (*i.e.* <1 h) exposures to low concentrations of SO_2 can induce bronchoconstriction in such subjects (Horstman et al, 1986, Balmes et al, 1987).

Unlike pollutants such as NO_2 and O_3, SO_2 has a rapid effect on the lung function of asthmatic subjects, and significant responses are observed within 2 min; maximal response is seen within 5–10 minutes. There can also be spontaneous recovery (30 min after challenge) and a refractory period of up to 4 h, whereas repeated exposure to low levels of SO_2 results in tolerance to subsequent exposure. Pharmacological studies seem to suggest a cholinergically-mediated neural mechanism. However, the mechanisms by which SO_2 can induce asthma have yet to be completely clarified.

SO_2 exposure augments responses to other environmental agents that exacerbate bronchospasm. In this context, exposure of guinea pigs to as little as 0.1 ppm SO_2 enhanced allergic sensitisation to inhaled ovalbumin, as measured by the development of bronchoconstriction by specific inhalation challenge testing, and increased concentrations of specific antibodies in both BALF and serum (Reidel et al, 1988).

2.8 Climate change and asthma

There is still much to learn about the effects of other climatic factors that seem to be important to asthma, *e.g.* wind speed and passage of cold fronts. It is well known that

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inhalation of cold air reduces lung function in asthmatics, thus, favouring bronchoconstriction. Moreover, exercise in polluted areas results in greater deposition of air pollutants, including allergen-carrying allergens, in the lower airways. In fact, exercise increases oral breathing, total ventilation and inertial impaction of inhaled particles in the airways. There is also the thorny question as to how increasing levels of greenhouse gases and concomitant climate changes will influence the frequency and severity of pollen-induced respiratory allergy(D'Amato et al, 2000; Emberlin et al, 1998). A variety of direct and indirect evidence suggests that climate changes may affect pollen release and consequently pollen-related asthma (Frenguelin et al, 1998). Climate variations are likely to influence vegetation with consequent changes in growth, reproductive cycle, *etc.*, and in the production of allergenic pollen (seasonal period and intensity) with a greater proliferation of weed species. Climate changes vary from region to region; some areas will be subject to increases in ultraviolet radiation and/or rainfall frequency and other areas to reductions.

In Italy, in the 20 yrs from 1981–2000, the average mean temperature has increased by 0.6°C. This warming is accompanied by an average reduction of 15% in rainfall, and the rain is concentrated in a shorter period causing more violent rainstorms (Ambiente Italia, 2001). How are allergenic plants responding to these changes? The increased temperature in winter and spring has brought about early pollination, and the increased summer temperature has resulted in a prolonging of the pollination of herbaceous, allergenic plants such as Urticaceae. In warmer years,allergy symptoms started 1-2 weeks earlier. Pollen seasons, and therefore seasonal allergic symptoms, tend to be longer in warmer years. The prolonging of autumn could prolong the presence of fungal spores in the atmosphere. Because of the "urban climate effect" (heating caused by high building density and soil sealing), pollination can occur 2–4 days earlier in urban than in rural areas.

Vegetation reacts with air pollution over a wide range of pollutant concentrations and environmental conditions. Many factors influence the interaction, including type of air pollutant, plant species, nutrient balance, soil conditions and climatic factors. At low levels of exposure for a given species and pollutant, no significant effect is observed. However, as the

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exposure level increases, there may be biochemical alterations of the plants(Emberlin et al, 1998)Plants can absorb pollutants through the leaves or through the root system. In the latter case, deposition of air pollutants on soils can alter the nutrient content of soil in the proximity of the plant, thus, leading to indirect or secondary effects of air pollutants on vegetation. Metabolic variations affect the plant's structural integrity and there are probably changes in the pollen proteins, including those acting as allergens.

2.9 Allergenic precipitants

The association of asthma and allergy has long been recognized. Recent studies confirm that sensitization among genetically susceptible populations to certain indoor allergens such as house-dust mite, animal dander, and cockroach or to the outdoor fungus Alternaria is a risk for developing asthma in children (Sears et al, 1993; Sporik et al, 1990).

Sensitization to outdoor pollens carries less risk for asthma (Sears et al. 1989), although exposure to grass pollen has been associated with seasonal asthma(Reid et al., 1986). It is widely accepted that the importance of inhalant sensitivity as a cause of asthma declines with advancing age (Pollart et al., 1989).

An allergic reaction in the airways, caused by natural exposure to allergens, has been shown to lead to an increase in inflammatory reaction, increased airway hyperresponsiveness (Boulet et al., 1983; Peroni et al., 1994; Piacentini et al., 1993), and increased eosinophils in bronchoalveolar lavage (Rak et al., 1991). Other research has demonstrated that asthma symptoms, pulmonary function, and need for medication in mite-sensitive asthma patients correlate with the level of house-dust mite exposure (Custovic et al, 1998; Huss et al, 2001; Sporik et al. 1990; Vervloet et al., 1991) and that reducing house-dust mite exposure reduces asthma symptoms, nonspecific bronchial hyperresponsiveness, and evidence of active inflammation (Morgan et al, 2004; Peroni et al, 2002; Piacentini et al, 1993; Simon et al, 1994).

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Inhalant allergen exposure to seasonal outdoor fungal spores (O'Hollaren et al. 1991; Targonski et al. 1995) and to indoor allergens (Call et al., 1994) has also been implicated in fatal exacerbations of asthma. These reports emphasize that allergen exposure must be considered in the treatment of asthma.The important allergens for children and adults appear to be those that are inhaled. Food allergens are not a common precipitant of asthma symptoms. Foods are an important cause of anaphylaxis in adults and children (Sampson et al. 1992), but significant lower respiratory tract symptoms are uncommon even with positive double-blind food challenges (James et al., 1994). However, asthma is a risk factor for fatal anaphylactic reactions to food or immunotherapy (Bernstein et al. 2004; Reid et al., 1993).

2.9.1 Aeroallergens

Aeroallergens are ubiquitous, although quantitative and qualitative differences depend on geographic location, climate, degree of urbanization and specific conditions in the home, school and workplace. Almost all adults appear to have T lymphocytes that are sensitized to at least some aeroallergens; thus, development of allergic disease may depend on quantitative differences in T cells (Upham et al, 1995). Several lines of evidence link aeroallergens to asthma:(Working Group on Environmental Allergens and Asthma, American Academy of Allergy, Asthma and Immunology,1998).

- A total of 60% of adults and 80% of children with asthma have positive skin-prick tests for environmental allergens, and allergen-bronchial challenge tests are positive only in those with allergen-specific positive skin tests.
- Allergen sensitization is a risk factor for severe, acute asthma, especially if the patient is exposed to high concentrations of the specific allergen.
- In general, severity of symptoms and of bronchial responsiveness correlates with degree of sensitivity to allergens; in some patients, allergy does not play an important role.
- Symptoms, PEF and bronchial responsiveness usually improve when allergens to which the person is sensitized are avoided.

Aeroallergens, which are carried on inhalable particles, are proteins that vary in molecular weight from 14 to 78 kilodaltons. Outdoor allergens arise from pollen or mold spores, indoor

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allergen sources include several species of dust mites, cats, dogs and other mammals, cockroaches and indoor mold spores. The molecular structure and functional properties of common and important indoor allergens, based on the World Health Organization's nomenclature, have recently been summarized (Platts Mills et al, 1997). Recombinant allergens with immunoreactivity comparable to that of the natural allergens are being produced and evaluated for allergen standardization, for diagnostic testing and for immunotherapy with specific epitopes and naked DNA vaccines.

Infants are exposed and become sensitized to aeroallergens as well as food allergens in utero.(Holt et al, 1995; Warner et al, 1997). In people who are genetically predisposed to allergy. antenatal factors, including maternal and, thus, fetal exposure to allergens and materno-placento-fetal immunologic interactions are important in determining whether the predisposition results in allergic disease (Holt et al, 1996). Exposure to low concentrations of indoor allergens in early childhood is associated with a low incidence of sensitization, but very low concentrations may be sufficient to sensitize children who are predisposed and have a family history of allergy, presumably after intrauterine priming (Munir et al, 1997).

2.9.1.1 Aeroallergens as a risk factor for asthma

There are no reliable tests to detect which infants are at risk of developing allergic disease and asthma. A positive skin-prick test for egg protein, as a marker of specific immunoglobulin IgE antibody, at 6 months of age in a group of high-risk children (i.e., with a family history of atopy) was associated with development of atopic dermatitis, wheezy illness and asthma by age 7 years (Burr et al, 1997) and was consistent with earlier studies.(Hattevig et al, 1987; Zeiger et al, 1995; Sigurs et al, 1994). In a community study (Lombardi et al, 1997) of 360 children, the cumulative incidence of newly diagnosed asthma from 6 to 11 years of age was 12%. Bronchial hyperresponsiveness to cold air at age 6 years was associated with a 2.6-fold increase in risk (95% CI 1.25-5.4). However, after adjusting for mild wheezing at age 6 years, which is associated wit h an increased risk of 7.5-fold (95% CI 3.6-15.0, $p < 0.001$), and for positive skin-test reaction to inhalant allergens at age 6 years, which is associated with an increased risk of 3.6-fold (95% CI 1.5-9.5, $p < 0.01$), the

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response to cold air.was no longer a significant predictor. Therefore, hyperresponsiveness to cold air is associated with a subsequent diagnosis of asthma, but depends on the presence of atopy and prior mild wheezing.

Earlier studies, as indicated in the 1995 consensus statement,(Ernst et al, 1996) identified exposure to household dust mites and indoor animals, especially cats, as risk factors for asthma. A recent 12-month study (Rosentreich et al,1997) of 476 children with asthma, aged 4-9 years, living in inner city communities in the United States found that 36.8% of the children were allergic to cockroach allergen, 34.9% to dust-mite allergen and 22.7% to cat allergen. Analyses of dust showed that 50.2% of bedrooms contained high concentrations of cockroach allergen, 9.7% contained dust-mite allergen and 12.6% contained cat allergen. Adjusted rates of hospital admission were 0.37 a year for those who were allergic to and exposed to high concentrations of cockroach allergen compared with 0.11 for those allergic to other allergens ($p < 0.001$) and 2.56 unscheduled medical visits for asthma compared with 1.43 ($p < 0.001$). Those allergic to cockroach allergens experienced more days of wheezing, missed school days, night sleep loss and changes in activities than those allergic to dust-mite and cat allergens. This suggests that exposure to high concentrations of allergens in those allergic to a specific allergen is likely to enhance asthma morbidity.

2.9.2 Animal Allergens

Animal allergens are glycoprotein that often induces an IgE response in humans. These allergies usually are found in saliva, sebaceous glands (dog and cat), or sometimes in the urine (rodents). Allergies to cow hair or horsehair and dander also have been reported primarily through occupational exposures(Institute of Medicine, 2000). The spread of allergies in the environment has been studied primarily for cats, however the route of spread is likely to be similar for domestic furry animals (Institute of Medicine, 2000). Cat allergen-containing material dries and adheres to many surfaces (eg, animal fur or hair, bedding and clothing) and often can be transported to other environment via this route. Once an animal enters the room, small airborne allergen-containing particle (diameter <5 m) can be detected. These small particle remains suspended in the air for hours. Once the allergen-containing

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particle is inhaled, it is easily deposited in distal airways. Clinical manifestation of animal allergy range from mild cutaneous urticaria to rhino conjunctivitis to life-threatening bronchospam and anaphylaxis. Although symptoms may occur instantly following exposure, it is more common for upper and lower respiratory tract symptoms to develop within 30 minutes. Low level chronic exposures may not show clinically significant symptoms for several days.

2.9.2.1 Cats

The severity of allergic reaction to cats is greater than reaction to other common domestic pets. More than 6 million US residents have allergies to cats, and up to 40% of atopic patients demonstrate skin test sensitivity(Wood et al, 1993) . The major allergen Fel d I is present in high concentration in the saliva and sebaceous and anal glands of cats. The grooming habits of cats results in a large amount of saliva on the fur, and cat allergen can be spread via small airborne particles. Children with cats can transmit cat allergen to schoolrooms and may create an environment that can precipitate asthma in sensitized children(Luczynska et al, 1990). Once a cat is removed from an indoor environment, the allergen may persist for months.

2.9.2.2 Dogs

Dogs are the most common domesticated animal specie found in US homes. Five percent to 30% of atopic patients have a positive skin test to the major allergen Can f I, although many do not demonstrate clinical symptoms or have positive bronchoprovocation tests (Almquist et al 2001) . It seems that more variations in clinical sensitivity exist between dog breeds, and breeds-specific allergens have been suggested (de groot H et al, 1991). Nevertheless, no dog breed is considered nonallergenic. As with cat allergen, the highest concentrations of Can f I are found in canine fur and dander (Lindgren et al, 1988).

2.9.2.3 Rodents

Exposure to rodents can occur from their presence as pests or pets in the home. Rats and mouse allergens are produced primarily in urine (institute of medicine, 2000). Through

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transfer, the fur and dander often contain high amount of allergen. The prevalence of mouse allergens can be widespread in inner-city homes (Phipatanakul et al, 2000). Among inner-city children with asthma, there was an association between mouse allergens in house-dust sample and sensitization to mouse allergen, especially among those asthmatics with atopy to multiply allergens on skin testing. The relationship between mouse allergen exposure and asthma morbidity was less clear (Phipatanakul et al, 2000).

2.9.2.4 Birds

In the occupational setting, hypersensitivity pneumonitis can be associated with antigens from bird excreta and proteinaceous material found in dust dispersed from bird; however, it is unclear whether birds cause allergy and asthma (Institute of medicine, 2000). Large quantities of dust mites have been documented in feathers, and dust mites allergen is the likely source of the allergic stimulus from feathers-containing items in the home, including pillows, comforters, bedding, and down-filled clothes (Institute of medicine, 2000).

2.9.2.5 Cockroach

Cockroach sensitivity and exposure are common among patients who have asthma and live in inner cities (Call et al., 1992; Gelber et al., 1993; Huss et al., 2001; Kang et al., 1993). In a study of asthma in an inner-city area, asthma severity increased with increasing levels of cockroach antigen in the bedrooms of children who were sensitized (Rosenstreich et al., 1997). Another major study demonstrated efficacy of cockroach avoidance as part of an overall plan for allergen avoidance (Morgan et al. 2004). Patients should not leave food or garbage exposed. Poison baits, boric acid, and traps are preferred to other chemical agents, because the latter can be irritating when inhaled by persons who have asthma. If volatile chemical agents are used, the home should be well ventilated, and the person who has asthma should not return to the home until the odor has dissipated. Care should be taken so that young children do not have access to cockroach baits and poisons.

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2.10 Obesity and asthma

Obesity has been associated with asthma persistence and severity in both children and adults (Camargo et al, 1999; Schaub and von Mutius, 2005; Shore and Fredberg, 2005; Weiss, 2005; Weiss and Shore, 2004). Although obesity itself causes alterations in pulmonary physiology that can lead to dyspnea, studies have documented specific increases in asthma among overweight and obese persons.

Increased risk from obesity appears to be greatest in postpubertal women and is associated with more severe symptoms, enhanced airway inflammation, and new-onset or persistent disease (Camargo et al., 1999; Guerra et al., 2004). Presently, the relationship of obesity to allergy is controversial. The effects of obesity on asthma appear to be independent of diet and physical activity, although these three factors are clearly interrelated. Many epidemiologic studies have controlled for potential effects of diet and physical activity when examining the relationship of obesity to asthma onset (Camargo et al., 1999). The few RCTs that have been done are small, but they show that weight loss in adults resulted in improvement in pulmonary mechanics, improved FEV1, reductions in exacerbations and courses of oral corticosteroids, and improved quality of life (Stenius-Aarniala et al., 2000). Weight loss following gastric bypass surgery improved self-reported asthma severity (Simard et al., 2004).

2.11 Stress, depression, and psychosocial factors in asthma

The role of stress and psychological factors in asthma is important but not fully defined. Emerging evidence indicates that stress can play an important role in precipitating exacerbations of asthma and possibly act as a risk factor for an increase in prevalence of asthma (Busse et al., 1995; Sandberg et al., 2005; Wright et al., 2002). Chronic stressors increase the risk of asthma exacerbations, especially in children who have severely negative life events and those who have brittle asthma (Miles et al., 1997; Sandberg et al., 2000). The mechanisms involved in this process have yet to be fully established and may involve enhanced generation of pro-inflammatory cytokines (Friedman et al., 1994). In a prospective study of a birth cohort predisposed to atopy, higher caregiver stress in the first 6 months after

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birth was significantly associated with an increased atopic immune profile in the children (high total IgE level, increased production of tumor necrosis factor-alpha (TNF-α) and a suggested trend between higher stress and reduced interferon-gamma (IFN-γ production) (Wright et al. 2004a). Equally important are psychosocial factors that are associated with poor outcome (e.g., conflict between patients and family and the medical staff, inappropriate asthma self-care, depressive symptoms, behavioral problems, emotional problems, and disregard of perceived asthma symptoms) (Brush and Mathé 1993; Strunk et al., 1985; Strunk 1993).

Asthma severity can be affected by personal or parental factors, and both should be evaluated in cases of poorly controlled asthma. For example, maternal depression is common among inner-city mothers of children who have asthma and has been associated with increased ED visits and poor adherence to therapy by these children (Bartlett et al., 2001, 2004). Furthermore. in a large prospective study of inner-city children who had asthma, increased exposure to violence, as reported by caretakers, predicted a higher number of symptom days in their children, with caregivers' perceived stress mediating some, although not all, of this effect (Wright et al.,2004b). It may also be important to evaluate psychosocial and socioenvironmental factors in children who have repeated hospitalizations; however, it is not clear whether psychosocial factors affect or result from the frequent hospitalizations (Chen et al., 2003).

2.12 Medication sensitivities and asthma

Alternatives to aspirin that usually do not cause acute bronchoconstriction in aspirin-sensitive patients include acetaminophen (7 percent cross-sensitivity) (Jenkins et al, 2004), salsalate (Settipane et al, 1995;) or the COX-2 inhibitor celecoxib (Gyllfors et al, 2003). Aspirin desensitization treatment, followed by daily aspirin, is a potential option to decrease disease activity and reduce corticosteroid requirements (Berges-Gimeno et al, 2003a,b). As many as 21 percent of adults and 5 percent of children who have asthma have aspirin-induced asthma, especially when identified through oral provocation testing rather than verbal history (Jenkins et al, 2004).

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2.12.1 Beta-blockers

Nonselective beta-blockers can cause asthma symptoms (Odeh et al, 1991), although cardioselective beta-blockers, such as betaxolol, may be tolerated (Dunn et al, 1986). A recent systematic review, primarily of single dose or short-term studies in younger subjects, indicates that patients who have mild to moderate airway obstruction can tolerate cardioselective beta-blockers; therefore, if needed for managing cardiovascular disorders, these agents may be administered after careful evaluation (Salpeter et al, 2002).

2.12.2 Sulfite sensitivity

Sulfites which are used as preservatives in processed potatoes, shrimp, dried fruit or drinking beer or wine have been known to be associated with asthma symptoms.. Sulfites have caused severe asthma exacerbations, particularly in patients who have severe persistent asthma (Taylor et al., 1988).

2.13 Infections and asthma

2.13.1 Viral Respiratory Infections

It is well established that viral respiratory infections can exacerbate asthma, particularly in children under age 10 who have asthma (Busse et al., 1993). Respiratory syncytial virus (RSV), rhinovirus, and influenza virus have been implicated (Busse et al., 1993), with rhinovirus being implicated in the majority of the exacerbations of asthma in children (Johnston et al., 1995). The role of infections causing exacerbations of asthma also appears to be important in adults (Nicholson et al., 1993). Rhinovirus infections in patients who have asthma may induce exacerbations due to abnormalities in epithelial cells' innate immune responses to infection (Wark et al., 2005).

Viral infections are the most frequent precipitants of wheezing during infancy and asthma exacerbations during childhood. Many infants and toddlers who wheeze with viral infections are predisposed to have bronchial obstruction during these illnesses because of very small airway size (Martinez et al., 1995), and they will not have further exacerbations during later childhood. However, chronic asthma also may start as early as the first year of life among

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infants who have a family history of asthma, persistent rhinorrhea, atopic dermatitis, or high IgE levels. Early identification of these infants would allow institution of environmental controls to reduce exposure to tobacco smoke, animal dander, and house-dust mites and, thus, potentially reduce symptoms. RSV infections severe enough to require hospitalization during infancy and early childhood may be a risk factor for subsequent chronic asthma (Sigurs et al. 2005).

In more recent years RT-PCR assays, which are extremely sensitive in detecting respiratory viruses, have been developed (Johnston et al, 1995). These techniques have been applied in studies investigating the relationship between viral infections and asthma severity. In a longitudinal study of 138 adults with asthma, nose and throat swabs and blood samples were collected to coincide with either symptoms of acute upper respiratory tract infection or worsening of asthma (Nicholson et al, 1993). One-quarter of confirmed viral infections were associated with mean decreases in peak expiratory flow rate (PEFR) of 50 ml/minute or more, and one-half were associated with mean decreases in PEFR of 25 ml/minute or more. Respiratory pathogens were implicated in almost half of all severe asthma exacerbations (decrease in PEFR of 50 ml/minute or more), and the most commonly identified virus was RV.

Viral respiratory infection is a well known provocative factor for episodes of asthma. As well, specific agents, including respiratory syncytial virus (RSV), adenovirus, mycoplasma and pertussis, can provoke episodes of wheezing illness and, in a few cases, prolonged bronchial hyperresponsiveness. Recent studies using polymerase chain reaction (PCR) have implicated human rhinoviruses (HRV) as important agents in all age groups, and one study using this technique suggested a high prevalence of chronic Chlamydia infection in asthmatic children (Johnston et al, 1997).

How viruses or other agents provoke asthma is not clear. There is evidence of increased IgE production during viral infection. A recent study(Rager et al, 1998) using a human B-cell culture system found that HRV-induced, double-stranded RNA activates an antiviral protein

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kinase that can induce Ig class switching to IgE, suggesting a mechanism for viral provocation of allergy and asthma. This is consistent with a study(Grunberg et val, 1997) of experimental HRV infection in asthmatic adults, which resulted in augmented eosinophilic inflammation (assessed in sputum) and enhanced bronchial responsiveness.

In another controlled study (Geru et al, 1997) of experimental HRV infection in people with allergic rhinitis (but no asthma) and a nonallergic control group, there was a significant increase in bronchial responsiveness to histamine in the allergic group. Rhinovirus infection of cultured human tracheal epithelium, confirmed by PCR, resulted in increased expression (up-regulation) of messenger RNA for intercellular adhesion molecule-1 (ICAM-1) mRNA (the major HRV receptor on epithelial cells) and increased secretion of IL-1b, which itself up-regulates ICAM-1. Because ICAM-1 has important eosinophil attractant properties, this may be an important way in which the bronchial airway inflammatory response may be increased by HRV infection in asthma (Terajima et al, 1997). RSV infection accompanying bronchiolitis is associated with persistent bronchial hyperresponsiveness in some children, but its role in causing asthma is unclear. Recent animal studies suggest that RSV infection in mice followed by aeroallergen exposure results in pulmonary inflammation with eosinophilic infiltration;(Schwarze et al 1997), in guinea pigs, prior sensitization to allergen followed by infection with RSV results in much more severe mucosal damage(Robinson et al, 1997).

Viruses are of greatest importance in causing wheezing illness in children under the age of 3 years. Reports from several centres (Wilson et al, 1994;Martinez et al, 1995) now confirm that 20% or more of infants in this age group respond to viral infections with recurring wheezing, which resolves in later childhood. These infants have reduced lung function before the onset of viral infection, have apparently normal immune responses to viral infection and do not have risk factors for asthma (i.e., increased IgE levels, bronchial hyperresponsiveness or a family history of asthma). They may have narrower intrapulmonary airways than normal infants. A second group, about 10% of wheezy infants, also wheeze with virus infections, have some or all of the risk factors for asthma and have recurring wheeze (asthma) in later

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childhood (Martinez et al, 1997). There is a great need to develop tests that will accurately differentiate these 2 populations.

2.13.2 Bacterial Infections

Recent studies in both children and adults suggest that infections with both *Mycoplasma* and *Chlamydia*, in addition to viral infections, may contribute to exacerbation rates and disease chronicity and severity (Cunningham et al., 1998; Esposito et al., 2000; Kraft et al., 2002). Studies to confirm and expand upon these initial observations have been impeded due to the lack of definitive serologic markers to document current or past infection, as well as the inherent difficulties in obtaining biologic specimens from the lower airway to confirm the presence of these infectious agents (Martin et al., 2001).

2.13.3 Influenza Infection

Recent evaluations in both children and adults have yielded inconsistent and unconvincing results regarding the ability of influenza vaccination to reduce either overall rates of asthma exacerbations or exacerbations specifically related to influenza infection during the influenza season (Abadoglu et al., 2004; Bueving et al., 2004; Cates et al. 2004; Kramarz et al., 2001). The Advisory Committee on Immunization Practices recommends inactivated influenza vaccine for persons who have chronic disorders of the pulmonary systems. including asthma. because they are considered to be at increased risk of complications from influenza, such as hospitalizations
and increased requirements for antibiotics (CDC 2006). Administration of partially inactivated influenza vaccine is safe in both adults and children who have asthma (American Lung Association Asthma Clinical Research Centers 2001).

Vaccination with cold-adapted, live, attenuated influenza vaccine has also been demonstrated to be safe in school-aged, adolescent, and adult patients who have asthma (Belshe et al., 2004). However, the observation of an increased risk of asthma/reactive airway disease in children <36 months of age is of potential concern (Bergen et al., 2004). In patients who have documented histories of anaphylactic reactions after ingestion of egg protein and documented

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evidence of current allergic sensitization to eggs (skin testing or in vitro antigen-specific IgE antibody testing), the risk/benefit ratio of administration of influenza vaccine should be reviewed carefully. If the decision is made to administer the live, attenuated vaccine, a subspecialist familiar with appropriate challenge testing and published safe administration protocols should be consulted prior to administration (Zeiger, 2002).

2.14 Female hormones and asthma

There is considerable interest in the effects of female hormones on asthma severity. Studies are not totally concordant in their findings, but most evidence suggests that some women have worsening of their asthma during the premenstrual and menstrual times of the cycle (Haggerty et al, 2003; Shames et al, 1998). Two ED studies, however, suggest that many women experience asthma exacerbations during the preovulatory phase (Brenner et al, 2005; Zinmerman et al, 2000). Studies on hormone replacement therapy (HRT) after menopause also demonstrate apparent discordance. A cross-sectional study reported better pulmonary function and less frequent asthma exacerbations (Kos-Kudla et al, 2001), whereas a prospective cohort study found higher risk of adult-onset asthma (Barr et al, 2004). Although associations between female hormones and asthma severity are not uniform or clear, it may be useful for clinicians, as they develop action plans with their patients, to appreciate the role that female hormone levels may have in the course of asthma.

2.15 Asthma and diet

Patients have great interest in whether dietary factors may influence the onset, persistence, or severity of asthma. Although people who have asthma frequently experience bronchoconstriction as part of an acute IgE-mediated reaction to a food, food allergy is rarely the main aggravating factor in chronic asthma in children and even more rarely in adults (Sampson,2003). Preliminary evidence suggests that antioxidant vitamins (Currie et al, 2005; Devereux et al, 2002; Kaur et al, 2001; Martindale et al., 2005; McKeever et al., 2004; Pearson et al, 2004; Shaheen et al, 2001) and omega-3 fatty acids (Broadfield et al, 2004; Dunstan et al, 2003; Kompauer et al., 2004; Mihrshahi et al., 2003, 2004; Woods et al, 2004)

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reduce asthma development and symptom severity, but no conclusive evidence shows that any dietary factors prevent or exacerbate the disease.

First, some foods can provoke asthma attacks by causing an allergic reaction. Foods that trigger attacks (according to your susceptibility), can be: Foods containing the additives benzoates (E210-19), sulphites (E220-8) orgallates, (E310-12) Cider,wineandbeer. Foods containing yeast or mould, such as bread and blue cheeses. Foods, drinks and snackscontainingcolorings E102, E104 and E110 Cow's milk, cereals (wheat), eggs, fish, soy, and nuts (especially peanuts).

Secondly, food can also help to control the severity of an attack. One of the best-known foods for doing this is coffee, due to the caffeine. Some Foods can actually dilate air passageways, by thinning the mucus and opening them up for freer breathing. The foods in this category include the spicy, pungent foods like chili, hot mustard, garlic and onions. These hot foods work by stimulating nerves, resulting in the release of watery fluid in the mouth, throat and lungs.

Thirdly, some foods can control inflammation of the airways because of their anti-inflammatory components. Foods that help to do this include onions (these are particularly good), fatty fish (fish oil is a proven anti-inflammatory high in omega 3 fatty acids) and vitamin C-packed foods.Because an allergy is highly individual condition, it is not possible to give a definitive list of 'good' and 'bad' foods. Those who suspect that foods are triggering their asthmatic attacks should keep a food diary and seek expert medical help.

Common foods that may trigger asthma in susceptible individuals, however, include cow's milk, wheat and other cereals, yeast and foods containing mould, such as blue cheeses. Nuts (especially peanuts), fish and eggs can produce the most immediate and dangerous reactions. Certain food additives may also be potential triggers in some people. The presence of sulphites must, by law, be indicated on the label of packaged foods, but it is not obvious when such foods are served in delicatessens or restaurants, where of course they do not have

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any labels. Sulphites are used in cider, beers and wines. These products may pose a risk for susceptible asthmatics when, for instance, they inhale the bouquet of a wine.

Perhaps as a result of bacterial activity during production, some wines also contain levels of histamine that may occasionally prompt asthmatic reactions in susceptible people. Benzoate preservatives, which are present in a range of products from soft drinks and reduced-calorie jams to chewing gum and fish roe, may bring on asthmatic attacks. Other potential triggers include the less common antioxidant additives - the gallates(E310-12), butylated hydroxyanisole (BHA or E320), and also butylated hydroxytoluene (BHT or E321) - which are used in certain fats and oils, as well as in some breakfast cereals. In susceptible people, the food colourings E 102 (tartrazine, yellow), #104 (quinoline yellow) and E110 (sunset yellow) may also trigger asthma. Under EU legislation the presence of all these additives must be indicated on food labels.

2.16 Genetics and asthma

2.16.1 Role of genetics in the development of asthma

Asthma runs strongly in families and is about half due to genetic susceptibility and about half due to environmental factors (2000 Duffy et al, 1990). The strong familial clustering of asthma has encouraged an increasing volume of research into the genetic predisposition to disease. Although identification of all asthma genes is incomplete, genetic findings are already changing the prevailing view of asthma pathogenesis.

Positional cloning is a process of systematic disease gene identification that begins by finding genetic region co-inherited with disease. It requires no assumption about likely disease pathogenesis. Five asthma genes or gene complexes have now been identified by positional cloning, including ADAM33, PHF11, DPP10, GRPA, and SPINKS(Zhang et al, 2003; Allen et al, 2003; Laitinen et al, 2004; Walley et al, 2001; Vvan Eerdeweegh et al, 2002). The functions of all of these genes are obscure but the expression of DPP10, GRPA, and SPINKS in terminally differentiating epithelium suggests that they deal with threat or damage from the external environment (Cookson et al, 2004). Many of the genes identified

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by candidate gene studies may also exert their effects within the cells that make up the mucosa. These include IL13 which modifies mucus production, FcsRI-β which modifies the allergic triggers on mast cells and microbial pattern recognition receptors of the innate immune system(Cookson et al, 2004).These findings all suggests that the airway epithelial barrier and its reaction to the microbial environment contain the most important element of asthma Pathogenesis.

2.16.2 Role of genetics in the management and prevention of asthma

It is to be hoped that genetic findings will lead to a better classification of complex diseases such as asthma and that novel therapies will result from genetic findings. Most polymorphisms so far identified do not seem to carry risks that would merit their use for the clinical classification of diseases, but combination of genetic polymorphisms may be much more informative. It is also to be hoped that genetic findings may help identify the environmental factors that protect against asthma. In this context, association between asthma and innate immune system receptors for microbial products are particularly existing. To become relevant to clinical asthma, potential asthma susceptibility genes now need to be tested in cases and controls with different manifestations of disease and diseases severity and in representative population samples with environmental risk factors. Several of the asthma susceptibility genes so far identified are potential targets for asthma therapy. However it will take some years to determine if any of these will be the basis for new treatments.

Polymorphisms may also predict the response to asthma therapy. A positive association between common arginine-16 variants in the β-adrenergic receptor gene and the responsiveness of asthmatic patients to β-adrenergic agonists is particularly interesting (Israel et al, 2000). It is not known whether these differences in response represent a failure of β-agonist in individuals carrying the arginine -16 genotype or whether therapy in these individuals will be adequate with an upward adjustment of dose. A proportion of individuals with severe intractable asthma do not respond to inhaled steroids. It is possible that these individuals also carry mutation in some of the genes that control the anti- inflammatory response of the steroids. The pathways are not completely understood and is not yet known if

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genetic testing will be helpful in these circumstances. Gene therapy is of use in some single gene disorder but gene therapy can only be administered with arduous clinical protocols and it is not without risk of serious side effects such as leukaema. It is therefore unlikely that gene therapy will be applied to asthma or related disorder in the foreseeable future.

2.17 Management of asthma

Immunotherapy is usually reserved for patients whose symptoms occur all year or during a major portion of the year and in whom controlling symptoms with pharmacologic management is difficult because the medication is ineffective, multiple medications are required, or the patient is not accepting the use of medication. Reports, however, that immunotherapy can prevent the development of new sensitivities in monosensitized children and adults (Des Roches et al, 1997; Pajno et al, 2001; Purello-D'Ambrosio et al, 2001) and that immunotherapy with birch and timothy pollen extracts can prevent the development of asthma in children who have allergic rhinitis (Moller et al, 2002), along with evidence of persisting effect for at least 3 years after discontinuation (Durham et al, 1999), suggest that immunotherapy should be considered when there is a significant allergic contribution to the patient's symptoms.

Controlled studies of immunotherapy, usually conducted with single allergens, have demonstrated reduction in asthma symptoms caused by exposure to grass, cat, house-dust mite, ragweed, *Cladosporium*, and *Alternaria* (Reid et al, 1986). A meta-analysis of 75 randomized, placebo-controlled studies has confirmed the effectiveness of immunotherapy in asthma, with a significant reduction in asthma symptoms and medication and with improvement in bronchial hyperreactivity (Abramson et al., 2003). This meta-analysis included 36 trials for allergy to house dust mites, 20 for pollen allergy, and 10 for animal dander. On the other hand, only three trials for mold allergy and only six trials with multiple allergen therapy were included.

In the United States, standardized extracts are available for house-dust mites, grasses, short ragweed, and cat, and there are unstandardized extracts of other pollens and for dog that

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appear to have similar potency (Nelson, 2007). Available extracts for cockroach and mold, on the other hand, are of very variable allergen content and allergenic potency, and their effectiveness in specific immunotherapy has not been demonstrated (Nelson, 2007).

2.18 Primary prevention of allergic sensitization and asthma

Primary prevention of asthma—preventing initial development—is an active area of investigation. Although a number of trials have investigated dietary and environmental manipulations as preventive measures for asthma and allergy, clinical trials have not been uniform in their approaches, making firm conclusions difficult. Also, most of these interventions have been evaluated over a relatively short period of time, thus limiting their weight for any long-term implications.

Evaluations of dust-mite mitigation in homes of children of atopic parents show effectiveness of interventions in decreasing dust-mite levels as well as decreased incidence of wheezing (Custovic et al., 2001). Prospective assessment of dust-mite reduction and cow's milk avoidance (breastfeeding or hydrolysate) appears to show protective effects at 8-year followup (Arshad et al., 2003), while breastfeeding, dust-mite and pet avoidance, and tobacco smoke avoidance were protective at 7-year followup (Chan-Yeung et al., 2005).

Trials evaluating breastfeeding have generally shown protective benefit (Chandra, 1997), although there are conflicting studies (Wright et al, 2001). Pet exposure as preventive or provocative is controversial (Celedon et al. 2002)

Several recent studies have suggested that acetaminophen may contribute to the pathogenesis of asthma and asthma-related symptoms. The effect has been observed in both children and adults in population-based, birth-cohort, and case-control studies. A comprehensive review of this topic has been published (Eneli et al, 2005). However, one potential limitation of many studies on intake of commonly available over-the-counter analgesics, such as acetaminophen, is the potential for confounding by indication (Signorello et al, 2002). In

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summary, preliminary evidence appears to indicate a possible association between acetaminophen intake and wheeze, but the data are limited and potentially confounded. Although choice of analgesic/antipyretic should always be made carefully, at the current time, it would be premature to recommend avoidance of acetaminophen.

Exposure to daycare in early childhood may be beneficial, while tobacco smoke exposure both in utero and in early childhood is a risk factor for asthma (Gilliland et al, 2001). Larger family size may be preventive, with the incidence of asthma decreasing with an increasing number of siblings (Bodner et al, 1998; Mattes et al, 1999; Rona et al, 1997). The weight of evidence regarding larger family size, daycare exposure with more likelihood of respiratory infection, and country living is in keeping with the hygiene hypothesis of the origin of atopy and asthma. This hypothesis purports that more developed societies are more prone to higher incidence of allergy and asthma because their cleanliness downregulates immune processes for fighting infection in favor of those that cause atopic disease. Rural lifestyle may be protective compared to urban living (Bibi et al, 2002; Kauffmann et al, 2002).

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CHAPTER THREE

METHODOLOGY

3.1 Study Design

The study design was an observational case control study in which diagnosed asthmatic cases in the children Outpatient clinic of University College Hospital, Ibadan and Oni memorial children Hospital, Ibadan were selected as cases while children attending the same clinic but not asthmatic were selected as the controls. Both primary and secondary data were collected from both the cases and controls.

3.2 Description Of The Stu dy Area

The study area is Ibadan, Nigeria. Nigeria is a large country with a population of 120 million people spread across a surface area of approximately 923,7659sqkm. It is the tenth largest country in Africa and most populous with almost one in every five African, a Nigerian (Ivbijaro, 2002). It is diverse in its geography, ecology, culture, language and pattern of health and disease (Hamilton and Olaloye, 1991).

Ibadan is the capital of Oyo State located on the South Western part of Nigeria, on longitude 3° 5 of the Greenwich meridian and latitude 7^{0} 25 north of the equator at a distance of some 145km. The city of Ibadan covers a total area of 160.45sqkm from 1993 estimates with11 local governments administrative areas, 5 within the metropolis and 6 in the peripherals with a mixture of urban and rural communities. It is the largest city in West Africa. The climate is made up of two seasons: Rainy season which begins from April to October and Dry season which commences from November to March. Geographically it is located in the tropical rain forest zone.

Ibadan presents fascinating landscape of traditional and modern features Prominent in its old, indigenous core area are the Iba's marked (Oja'ba) and the king's place. From Mapo Hill,

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one can easily see the sea of rusted brown roofs and buildings in places like Agugu, Ayeye, Idi Arere, Odinjo, Gege and Foko, to mention but a few. These are interspersed by neighborhoods, of new and modern buildings, which are linked and crisscrossed by winding roads. Other places are at the periphery of the core centre. They include Odo Ona, Apata Ganga and Owode Estate, to the west, to the south are Challenge, Molete and Felele areas. Those to the north are Orogun, Ojoo, Sasa etc, while to the north – east are Bodija, Akobo, Monatan and Isebo.

With its strategic location on the railway line connecting Lagos to Kano, the city is a major center for trade in cassava, cocoa, cotton, timber, rubber, and palm oil. The main industries in the area are tire rethreading, cigarettes manufacturing and the processing of agricultural products; including flour-milling, leather-working and furniture-making. The largest companies with major infrastructure based in Ibadan are Kakanfo Inn , Coca-Cola , Nigerian Breweries , Galaxy Television, Globacom , NTA Ibadan , and Zartech Limited. There is abundance of clay, kaolin and aquamarine in its environs, and there are several cattle ranches, a dairy farm as well as a commercial abattoir in Ibadan. Ibadan has a few other important industries establishment like the confectionaries, oil processing plants, soft drinks, bottling and food factories, feed mills, tobacco factory and flour mills. Other are sawmills, paper mills, foam products, concrete poles and block making, chemicals, paints and petroleum oil deport.

3.3 Study Population

The study group consists of 64 children within the age range of 3-15 years. These children were selected from University College Hospital, Ibadan and Oni Memorial; Children Hospital. They were classified into 32 cases and 32 controls. The cases were selected from children attending the children outpatient clinic of these selected health institutions that have been diagnosed of asthma. The controls were selected from children attending the same clinic having other diseases except asthma.

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3.3.1 Selection Criteria

Inclusion criteria for the cases:

- The participants must be between the ages between 3-15 years,
- The participants must be diagnosed asthmatic cases residing in Ibadan

Exclusion criteria for the cases:

- The participants that were aged less than 3 and greater than 15,
- Must not be diagnosed asthmatic and not residing in Ibadan.

Inclusion criteria for the controls:

- ✓ children aged 3-15 years
- ✓ non- asthmatic residing in Ibadan

The exclusion criteria for the controls :

- ✓ children aged less than 3 and greater than 15,
- ✓ asthmatic and not residing in Ibadan.

3.4 Calculation Of Sample Size

Using the formular below, the required sample size was calculated as follows:-

$$N = \frac{2(Z\alpha+Z\beta)^2\ \sigma^2}{(N_1-N_2)^2}$$

Where $Z1-\alpha/2$ =Standard Normal deviation at 5% level of significance(2 sided =1.96)

$Z1-\beta$= Standard Normal Deviation at 9% power = 1.28

σ =Standard Deviation of the indoor air pollutant(1.19 for NO_2 from a previous study(V.N Mbanya, 2003)

N_1-N_2= mean difference between cases and controlswhich the study want to detect= 1ppm

$$n = \frac{2(1.96+1.28)^2\ (1.19)^2}{1}$$

=29.73

=30.

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3.5 Data Collection

3.5.1 Identification of subjects

The cases which comprised of children aged between 3-15 years were selected from among the children attending the children outpatient clinics of University College hospital, Ibadan and Oni memorial Children Hospital, Ring road Ibadan. The controls were also selected from among the children attending the same clinic from the same health institutions but these were made up of children having other diseases but not diagnosed asthmatic. Both the cases and controls were recruited from the hospitals after which they were followed to their various homes for environmental assessment.

3.5.2 Questionnaire survey

The parents of these children were interviewed using a validated questionnaire which was interviewer administered. This questionnaire which was developed in English was translated to Yoruba and again to English to ensure uniform questioning variables and to facilitate easy understanding of the questions. The questionnaire was divided into five sections viz: socio demographic characteristics, family and social history, knowledge of asthma, environmental health characteristics, health status and conditions. Most of the questions were formal with both open and close ended items. The questionnaires were pretested in the Federal Medical Centre, Abeokuta after which necessary corrections were made. The pretesting was necessary to ensure validity and reliability of the instruments.

3.5.3 Determination of air quality

The subjects were followed to their homes where the quality of air was determined using Dust track for Suspended Particulate Matter(SPM) and Digital gas meters for Carbon monoxide (CO), Nitrogen dioxide (NO_2) and Sulphur dioxide (SO2). The instruments were zeroed outside the specific locations and calibrated before use. The concentrations of the gases were in parts per millions (ppm) while the concentration of suspended particulate matter was in $\mu g/m^3$. Measurements were made in three locations inside the houses: living room, bedroom and kitchen and in one location outside the houses. Measurements were taken

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mostly during the day between the hours of 8am and 4pm.The description of the equipments used is shown in Table 3.1.

Table 3.1 Equipments Profile

EQUIPMENT NAME	MAKE	MODEL	COUNTRY	DETECTION LIMIT	RANGE
Dust track	TSI		USA	0.000µg/m3	10,000µg/m3
Digital gas CO meter	Crowcon	19255h	England	0.01ppm	2000ppm
digital gas NO_2 meter	Crowcon	19832h	England	0.01ppm	500ppm
Digital gas SO_2 meter	Crowcon	19610h	England	0.01ppm	1000ppm

3.5.4 In –situ Direct Observations

The observational Checklist was used to document the in situ environmental conditions of the homes of the respondents. The houses were assessed for the presence of smoke, farmlands, industry, pets, molds, major road, dusts, vectors and electricity generating sets,the types of housing, state of housing and the types of roofs available.

3.6 Statisitical analysis

Data available from well structured questionnaire was well sorted, coded and fed into the computer using the statistical package for social sciences (SPSS). The data were also analyzed using the same package. Data were presented in tables, charts and graphs. Summary statistics such as mean and standard deviations were used to analyze quantitative variables while qualitative variables were summarized by proportions and percentages. Chi square test was used to establish association between qualitative variables while Mann

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Whitney U test was used for quantitative data. All analysis was done at 5% level of significance.

3.7 Ethical Consideration

Informed consents were obtained from the parents of the children that were used. The purpose of the study was explained to participants before instruments for data collection was administered. The respondents were assured of confidentiality and their freedom to decline participation or to withdraw at anytime. The study proposal was submitted to ethical review committee for approval before commencement of the study. The approved informed consent is attached in the appendix.

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CHAPTER FOUR

RESULTS

This chapter presents the results of the questionnaire survey which includes socio-demographic characteristics, smoking status of parents, prevalence rate of mold growth, housing types and number of rooms, respiratory symptoms experienced by respondents, types of cooking fuels used by respondents, age at onset of asthma and number of asthmatic attacks. Also presented are the levels of air pollutants in the homes of the respondents, such as carbon dioxide, nitrogen dioxide, sulphur dioxide and particulates matter.

4.1 Socio-demographic characteristics

A higher proportion of the participants were males. Among the cases twenty (62.5%) were males as compared with twenty-four (75.0%) among the controls. Most of the respondents were Yoruba. Twenty-seven (84.4%) of the cases were Yoruba, 2(6.3%) were Ibo, 1(3.1%) were Hausa while 2(6.2%) were from other ethnic groups. The controls were made up of only Yoruba 31(96.9%) and Ibo 1 (3.1%). Most of the respondents were Christians, 27(84.4%) and 21(65.6%) for both cases and controls respectively, while 5 (15.6%) for cases and 10(31.3%) controls were Muslims. None practiced traditional religion among the cases while 1(3.1%) practiced traditional religion among the controls. Among the cases' fathers 27,84.4% had tertiary education, 2(6.3%) secondary, 2(6.3%) primary and 1(3.1%) had other forms of education. None of the cases' father was without any form of education. Among the control, 22(66.8%) of the 32 fathers interviewed had tertiary education, 7(21.9%) secondary, 2(6.3%) primary while 1(3.1%) was without any form of education. Among the cases, 26(81.3%) of the mothers had tertiary education, 3(9.4%) secondary, 2(6.3%) primary, 1(3.1%) had other forms of education while none was without any form of education. For the controls 17(53.1%) had tertiary education, 11(34.4%) secondary, 1(3.1%) primary while 3(9.4%) had no education. Details of the results are shown in Table 4.1.

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Table 4.1: Sociodemographic characteristics of respondents

Demographic Characteristics	Cases (N=32)	Controls (N=32)
Sex		
Male	20(62.5%)	24(75.0%)
Female	12(37.5%)	8(25.0%)
Ethnic group		
Yoruba	27(84.4%)	31(96.9%)
Ibo	2 (6.3%)	1(3.1%)
Hausa	1(3.1%)	0(0%)
Others	2(6.3%)	0(0%)
Religion		
Christian	27(84.4%)	21(65.6%)
Muslim	5(15.6%)	10(31.3%)
Traditional	0(0%)	1(3.1%)
Educational status of father		
None	0(0%)	1(3.1%)
Primary	2(6.3%)	2(6.3%)
Secondary	2(6.3%)	7(21.9%)
Tertiary	27(84.4%)	22(66.8%)
Others	1(3.1%)	0(0%)
Educational status of mother		
None	0(0%)	3(9.4%)
Primary	2(6.3%)	1(3.1%)
Secondary	3(9.4%)	11(34.4%)
Tertiary	26(81.3%)	17(53.1%)
Others	1(3.1%)	0(0%)

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4.2 Participants Knowledge About Asthma

Majority 20(68.7%) of the cases parents were knowledgeable about the causes of asthma and reported the factors to be dust, smoke, cold heredity and allergic reactions while 10(31.3%) had no idea of what causes asthma. More 21(65.6%) of the controls' parents had no idea of what causes asthma while 11(34.4%) reported causes of asthma to be dust, smoke, cold and allergic reactions.

Few 8 (25.0%) of the cases parents had no idea of what triggers asthma attack while 24(75.0%) believed that the occurrence of asthma was enhanced by dust, lack of medical attention, stress , frying and cold. Close to half 14(43.8%) of the controls'parents had no idea of what enhanced the occurrence of asthma attack while 18(56.2%) believed it was due to dust, cold, lack of medical attention and stress. Most 21(65.6%) of the cases' parents believed that asthma can be prevented, 9(28.1%) reported No while two said they did not have an idea. Twenty (62.5%) of the controls'parents believed that asthma can be prevented, 2(6.3%) believed it can not be prevented while 10 (31.3%) said they did not have an idea.

Majority of the cases 26(84.4%) reported symptoms of asthma to be difficulty in breathing and coughing, 5(15.5%) itching of eyes and sneezing, 1(3.1%) leanness. Whereas seventeen (53.1%) of the controls'parents reported symptoms of asthma to be difficulty in breathing and coughing, 2(6.3%) leanness while 13(40.6%) said they had no idea. Six (18.8%) cases' parents believed that children <5 were mostly affected by asthma, 8(25.0%) repored children>5, 1(3.1%) adolescents, 2(6.2%) adults, 11(34.4%) any age while 4(12.5%) had no idea of the age that is more vulnerable. One (3.1%) control's parent believed that children <5 were mostly affected by asthma, 1(3.1%) children>5, 1(3.1%) adolescents, 8(25.0%) adults, 9(28.1%) any age while 12(37.5%) said they had no idea.

Most of the cases 29(90.6%) believed that asthma could be cured, 2(6.3%) said No while 1(3.1%) said they had no idea. Most of the controls 22(68.8%) also believed that asthma could be cured while 10(31.3%) said they had no idea.

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4.3 History of asthma among study participants

More than half of the cases 18 (56.3%) interviewed reported they had history of asthma in their families while only 5(43.7%) had family history of asthma among the controls.(See figure 4.1)

4.4 Housing conditions of the subjects

Half 16(50.0%) of the cases lived in storey buildings as against 17(53.1%) among the controls. Two (6.3%) of the cases lived in a duplex as compared with None for the controls. Fourteen (43.8%) of the cases lived in a bungalow as against 15(46.9%) of the controls. About half of the respondents 18(56.2%) occupied 3-4 rooms apartment for the cases and controls.(Details are shown on Table 4.2a). Only 1(3.1%) respondent among the cases and the controls occupied between 7- 8 rooms apartment. Six(18.8%) among the cases occupied 1-2 rooms apartment as compared with 11(34.4%) among the controls. Also, more of the cases 7(21.9%) occupied 5-6 rooms apartment as against 2(6.3%) among the controls.(See Table 4.2b).

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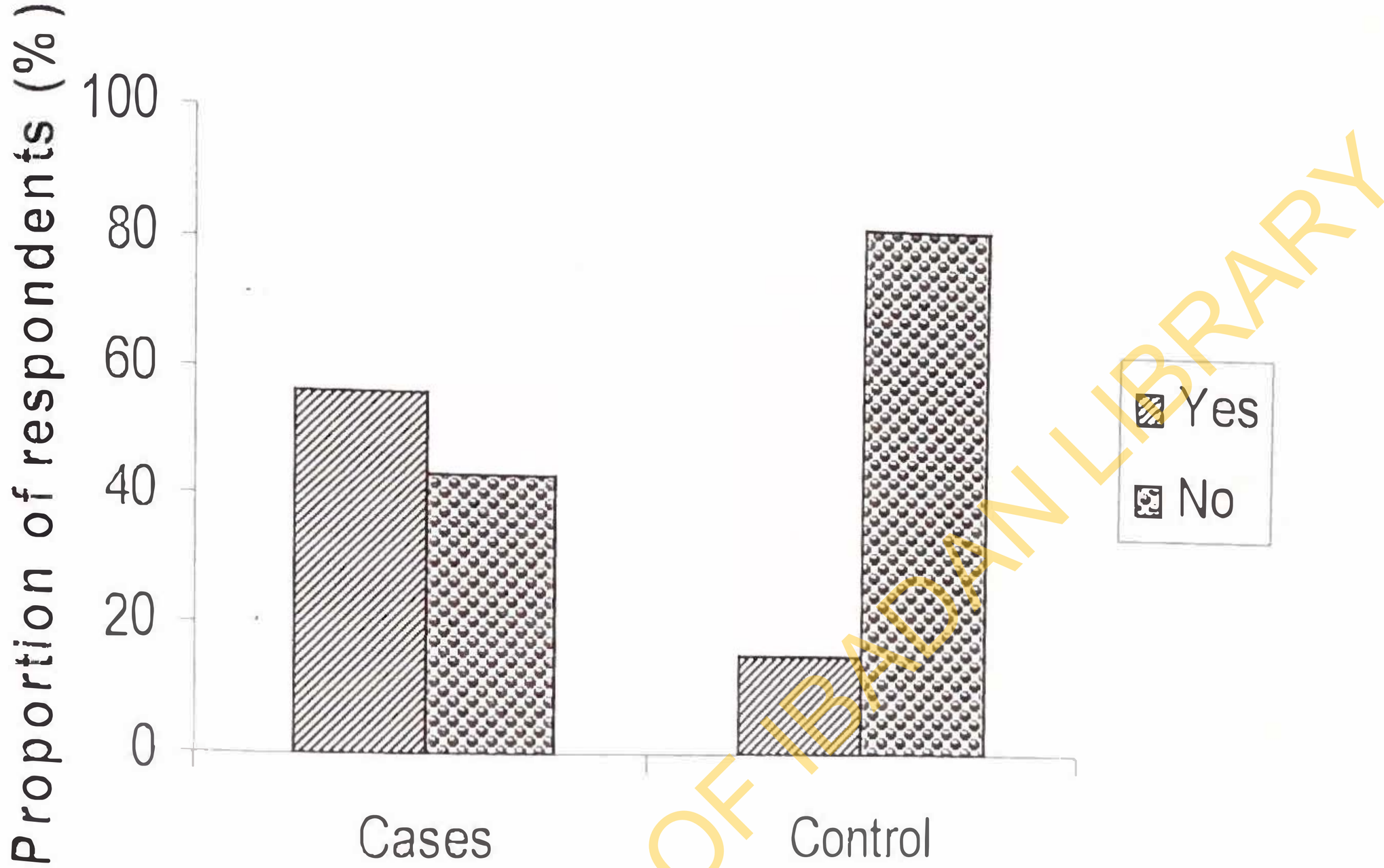


Figure 4.1 **History of asthma among study participants.**

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Table 4.2a: Housing Types

	Cases		Controls	
	Frequency(f)	Percentage(%)	Frequency(f)	Percentage(%)
Bungalow	14	43.8	15	46.9
Duplex	2	6.3	0	0
Storey building	16	50.0	17	53.1
Total	32	100	32	100

Table 4.2b : Number of rooms occupied by respondents

	Cases		Controls	
	Frequency(f)	Percentage(%)	Frequency(f)	Percentage(%)
1-2rooms	6	18.8%	11	34.4%
3-4rooms	18	56.2%	18	56.2%
5-6rooms	7	21.9%	2	6.3%
7-8rooms	1	3.1%	1	3.1%

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4.5 Environmental Risk factors associated with asthma

Four (12.5%) out of the 32 cases interviewed reported that their parents smoked compared to 2 (6.3%) among the controls. (Table 4.3.). 13(40.6%) out of 32 cases said they experienced mold growth while only 11(34.4%) out of the controls reported presence of mold growth on their walls (see Figure 4.2). 23(71.9%) cases had electricity generating set in their houses while 20(62.5%) had electricity generating sets among the controls. (See Figure 4.3, Plate 4.1).

Thirteen (40.6%) cases had industries situated close to their houses while only 7(21.9%) of the controls had industries situated close to their houses (See Figure 4.4). 14(43.8%) of the cases had farms close to their houses while 11(34.4%) had farms close to their houses among the controls. (See Figure 4.5).

About one-third of cases 11(34.4%) were keeping pets(any of dogs , goats and poultry) as at the time of this study while only 8(25.0%) controls kept pets as at this time .(See Figure 4.6).

Risk factors found significant with asthma were gender, history of asthma, type of floor covering and presence of allergy to cold, dust, foul smell and smoke. The strongest risk factor found was the presence of allergy to cold, dust, foul smell and smoke. Patients with any form of allergy were more likely to develop asthma compared to those without allergy (P= 0.001, adjusted OR=121.39, 95% C.I (7.15-2061.143)). Females were more likely to develop asthma compared to males (adjusted OR=31.01, 95% C.I (1.50-641.54)). Family history of asthma(P=0.03) and use of carpet as floor covering was also found significant, P=0.026). Patients that had family history of asthma were more likely to develop asthma compared to those who had no history of asthma (OR=22.14, 95% C.I (1.36-360.8114)) Patients whose floor were covered with carpet were less likely to develop asthma than those whose floor has no covering (OR=0.02, 95% C.I (0.00-0.64)).(See Table 4.4, Plate 4.3).

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Table 4.3: Smoking status of parents

Does any of your parents smoke	Cases(n=32) f (%)	Controls n=32) f (%)
Yes	4(12.5%)	2(6.3%)
No	28(87.5)	30(93.7%)

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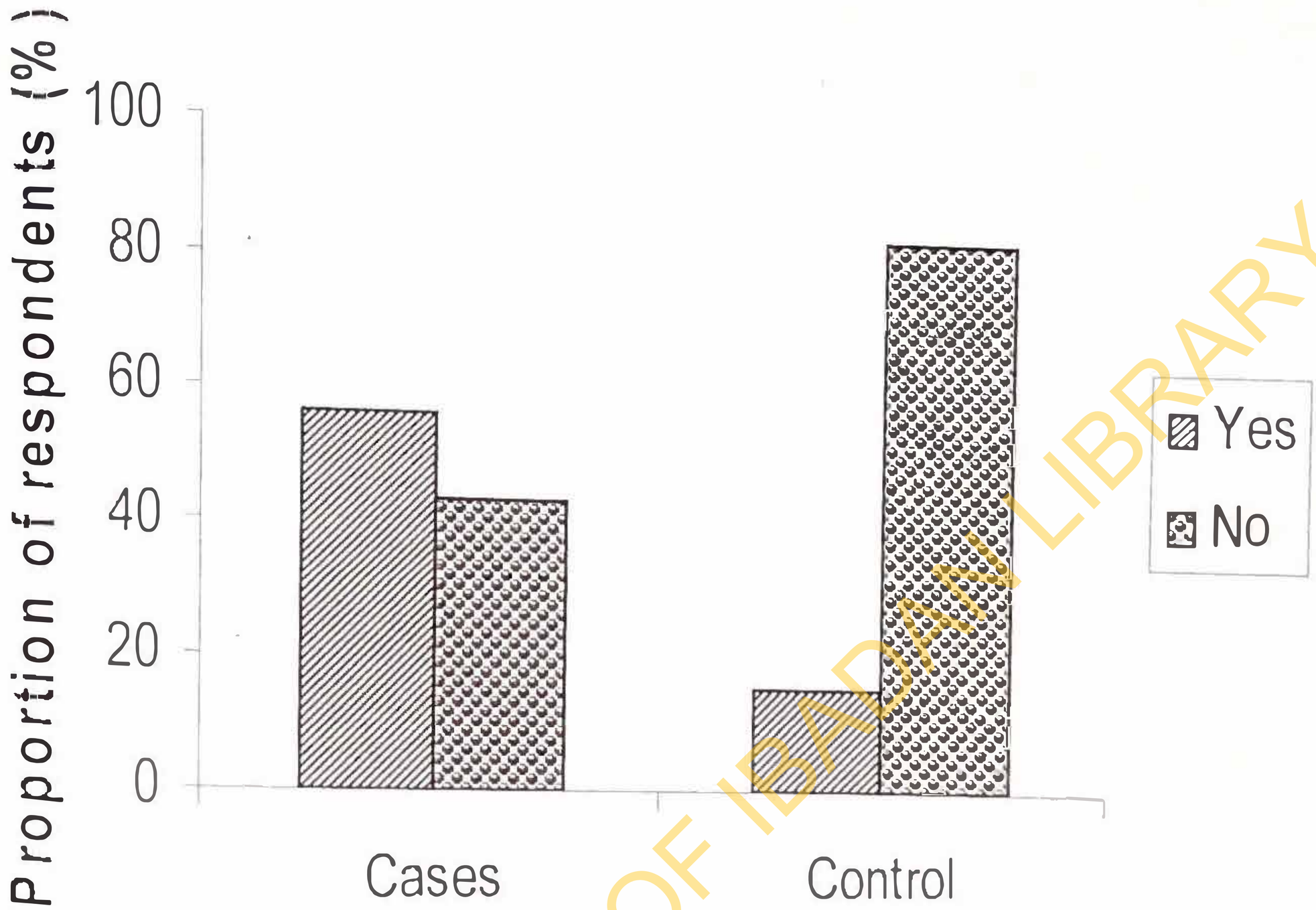


Figure 4.2: Reported prevalence of mold growth among the participants

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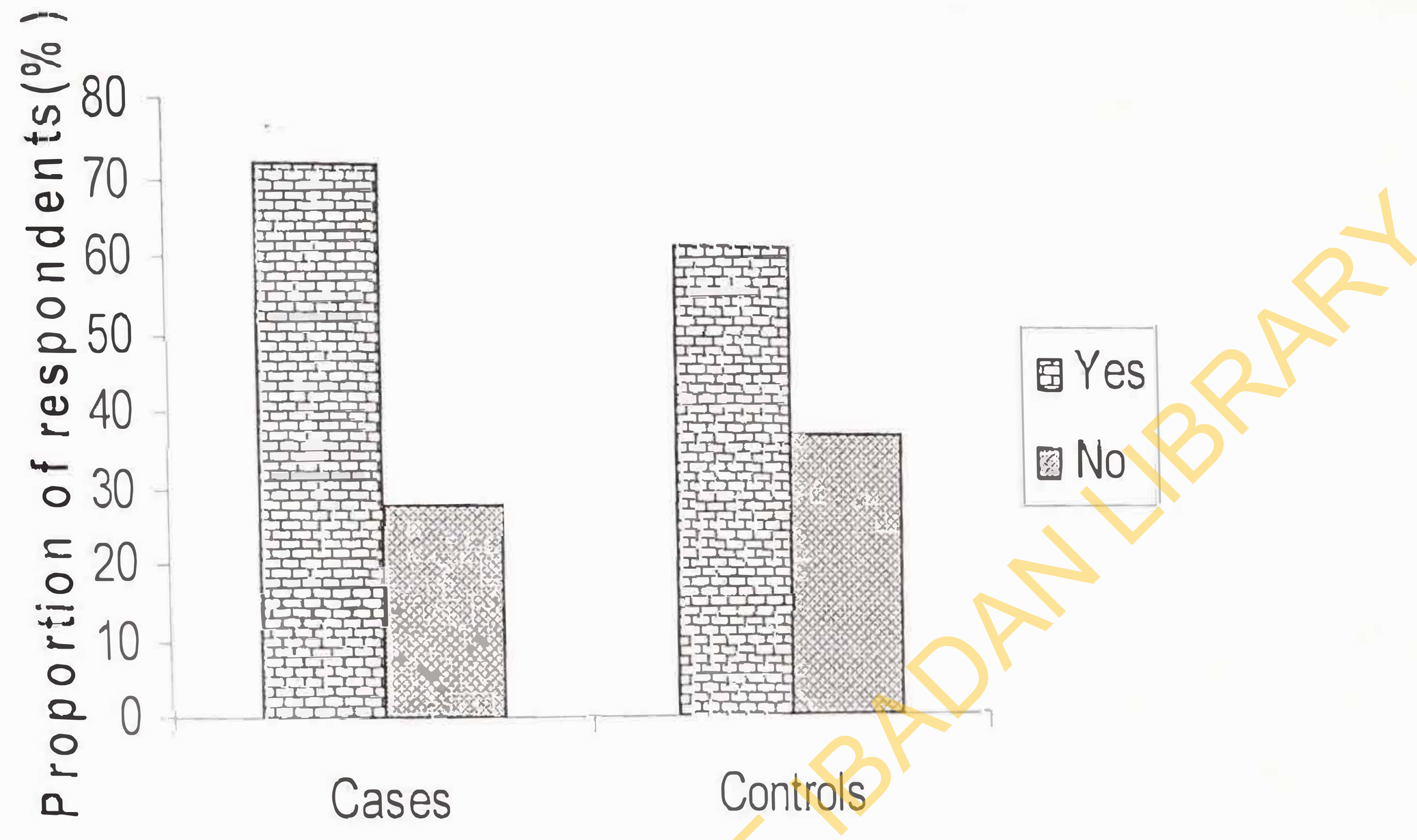


Figure 4.3: Usage of Electricity generating sets among the participants

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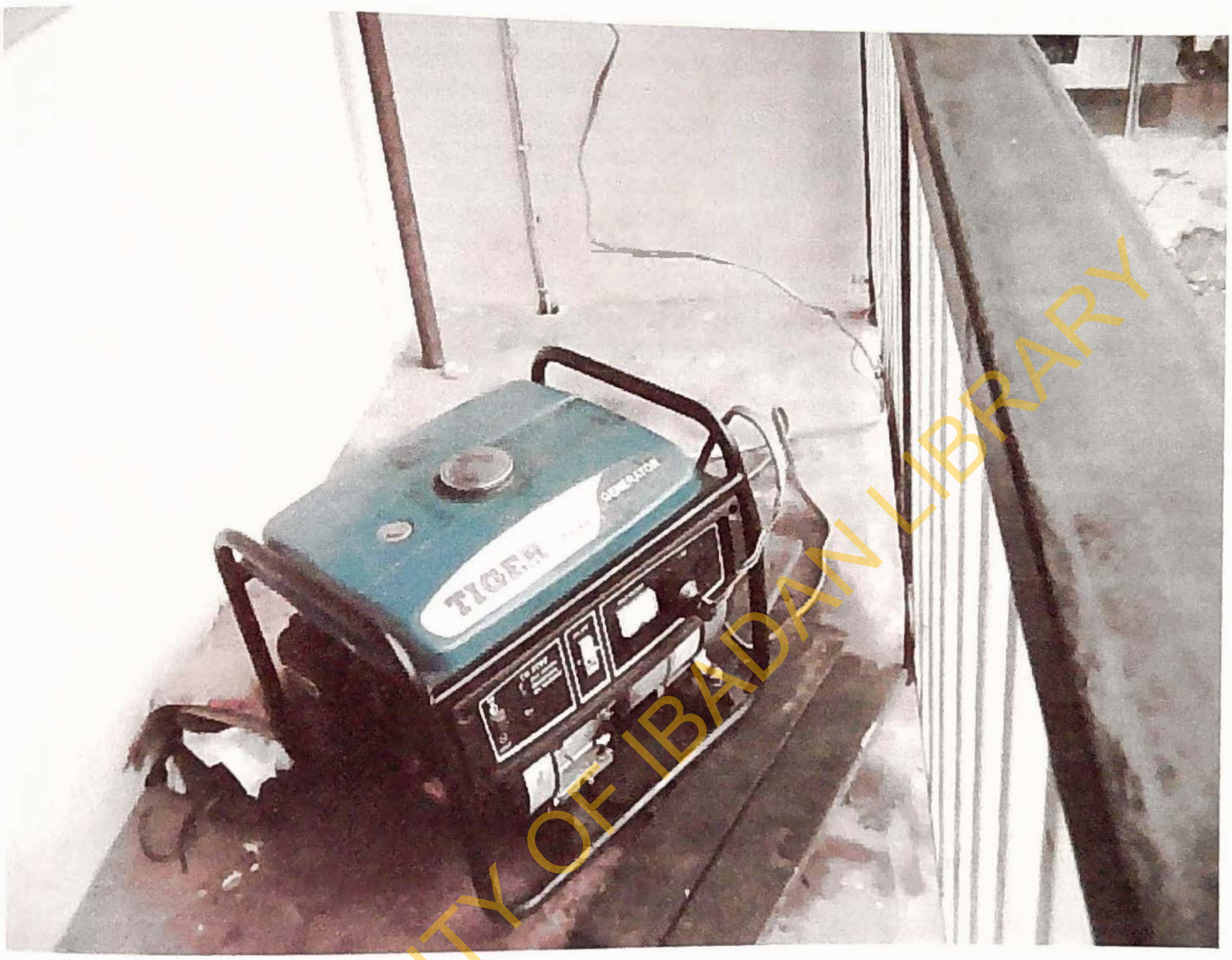


Plate 4.1: Generator placed at the entrance of a case's house

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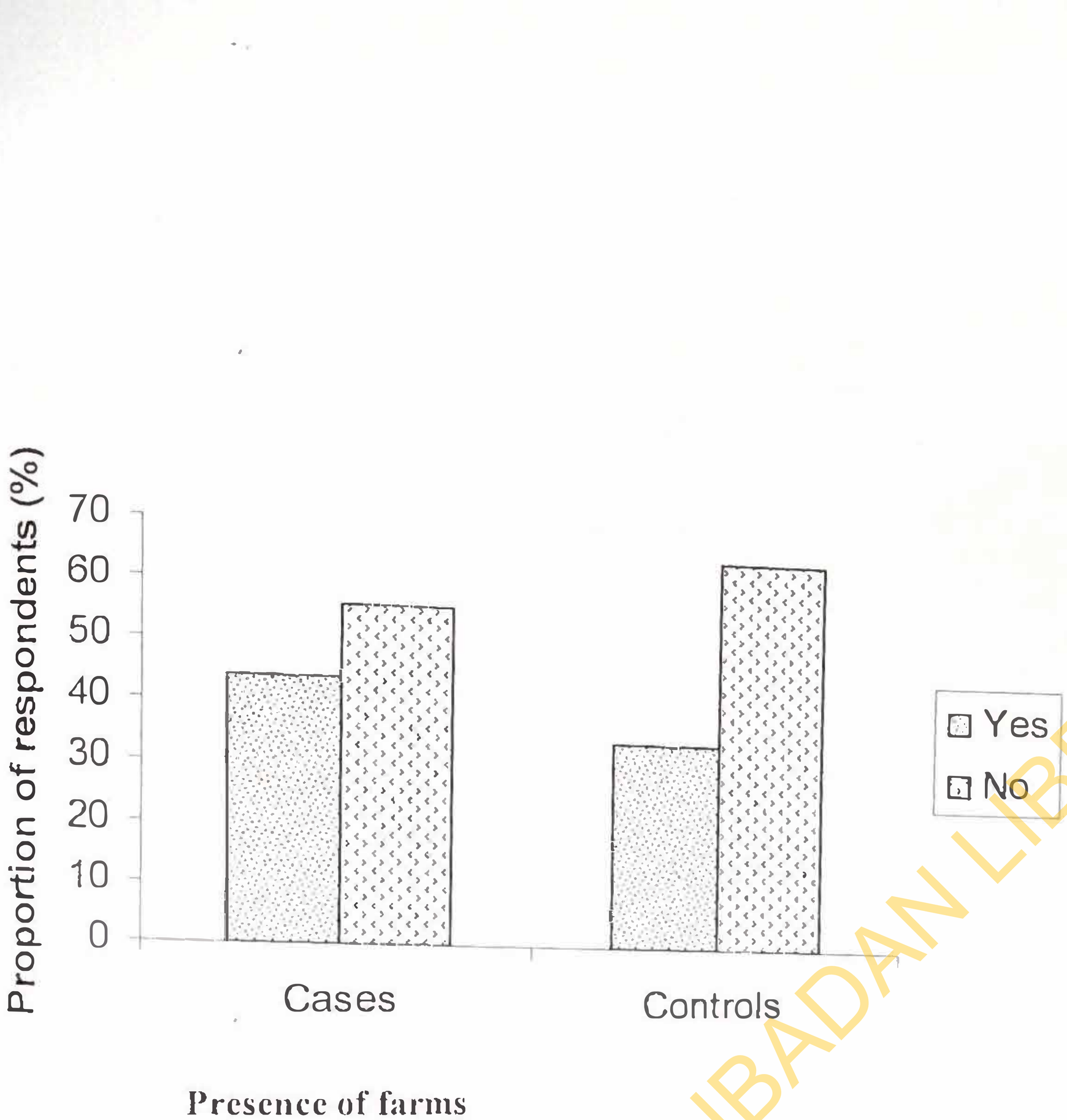


Figure 4.4: Presence of farm close to the residential areas of the participants

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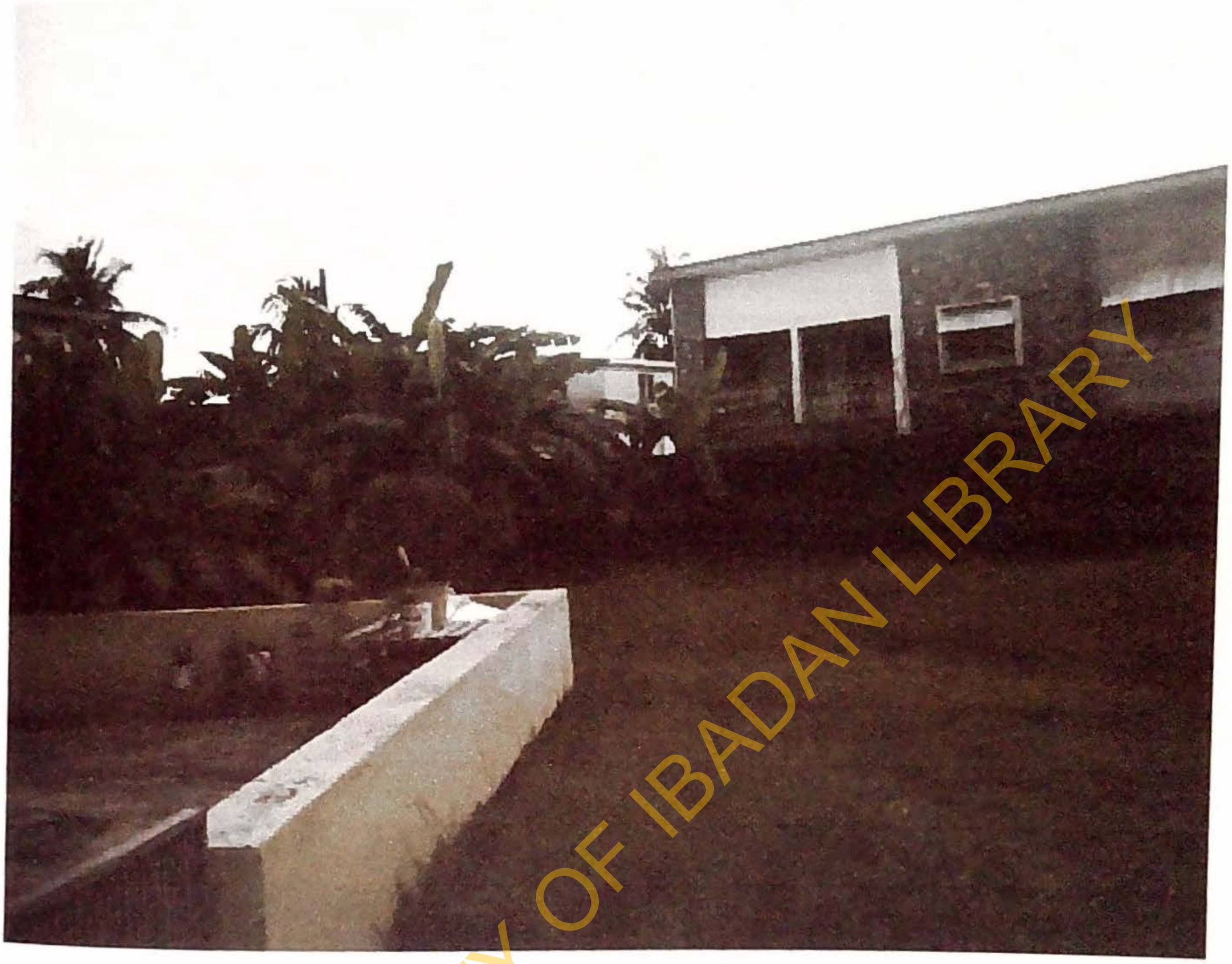


Plate 4.2: Farms and bushes inside a case's compound

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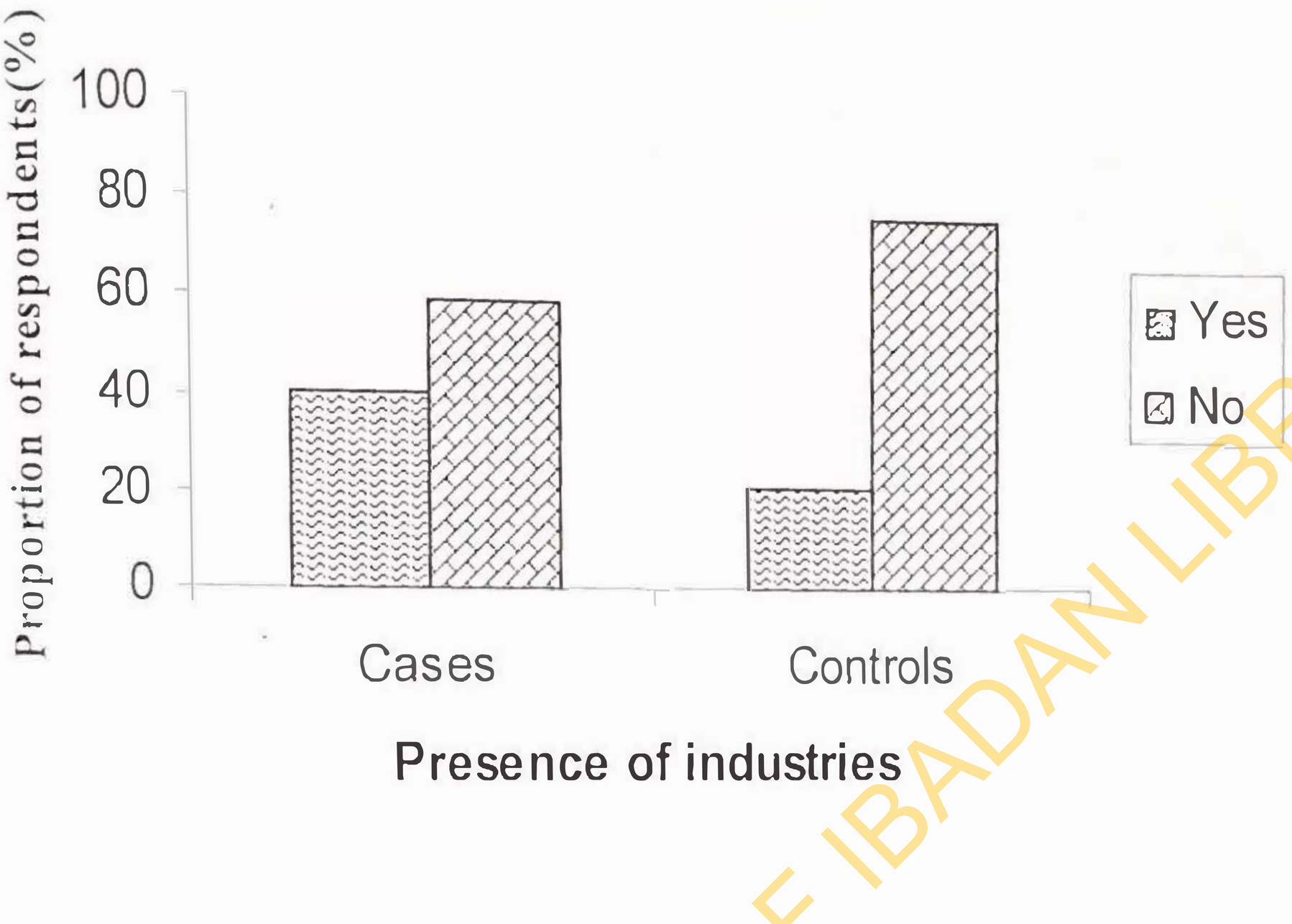


Figure 4.5: Presence of industries close to the residence of participants

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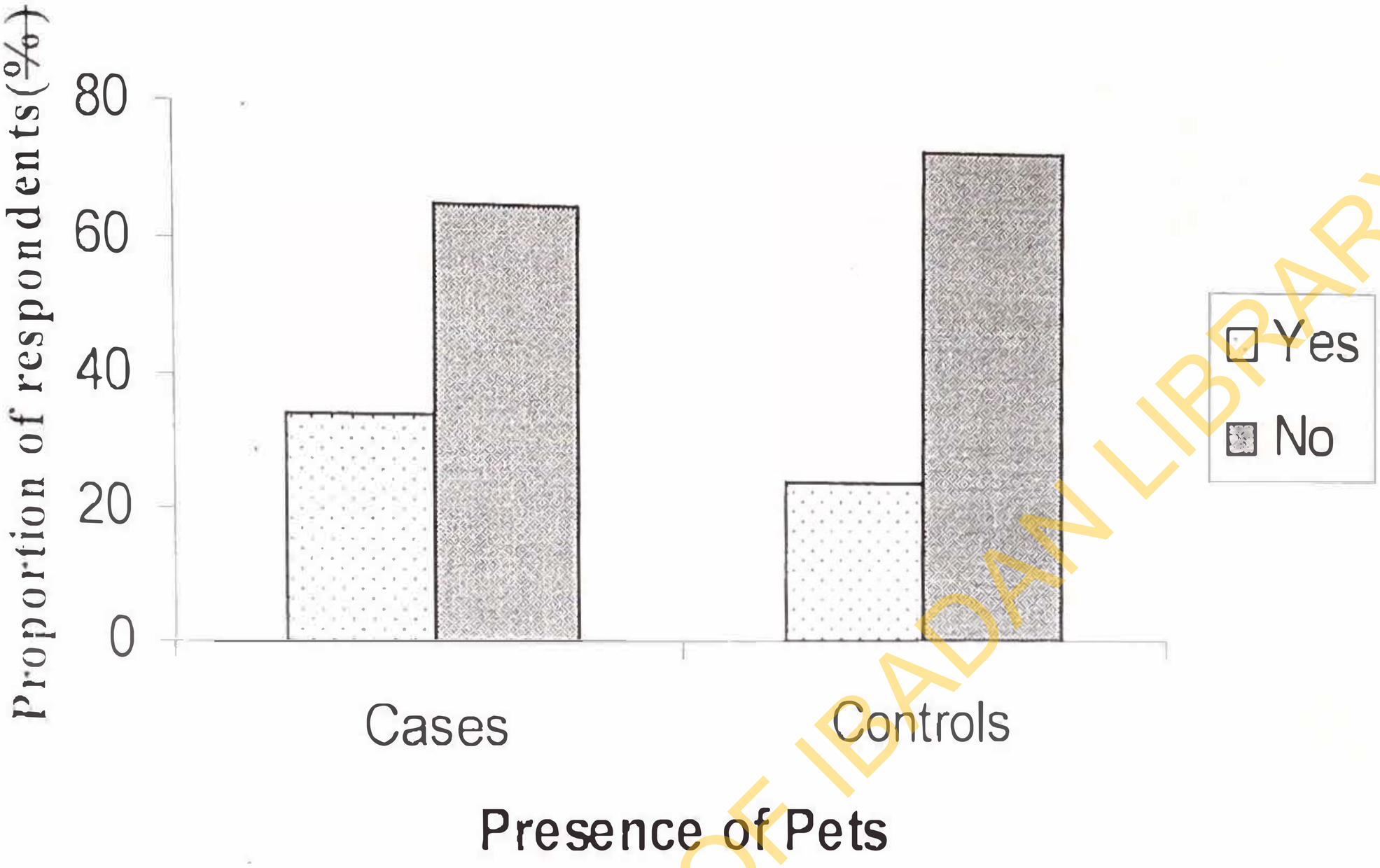


Figure 4.6: Exposure to Pets

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Table 4.4: Risk factors for asthma using Multivariate Logistic Regression

Variable	Unadjusted OR (95% C.I)	P-value	Adjusted OR (95% C.I)	P-value
Gender				
Male	1		1	
Female	1.80 (0.62-5.27)	0.283	31.01 (1.50-641.54)	**0.026**
History of asthma				
No	1		1	
Yes	6.94 (2.13-22.65)	0.001	22.14 (1.36-360.811)	**0.030**
Type of floor covering				
Not covered	1		1	
Rug	1.25 (0.22-7.08)	0.801	0.16 (0.00-6.46)	0.334
Tiles	2.50 (0.34-18.33)	0.367	2.97 (0.03-266.32)	0.635
Carpet	0.31 (0.07-1.42)	0.132	0.02 (0.00-0.64)	**0.026**
Terrazzo	1.00(0.10-9.61)	1.000	0.11 (0.00-18.87)	0.404
Carpet/tile/rug	4.50 (2.31-89.94)	0.122	2.00 (0.10-38.36)	0.647
Allergy				
No	1		1	
Yes	25.00 (6.54-95.63)	<0.001	121.39 (7.15-2061.143)	**0.001**
No of persons per room				
<=3	1		1	
>=4	2.27 (0.72-7.16)	0.162	4.14 (0.22-77.02)	0.341

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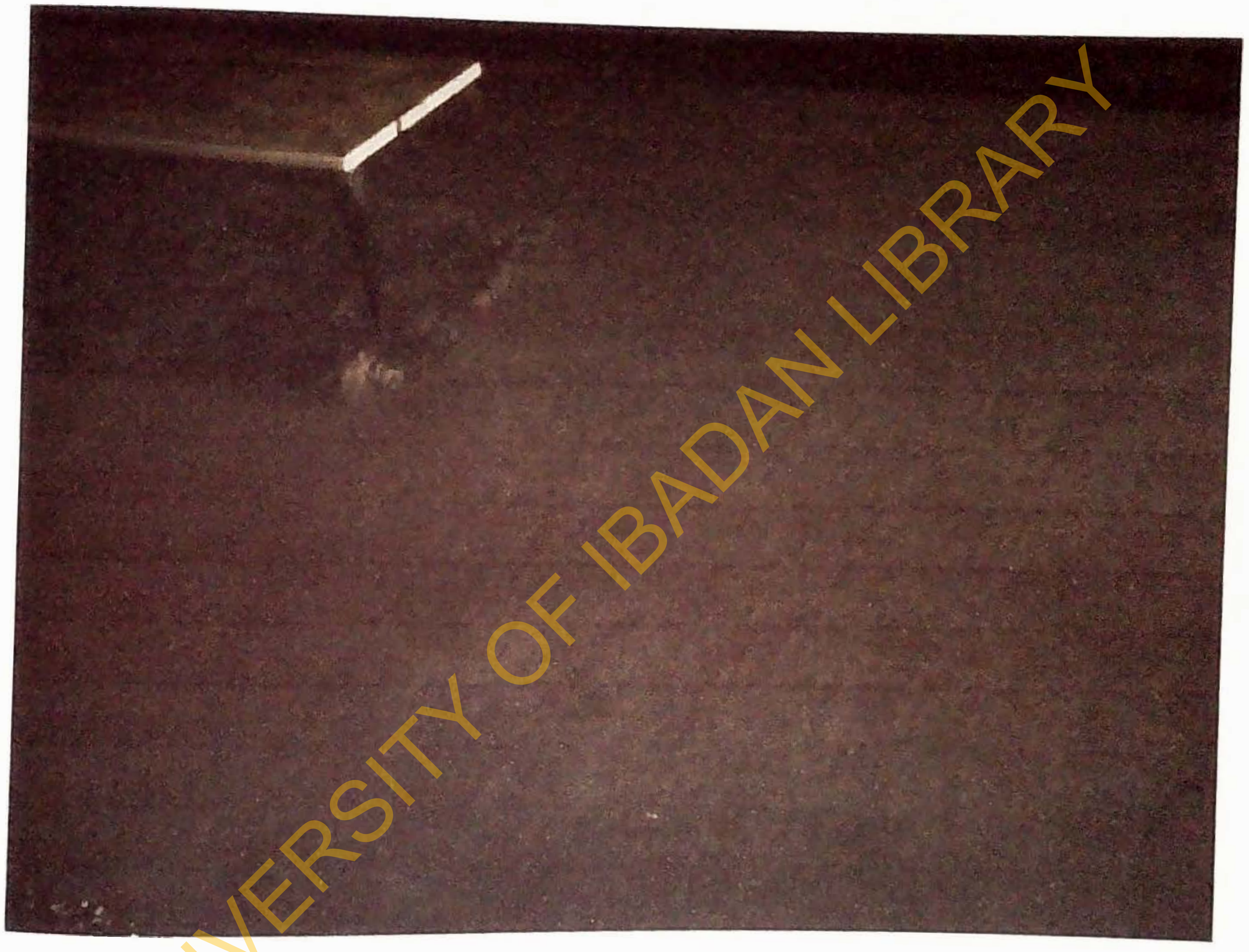


Plate 4.3: Rugs on the floor in one of the respondents's living room

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4.6 Types of cooking fuels used by respondents

Figure 4.4 highlihts the types of cooking fuel used by the respondents.Most of the cases 68.8% use kerosene as thier cooking fuel, 15.6% used electric cooker, 6.2% used gas coooker, 6.2% used coal while 3.1% used firewood. Among the controls, no respondent used firewood or gascooker, majority 78.1% used kerosene, 6 (18.8%) used electric cooker and only one respondent (3.1%) used coal. (See Figure 4.7)

4.7 Participants response to allergies

Only four(12.5%) of the cases reported absence of allergy as compared with 25(78.1%) among the controls. Six(18.8%), 1(3.1%), 10(31.3%) and 3(9.4%) among the cases reported that they were allergic to dust, smoke, cold and foul smell respectively as against 2(6.3%), 0(0%), 5(31.3%) and 0(0%) among the controls.6(18.8%) of the cases reported allergy to dust and smoke and 2(6.3%) to dust and cold as compared with 0(0%) and 0(0%) respectively among the controls. (See Figure 4.8.).

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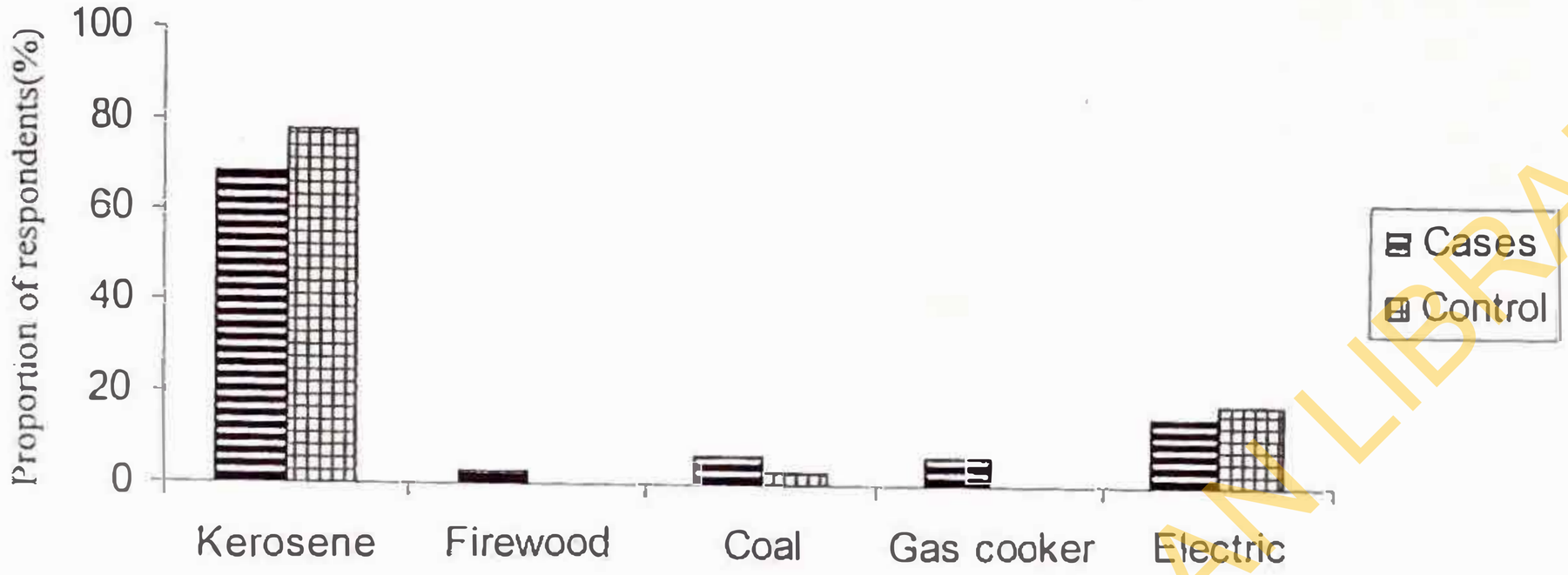


Figure 4.7: Different types of cooking fuel used by the participants

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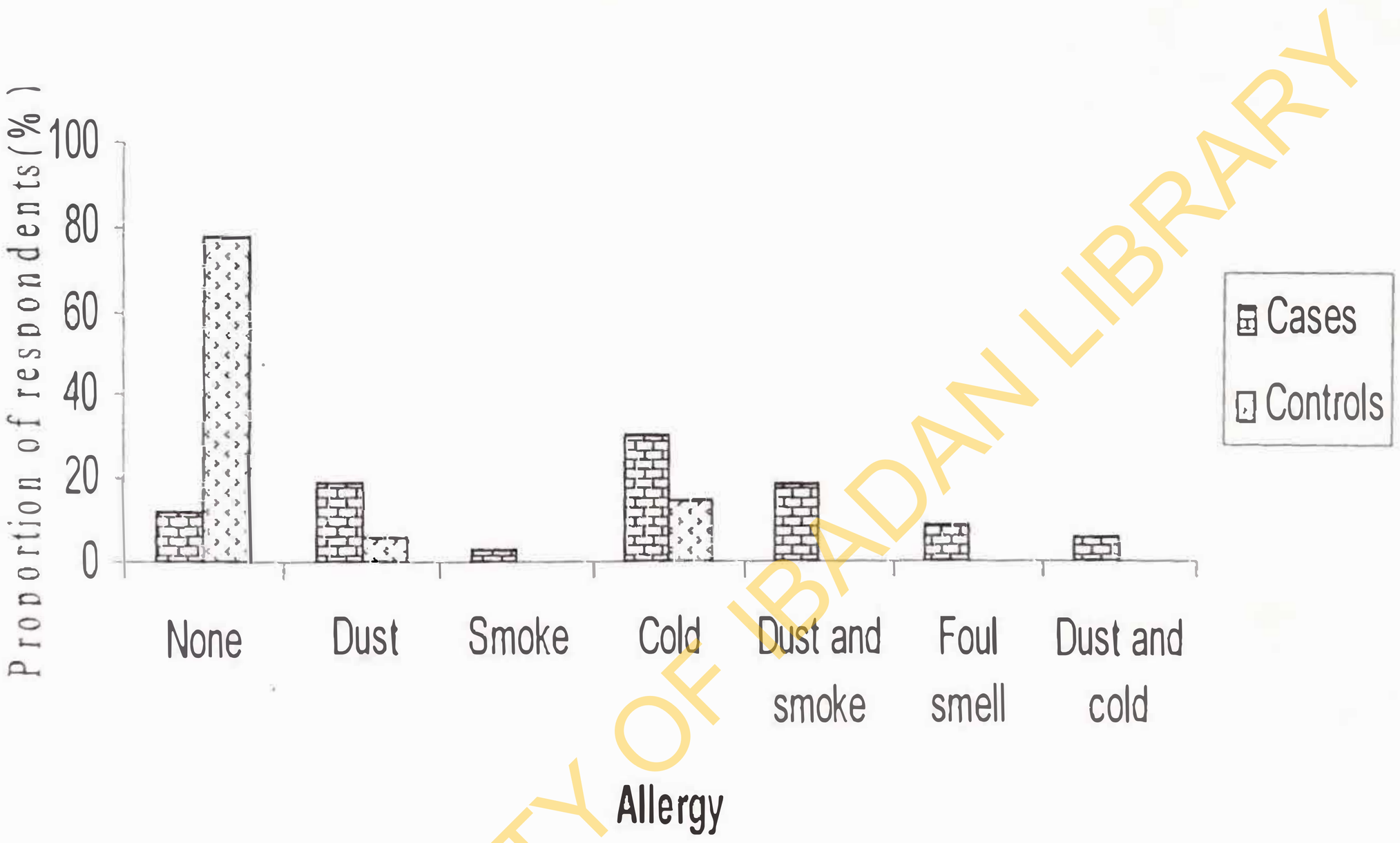


Figure 4.8: Participants' response to allergy

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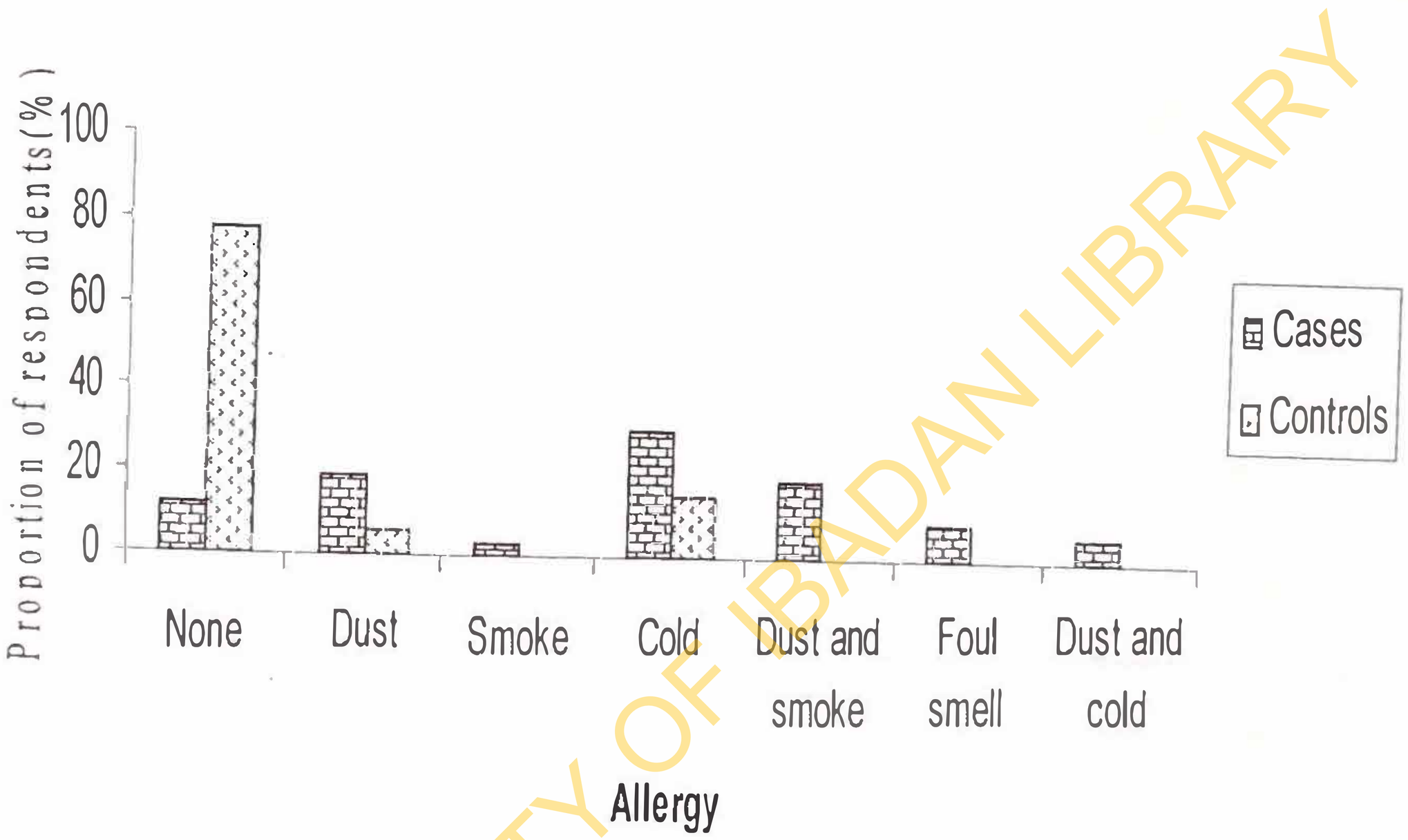


Figure 4.8: Participants' response to allergy

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4.8 On-site Observation

The result of the observational checklist shown in Table 4.5 shows that pets were highly present in the homes of the cases than the controls. Also generators were observed in most the cases' residence while only few of the controls had generators. Farms and Bushes were observed close to more of the cases' residence while just few of the controls had farms close to their residences. Dust was highly present in more of the controls' apartment while at most of the cases apartment, there were no dusts observed. Moderately, both the cases and the controls had major roads close to their residences. Also, smoke was observed in the case's residence (Plate 4.4), while in the controls residences, there were no smoke observed.

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Table 4.5 On-site Observation

Indicators observed	Cases	Controls
Farmlands/garden	++	+
Pets	+++	+
Dusts	++	+++
Major road	++	++
Smoke	++	-
Electricity Generating set	+++	++

Key: +++Highly present + +Moderately Present Present + -Absent

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Table 4.5 On-site Observation

Indicators observed	Cases	Controls
Farmlands/garden	++	+
Pets	+++	+
Dusts	++	+++
Major road	++	++
Smoke	++	-
Electricity Generating set	+++	++

Key: +++Highly present + +Moderately Present Present + -Absent

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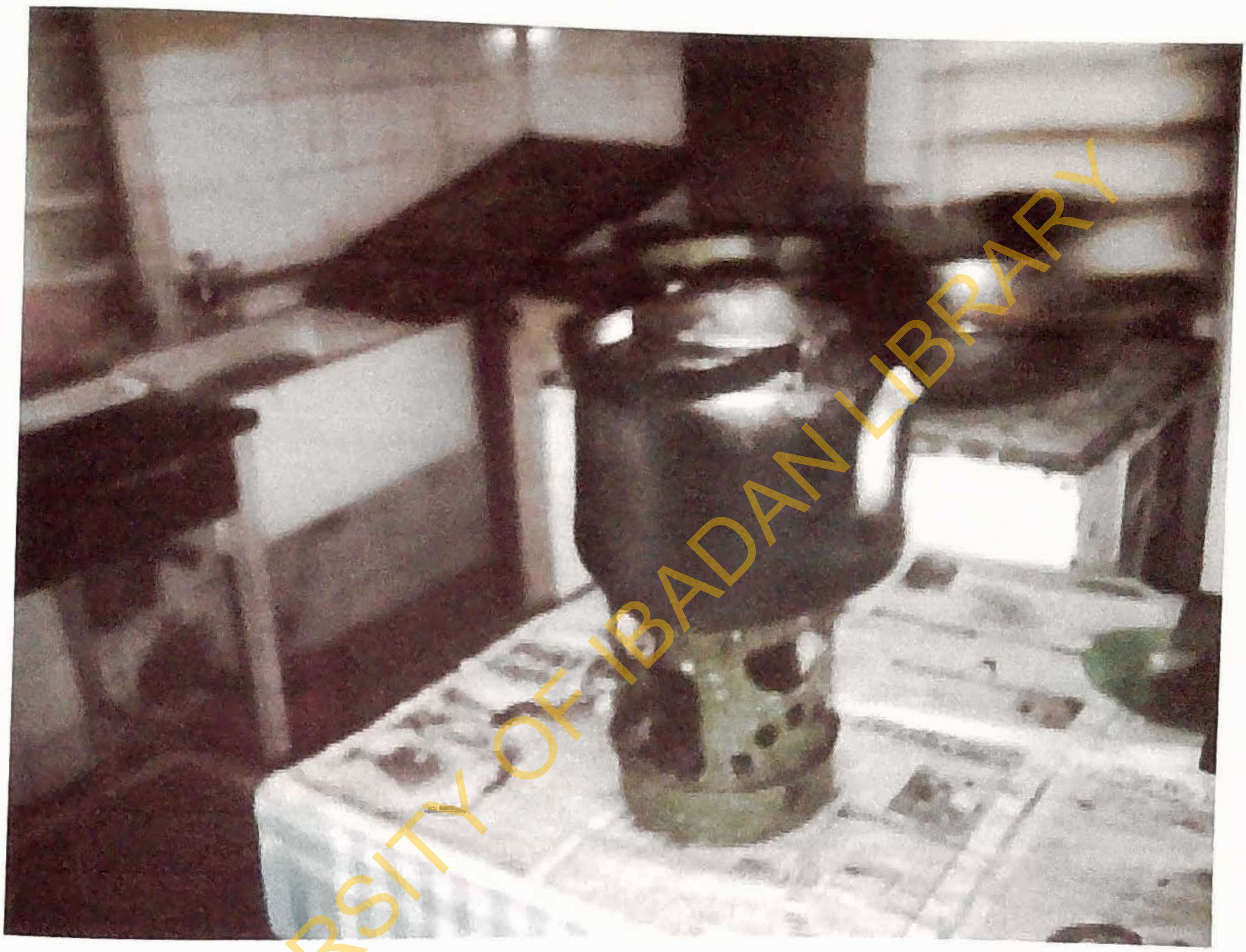


Plate 4.4: A source of smoke observed in the kitchen of one of the cases

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4.9 Indoor air quality in the living room of respondents

The median concentration of particulate matter produced in the living room of the cases was 21.65μg/m^3 which was less than that among the controls (25.4μg/m^3). Among the cases, the median concentration of CO detected was 0.2ppm with a maximum value of 0.67, while NO_2 and SO_2 had the same median concentration of 0.01ppm and maximum values of 0.03ppm and 0.01ppm respectively. The median concentration of CO detected among the controls was 0.2ppm with a maximum value of 0.3ppm while NO_2 and SO_2 had the same med ian concentration of 0.01 with maximum values of 0.02ppm and 0.01ppm respectively. (See Table 4.6 and figures 4.9, 4.10 and 4.11.)

4.10 Indoor air quality at the bedroom of respondents

The maximum value of particulate matter detected in the bedroom of the cases was 123.4(μg/m^3) with a median value of 16.8(μg/m^3), this was less than that detected among the controls which had maximum value of 266.5μg/m^3 and a median concentration of 29.1ug/m^3. The median concentration of CO detected among the cases was 0.19ppm which was very close to that of the controls 0.2ppm, while they both had the same maximum values of 0.3ppm. Both the cases and controls had the same median concentration for NO_2 ,0.01ppm as well as the same maximum values (0.02ppm). SO_2 was not detected in the bedroom of the cases while among the controls the median concentration and the maximum value detected was the same (0.02ppm). (See Table 4.7 and figures 4.9, 4.10 and 4.11).

4.11 Indoor air quality within the kitchen of respondents

The median concentration of particulates matter detected in the kitchen of the cases and controls were 29.4μg/m^3 and 34.5μg/m^3 respectively while the maximum values were 203.3μg/m^3 and 255.5μg/m^3 respectively. CO was detected with a maximum value of 1.3ppm and median concentration of 0.3ppm among the cases and a maximum value of 1.2 and a median concentration of 0.5 among the controls. NO_2 was detected among the cases with a maximum value of 0.2ppm and a median concentration of 0.02ppm while maximum value of 0.04ppm and median concentration of 0.01ppm was detected among the controls. SO_2 was detected with a maximum value of 0.02ppm and a median concentration of

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0.001ppm among the cases while the controls had the same maximum value and median concentration (0.02ppm). (See Table 4.8 and figures 4.9, 4.10 and 4.11).

4.12 Outdoor air quality in the vicinity of the respondents'homes

The median concentration of particulates matter in the outdoor environment of the cases was 28.75μg/m^3 with a maximum concentration of 186.7 μg/m^3 and a minimum concentration of 0.138 μg/m^3. The median concentration of particulates matter among the controls was 34.7 μg/m^3 with a maximum and minimum concentration of 360.2 μg/m^3 and10.6 μg/m^3 respectively. Carbon monoxide in outdoor environment of the cases was detected with a maximum and minimum value of 3.8 ppm and 0.12 ppm respectively and had a median concentration of 0.4ppm, while among the controls, the maximum and minimum values of 2.4ppm and 0.07ppm were recorded respectively with a median concentration of 0.42ppm. Nitrogen dioxide level in the outdoor environment of the cases recorded maximum and minimum values of 0.04ppm and 0.01ppm respectively with a median concentration of 0.02ppm while maximum and minimum values of 0.1ppm and 0.01ppm and a median concentration of 0.02ppm was recorded among the controls . (See Table 4.9 and Figures 4.9, 4.10 and 4.11).

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Table 4.6: Min and Max concentration of air pollutants in the living room

	Cases		Controls	
	Min	Max	Min	Max
Particulate matter(ug/m^3)	3.5	126.7	14.4	266.5
Carbon monoxide(ppm)	0.04	0.67	0.03	0.3
Nitrogen dioxide(ppm)	0.001	0.03	0.01	0.02
Sulphur dioxide(ppm)	0.001	0.01	0.01	0.01

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Table 4.7: Min and Max concentration of air pollutants in the bedroom

	Cases		Controls	
	Min	Max	Min	Max
Particulate matter (ug/m^3)	1.6	123.4	3.3	266.5
Carbon monoxide(ppm)	0.02	0.3	0.03	0.3
Nitrogen dioxide(ppm)	0.001	0.02	0.01	0.02
Sulphur dioxide(ppm)	ND	ND	0.02	0.02

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Table 4.8: Min and Max concentrations of indoor pollutants in the kitchen

	Cases		Controls	
	Min	Max	Min	Max
Particulate matter (ug/m^3)	4.1	203.3	13.3	255.5
Carbon monoxide(ppm)	0.08	1.3	0.1	1.2
Nitrogen dioxide(ppm)	0.001	0.2	0.01	0.04
Sulphur dioxide(ppm)	0.001	0.02	0.01	0.02

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Table 4.9: Min and Max concentration of outdoor pollutants

	Cases		Controls	
	Min	Max	Min	Max
Particulate matter (ug/m^3)	0.138	186.7	10.6	360.2
Carbon monoxide(ppm)	0.12	3.8	0.07	2.4
Nitrogen dioxide(ppm)	0.01	0.04	0.01	0.1
Sulphur dioxide(ppm)	0.01	0.02	0.01	0.03

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Figure 4.9: Median concentrations of particulates matter in the homes across the different locations.

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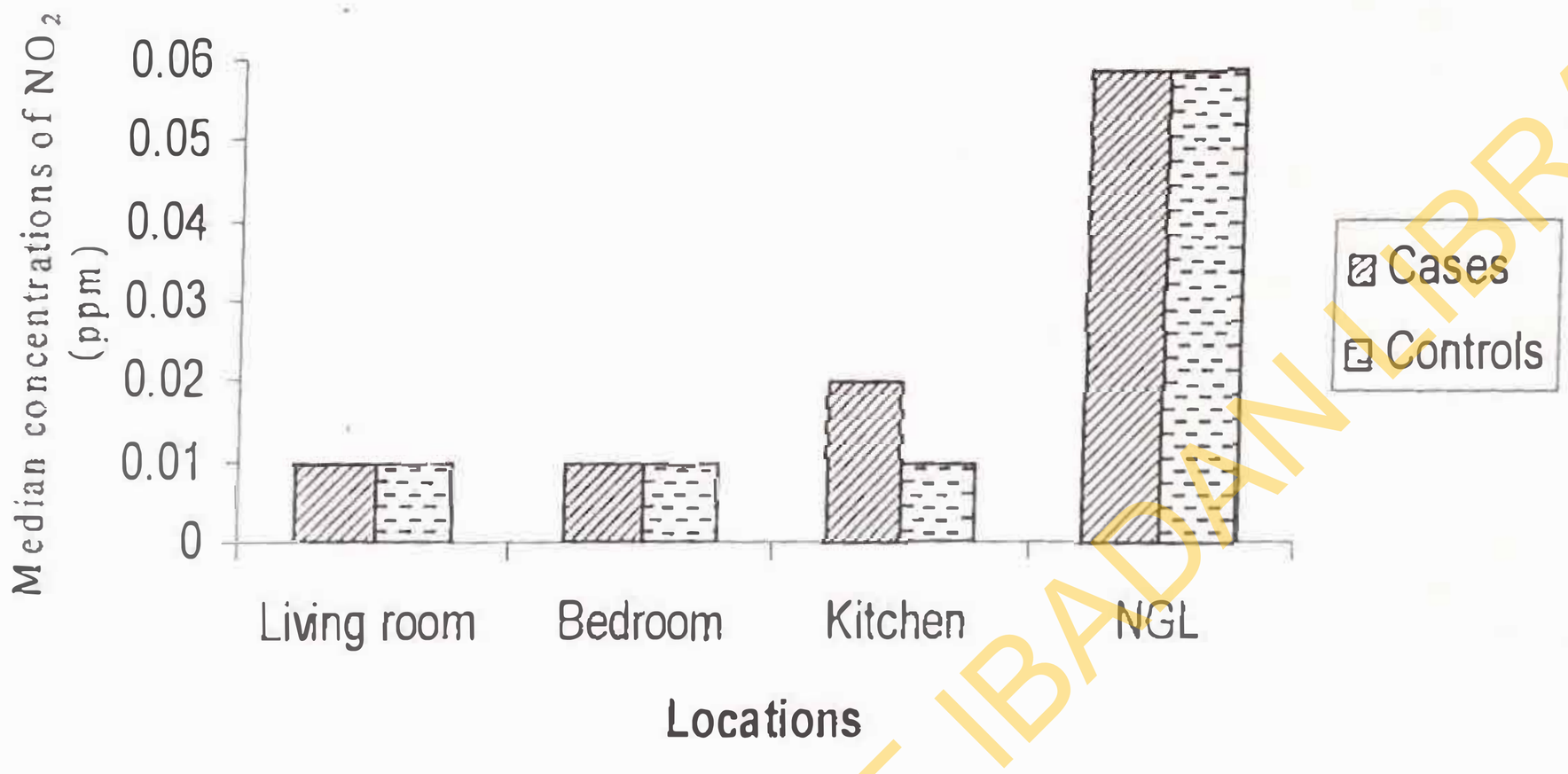


Figure 4.10: median concentration of NO_2 in the homes across the different locations.

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Figure 4.11: median concentration of CO in the homes across the different Locations.

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4.13 Respiratory symptoms experienced by respondents

The most common respiratory symptoms experienced by the cases in the past three months preceeding the study was coughing(90.6%), followed by cold/catarrh(87.5%) and then difficulty in breathing 18(56.3%). The least common respiratory symptoms experienced among the cases was nose bleeding 2(6.5%). The most common respiratory symptoms experienced by the controls within the past the past three months preceeding the study was cold/catarrh 17(53.1%), followed by cough 10(31.3%) and then rashes7(21.9%). The least common perceived respiratory symptoms anomg the controls was chest pain 5(15.6%). Only few 6(18.8%) experienced difficulty in breathing. (See Table 4.10).

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Table 4.10: Respiratory symptoms experienced by the respondents

	OPTION	CASES	CONTROLS
Difficulty with breathing	Not at all	11(34.4%)	25 (78.1%)
	<3 months	18 (56.3%)	6 (18.8%)
	< 6 months	3 (9.3%)	1 (3.1%)
Coughing	Not at all	1 (3.1%)	18 (56.3%)
	<3 months	29 (90.6%)	10 (31.3%)
	< 6 months	2 (6.3%)	4 (12.6%)
Cold/catarrh	Not at all	3 (9.4%)	10 (31.3%)
	<3 months	28 (87.5%)	17 (53.1%)
	< 6 months	1 (3.1%)	5 (15.6%)
Chest pain	Not at all	23 (71.9%)	26 (81.3%)
	<3 months	7 (21.9%)	5 (15.6%)
	< 6 months	2 (6.3%)	1 (3.1%)
Rashes	Not at all	22(68.8%)	24 (75.0%)
	<3 months	10 (31.2%)	7 (21.9%)
	< 6 months	0	1 (3.1%)
Nose bleeding	Not at all	29 (93.5%)	32 (100.0%)
	<3 months	2 (6.5%)	0
	< 6 months	0	0

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4.14 Age at onset of asthma

More than half of the cases 53.2% were diagnosed of asthma between the ages 0-5 years while 40.6% were diagnosed at ages between 6- 10 years. Only 2 (6.2%) were diagnosed at ages between 11- 15 years.(See figure 4.10).

4.15: Episodes of attacks in the past one year

Majority of the cases(59.5%) had more than one attack within one year while only 6 (18.6) had more than 6 attacks in the past one year. (See figure 4.11).

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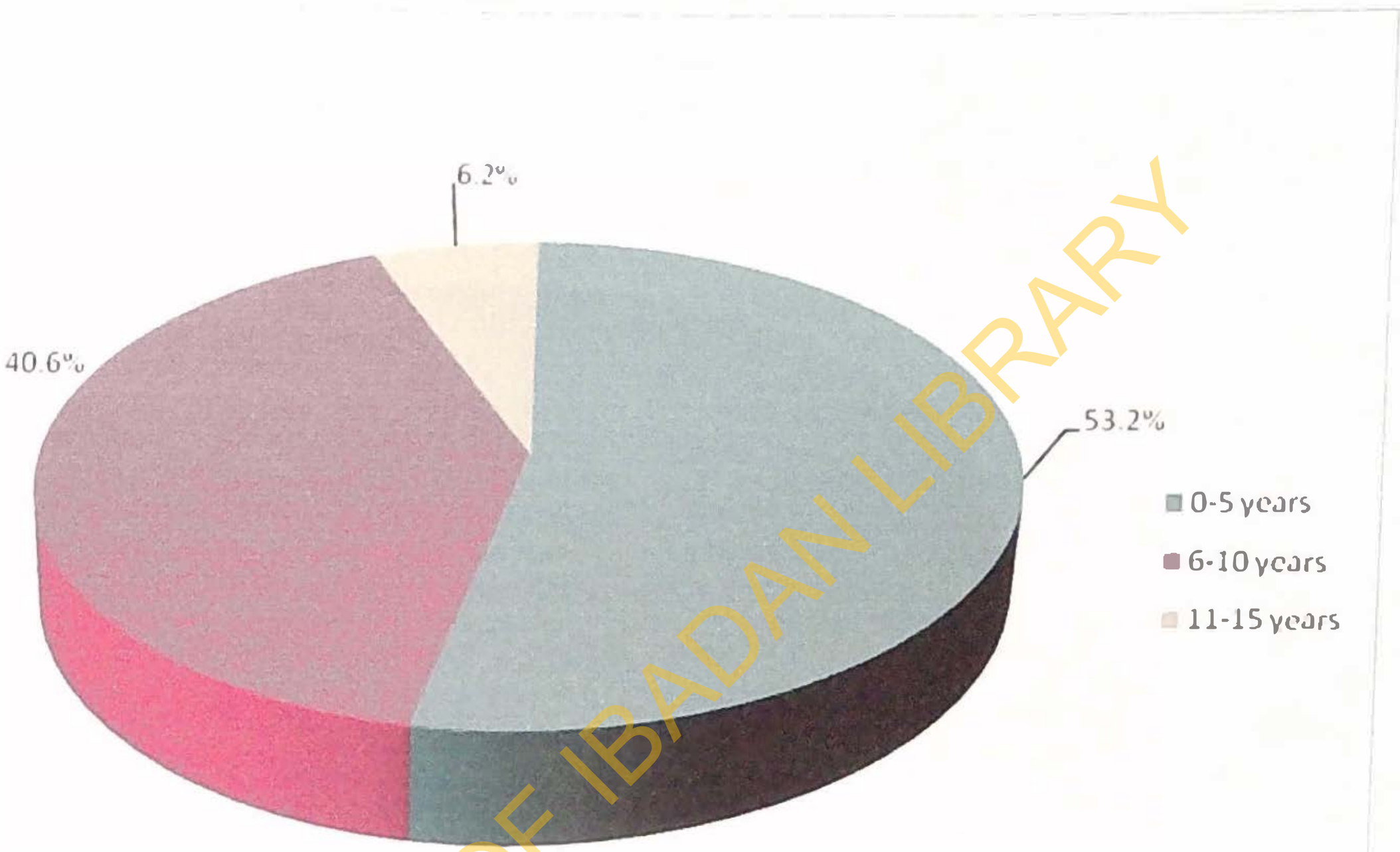


Figure 4.12: Age at onset of asthma

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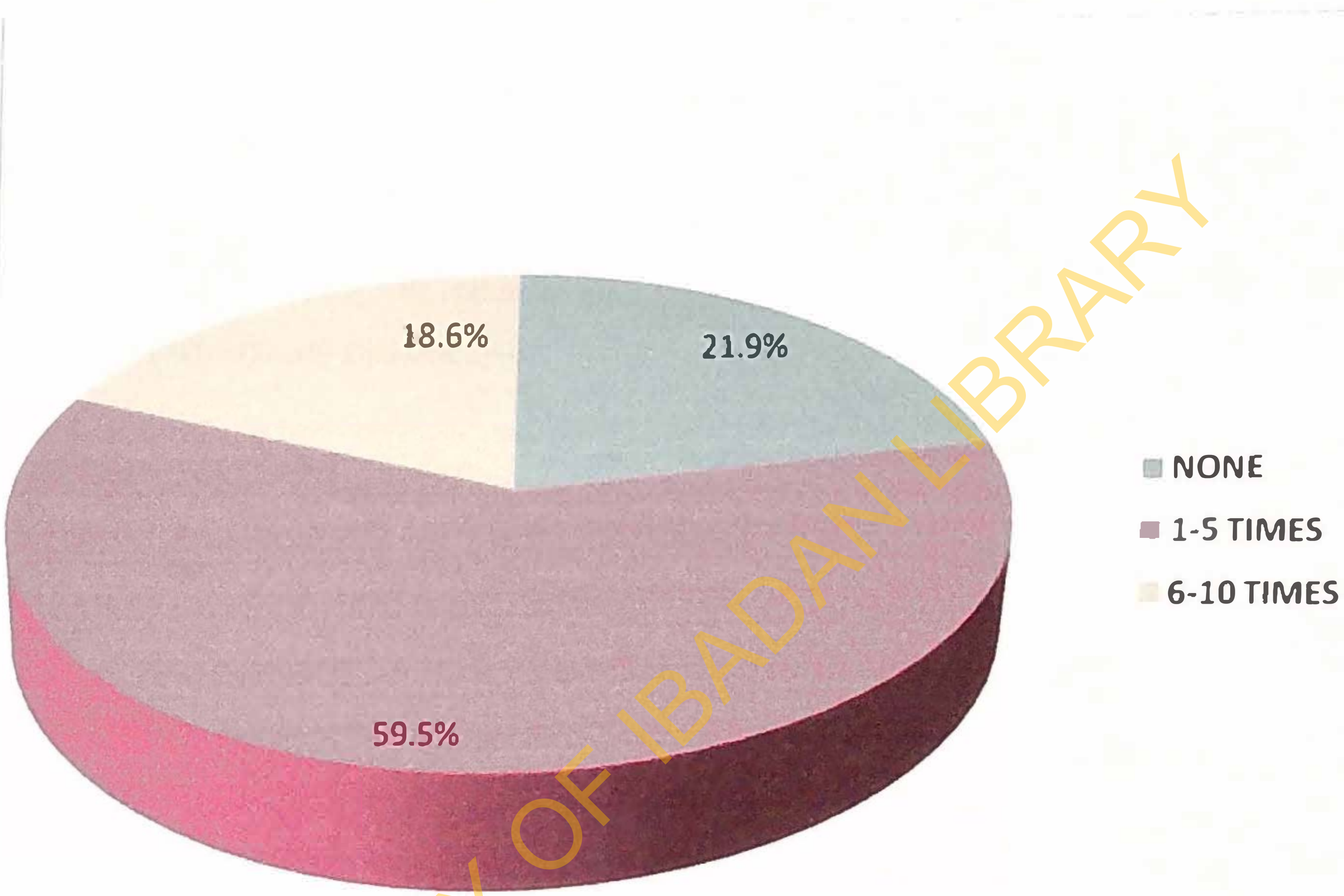


Figure 4.13: Number of attacks in the past one year

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CHAPTER FIVE

DISCUSSION

This chapter presents the implication of the results obtained from the questionnaire survey which includes socio-demographic characteristics,history of asthma, smoking status of parents,prevalence rate of mould growth, respiratory symptoms experienced by respondents, housing types,presence of pets. The results of the environmental assessment carried out in the homes of the participants are also discussed.

5.1 Socio demographic characteristics

The mean age of the participants was 8.9±3.4. The outcome of this study showed that there were more males than females among the asthmatic children and this is similar to previous studies carried out by Dawson et al, (1969), and Davis, (1976). Also in previous studies carried out by von Mutis et al (2000) and Chambtra et al, (1999) male sex was reported as a risk factor for asthma in childhood. According to the results of this study, prevalence of asthma was found to be significantly higher in boys than in girls as in literature. Most of the respondents both among the cases(84%) and the controls(96.9%) were from the yoruba ethnic group and this is because the study was carried out in Ibadan which is predominantly a Yoruba land.

In this study parental educational status was higher among the cases than the controls which was similar to the findings of previous studies.Yung Lee et al, 2003 reported that higher parental educational level was associated with the occurrence of childhood asthma.

In a study carried out by Sandra et al, (2009) they discovered that atopic recurrent wheezing was positively associated with the education of the parents, whereas non-atopic recurrent wheezing was negatively associated. They also found out that when maternal and paternal education were considered separately, only maternal education had a significant influence

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and concluded that aspects associated with a high maternal educational level may play an important role in the development of atopic disorders.

5.2 History of asthma among the participants

As far as genetic factors are concerned, a family history of allergic disease is considered to increase the risk of asthma development (Laitinen et al, 1998; Jaakola et al, 2001). Some genetic markers could impose susceptibility to the effects of environmental factors. For example, children whose parents were atopic (1 or both) were more sensitive to environmental tobacco smoke than those without parental atopy (Jaakola et al, 2001).

This study shows that reported prevalence rate of parental asthma among the cases (56.3%) was significantly higher than the controls(15.6%) which is similar to the findings of Yung Ling Lee et al, 2003 who after adjusting for host factors such as age, parental education level, number of siblings, and maternal smoking during pregnancy, found that parental asthma were significantly related to physician-diagnosed asthma in both sexes. It has also been established from various other studies that a family history of allergic diseases was associated with an increased risk of asthma, suggesting that genetic factors play a central role in the development of asthma(Jaakola et al, 2001, Quah et al, 2000 and Skadhauge et al, 1999)). Some genetic markers could impose susceptibility to the effects of environmental factors in childhood(Jaakola et al, 2001). A recent Malaysian cross-sectional study(Quah et al, 2000) also found that parental asthma was also the strongest determinant of asthma development in children.

In a twin study from Denmark,Skadhauge et al,(1999) 73% of the variation in liability to childhood asthma was explained by genetic factors, which suggested that a family history of asthma was stronger than other risk factors. Yung Ling Lee et al, (2003) in a study carried out on Taiwanese children demonstrated that paternal and maternal asthma were stronger risk factors for childhood asthma in both sexes than were allergic rhinitis and atopic eczema in parents. Children with a parent carrying any atopic disease were also found to have a higher probability of developing asthma in later life than those who lived at home with indoor or outdoor factors. In the mutually adjusted models, hereditary factors—defined as parental

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asthma or allergic rhinitis/atopic eczema —also possessed the highest attributable risks, which were consistent with 55, 900 excess cases annually in Taiwan ,Yung Ling Lee et al, (2003).

5.3 Housing characteristics of the respondents

This study shows that more of the cases lived in a big apartment with the number of room greater than or equal to three suggesting that the size of apartment is not associated with the occurrence of asthma. This however is in contrast to the findings of Splenger et al (2002) who repored that living in smaller houses is associated with an increased risk of of doctor-diagnosed asthma and asthma related symptoms in children.

5.4 Seasonal variation and the occurence of asthma

There were more episodes of asthma attack (65.6%) during the rainy season than the drying season (9.4%). This shows that rainy season could be a predisposing factor for asthma. Aderele (1979) reported rainy season as one of the predisposing factors of asthma in the history of 200 asthmatic children studied. Climate is undoubtedly crucial and it is evident in this study that in Ibadan, less than 100 miles from the south coast, the rainy season is an important predisposing factor of asthmatic attacks.

5.5 Smoking status of parents and the occurence of asthma

This study shows that more parents of the cases(12.5%) smoked than the controls.This is similar to the study carried out by Murray, (1993)who reported that asthmatic children of smoking mothers have more severe asthma than those whose mothers were nonsmokers, and when parents of an asthmatic child give up smoking, the child's condition improves. The greatest effect seems to be related to maternal smoking during pregnancy and/or early infancy(Martinez et al,1992), perhaps due to inflammatory stimuli on lung parenchyma during a period of rapid lungs development and prolonged close exposure to the mother. Exposure to ETS is associated with increase in asthma attacks, earlier symptom onset, increased medication use, and a more prolonged recovery from acute attack. Maternal smoking of half a pack or more per day has been associated with an increased risk of developing asthma in children as reported by Martinez et al,(1992). Gilliland et al, (2001)

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also reported that environmental tobbacco smoke operates as a co-factor with other insults such as intercurrent infection as a trigger of wheezing attacks rather than as a factor that induces asthma.

5.6 Mold and dampness in relation to asthma

This work shows that more of the cases (40.6%) had mold growth on their walls. This is similar to the findings of a previous study by Williamson et al, 1997 who confirmed dampness in homes of 64% asthmatic subjects compared with 41% dwellings of control subjects and also reported that asthma is asociated with living in damp houses and that there appears to be a dose response relationship. In a study carried out by Simoni et al , 2005 the prevalence rate of reported mould/dampness was low(10%) which is at variance with the findings of this study. Simoni et al, however reported that exposure to home mold/dampness accounts for a sizeable proportion of respiratory disorder such as wheeze and asthma among children especially. when the exposure occurs early in life(Simoni et al, 2005). Reported prevalence rates of home mould/dampness range widely around the world: from 10–13% in Russia (Splengler et al, 2002) and Taiwan (Yan et al), to15% in UnitedKingdom (Williamson et al, 1997), Finland, (Kilpelainer et al, 2001)and Poland,(Jedrychowski et al,1998) to 25% in the Nethelands, (Brunekreef et al, 1989)to 27–28% in Sweden (Engwall et al,2001), to 38% in China (Li et al, 1996), and Canada, (Dales et al, 1991), and up to 50% in the USA(Brunekreef et al, 1989) This wide dispersion, despite different climates might also reflect different methods of data collection. Indeed, a significantly lower prevalence of mould/dampness was found in southern than in northern/central Italy,probably due to the milder climate of the South (Simoni et al, 2005)

The association of current wheeze with mould/dampness exposure seemed stronger in children than in adolescents. In the former, the highest OR was found for both exposures. In another study, a stronger association of wheeze with current than with ''ever'' exposure was found in Russian children exposed to mould(Splengler et al,2002). In adolescents, the strongest risk factor for having wheeze was only early exposure, as also found by other authors(Lau et al, 2002). A significant risk for having wheeze after exposure in the first year

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of life was also reported by Belanger et al on infants at risk (with an older asthmatic sibling) (Belanger et al, 2003).The relation between exposure and current asthma was similar for children and adolescents. According to Simoni et al, only early exposure seems to be the strongest risk factor for having asthma (Simoni et al, 2005) which is also consistent with the findings of Forsberg et al, (1997) who reported a more consistent association of asthma with the exposure in the first two years of life.

A case-control study by Williamson et al (1997) used independent assessment of homes along with spirometry and questionnaires. It was discovered that both asthma patients and controls tended to underreport dampness. In this study, a significant association was found between severity of asthma and total dampness and total mold scores. The greater the dampness or mold in the home, the greater the severity of asthma.

Rylander et al(1998) studied two schools in Sweden—one that had a documented mold problem and one that had no mold problem—to measure the effect of mold on airway inflammation and atopy in school children in the first to fifth grades and reported that the incidence of respiratory symptoms was higher in the problem school than the control school regardless of children's atopic status. However, atopic children in the problem school had more respiratory symptoms—specifically, cough with or without phlegm, dry cough at night, or hoarseness—than did either the atopic children in the control school or the non atopic children in the problem school

5.7 Presence of pets in relation to asthma Episodes

In the present study, more of the cases(34.4%) were keeping pets as at the time of the data collection as against 25.5% of the controls. This suggests that the presence of pets in the house may aggravate the condition of asthmatic children. This is similar to the findings of Apelberg et al (2001) who performed a systematic review to synthesize the evidence of the effect of exposure to pets in the home on the risk of asthma and asthma-related symptoms. They concluded that exposure to pets increases the risk of asthma and wheezing in older children (>6 years) but not in children under 6 years. This is also consistent with the findings

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of Kerkhof et al, 2009 which reported that children with pets had more frequent transient or intermittent asthma symptoms.

In contrast, however, three cohort studies provided evidence of a lower risk of asthma among children exposed to pets in early life compared with unexposed children,(Hesselmar et al, 1999; Nafstad et al, 2001).A longitudinal birth cohort study in Norway demonstrated a reduction in the odds of asthma at 4 years of age among children who were exposed to pets in the home when the child was born. (Nafstad et al, 2001) In a prospective birth cohort study, living in a household with at least 1 indoor dog at birth was associated with a lower cumulative incidence of frequent wheezing during the first 13 years of life (Remes et al 2001). Other studies have also found that individuals living with a pet have significantly less asthma or less severe bronchial hyperresponsiveness (Withers et al, 1998, Dekkers et al, 1991). The role of exposure to pets in childhood asthma seems to vary with geographic region, because children with asthma tend to become sensitized to local allergens (Wood et al, 1993:Martinez et al., 1995). Recent epidemiological studies have shown an increase in the prevalence of asthma and allergic diseases. Among etiological factors, the domestic environment and, in particular, pet ownership may be a part of it. The relationship between severity of asthma and pets is still unclear.

5.8 Effects of air quality on asthma

This work revealed that the median concentration of the particulate matter in the homes of the respondents was higher among the controls(34.7 µg/m3) than the cases(28.75 µg/m3) and this also fell within the National Guideline Limit (NGL) of 250 µg/m3. This may be attributable to the fact that most of the cases had higher parental educational status as compared with the controls, hence a cleaner and neater environment. Also, the cases being aware of their state of health tend to get rid of anything that could aggravate the conditions. However this is similar to a study conducted in Sydney, Australia, from 1990 to 1994 which indicated no significant effects of fine particulate (~PM 2.0) on asthma hospitalization for both children and adults. Another study from Birmingham, England (Wordley et al, 1997), where the maximum level of PM_{10} (130.9 $\mu g/m^3$)exceeded the levels found in Toronto and

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Seattle (Scwartz et al,1993)) by over 30%, also showed no significant effect of PM_{10} on asthma hospitalization.

Nevertheless, the results from a number of other studies are at variance with this report. Significant effects of PM_{10} and $PM_{2.5}$ were found in Seattle, with estimated relative risks of 1.05 (19 μg/m3) and 1.04 (11.8 μg/m 3), respectively (Mittleman et al, 1995). Three time series analyses conducted in the same area found significant effects of PM 2.0 and PM 10 on asthma emergency department visits for children younger than 18 years old Norris et al, 1999) and adults younger than 65 years old (Sewartz et al, 1993), and on decreased lung function for elementary school children (Koenig et al, 1993). Stronger effects of PM 10 related to asthma hospitalizations or emergency room visits were also found in Utah Valley (Pope, 1991) and in Sama Clara County,California (Lipsett et al, 1997). Although there is no clear explanation for such a discrepancy regarding the influences of PM_{10} and $PM_{2.5}$ on asthma hospitalizations, the reasons may include variability in population characteristics, natural systems, and the complex mixture of fine particles with a different level and composition over time and space.

Products of indoor combustion, such as nitrogen dioxide from gas stoves and wood smoke, may increase respiratory allergy in people with asthma (Hazucha et al, 1983), but evidence for this is not conclusive. Although this study discovered no difference in the concentration of nitrogen dioxide in the bedroom and living room of both cases and controls, the concentration was higher in the kitchen of the cases (0.02ppm) compared with the control(0.01ppm). This might be as result of usage of Gas cooker among the cases and this is consistent with the findings of Phoa et al. (2004) and Ponsonby et al. 2000, 2001 who reported that exposure to gas heaters and appliances in infancy was found to be a risk for wheezing, asthma, and bronchial hyperresponsiveness in school-aged children. Infants at high risk for asthma who were exposed to higher levels of NO_2—but levels which currently are not considered to be harmful—had increased days of wheezing and shortness of breath according to the report of a study carried out by van Strien et al.(2004). Also, Shima and Adachi (2000) reported that in school-aged children, increased levels of NO_2 were associated

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with increased bronchitis, wheeze, and asthma in girls but not boys. When unflued gas heaters in schools were replaced, NO_2 levels decreased by two-thirds, accompanied by significant reduction in both daytime and nighttime asthma symptoms (Pilotto et al, 2004).

However, controlled exposure studies of subjects with asthma have produced inconsistent results regarding the ability of NO_2 to enhance nonspecific airway responsiveness, with some evidence of a subgroup with increased sensitivity (Hazucha et al, 1983; Bauer et al, 1986; Linn et al, 1986; Moshenin et al, 1987; Roger et al, 1990). Limited data from epidemiological studies suggest that exposure to high levels of NO_2 may be associated with acute decrements in lung function in subjects with asthma (Lebowitz et al, 1985; Goldstein et al, 1988).

SO_2 has clearly been shown to induce acute bronchoconstriction in asthmatic subjects at concentrations well below those required to induce this response in healthy subjects (Sheppard et al, 1980;Linn et al, 1987). However this study shows no significant difference in the median concentration of SO_2 among the cases and controls.

5.9 Perceived asthmatic symptoms among the respondents

The outcome of this study revealed that the most common symptoms of asthma reported among the cases were cough, cold and difficulty with breathing. This is similar to a study carried out by Bacharrier et al, (2007) who reported that in most cases of asthma involving preschool-aged children, the main expression of the disease is a relatively sudden deterioration in health status, usually associated with a cold that results in signs of airway obstruction, cough, and wheezing. Also, Koopman et al, 2001 in his study reported that in preschool children asthma symptoms are wheezing, shortness of breath or dyspnea.

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CHAPTER SIX

CONCLUSIONS AND RECOMMENDATIONS

6.1 Conclusions

This study assessed the environmental risk factors which have potentials for influencing the occurrence of asthma among children in Ibadan. Questionaires and observational checklists were used to determine those environmental risk factors which could influence the occurrence of asthma mong the subjects. Air monitoring equipments were also used to determine the level of some environmental pollutants (sulphur dioxide, nitrogen dioxide, carbon monoxide and particulate matter) in the homes of the participants.

The major proportion of the cases were more knowledgeable about asthma than the controls. This may be due to the fact that the cases due to their state of health had received more enlightenment and education about asthma over time than the controls.

This study revealed that there are more episodes of asthmatic attacks during the rainy season than in the dry season.

The outcome of this study revealed that parental smoking, mold growth, presence of pets, and allergy are the environmental risk factors which are associated with the occurrence of asthma. It was also observed in this work that more of the cases live close to industries and farm suggesting that pollutants and pollens from these sites could influence the occurrence of asthma among these cases.

The indoor air monitoring that was carried out in the homes of the participants revealed a higher concentration of NO_2 in the kitchen of the cases than the controls. This can not be unconnected with the fact that higher proportion of the cases as compared with the controls

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were using gas as a cooking fuel thus suggesting the possibility of linkage between NO_2 and the occurence of asthma.

Majority of the cases reported allergy to dust. However, the in door air quality monitoring that was carried out in the homes of the participants revealed higher concentration of particulate matter in the homes of the controls than the cases. This might be as a result of the fact that the cases being sensitized and educated about their conditions tend to take precautions by keeping their environments free of dusts.

Also the study revealed that most of the cases come from high socio economic group, hence a cleaner and neater environment.

Although this study revealed that allergy to dust has great potentials for the occurrence of asthma further research is needed to ascertain the link between particulates matter and the occurrence of asthma.

This work revealed that more of the cases had history of asthma than the controls thus suggesting the possibility of an interplay between environmental factors and genetic factors in the occurrence of asthma among children.

6.2 Recommendations

1. There should be adequate health education, awareness and advocacy in order to increase people's knowledge about indoor and outdoor air qualities.
2. Pregnant women and parents or caregivers of children with asthma should be particularly encouraged not to smoke
3. Multifaceted programs that focus on educating patients and providing tools for reducing exposure to gaseous pollutants like NO_2 should be introduced.
4. There is need for adequate health education and awareness on prevention and management of asthma.

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5. More researches should be carried out to identify environmental agents that protect against asthma.
6. There should be regular monitoring of ambient air quality in the homes in order to reduce the exposure of children to gaseous pollutants.
7. Indepth researches should be carried out to prove that elimination of indoor and outdoor exposures to environmental factors like mold, pets and gaseous pollutants will result in lower rates of childhood asthma.
8. Government and other stakeholders should ensure that industries are not cited close to residential areas.
9. Parents should avoid keeping pets inside the same building with their children, instead pets should be kept in a separate building far away from the residential places.
10. Parents should be very careful not to expose the children to gas stoves and wood smoke especially at infancy.
11. Keeping of farms around the homes should be discouraged, also bushes around the residences should be cleared as often as possible because of pollens.
12. Further research should be carried out to ascertain the link between particulates matter and the occurrence of asthma.

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APPENDIX 1

ENVIRONMENTAL HEALTH SURVEY

QUESTIONAIRE ON THE ENVIRONMENTAL RISK FACTORS WITH POTENTIALS FOR INFLUENCING THE OCCURRENCE OF ASTHMA AMONG CHILDREN IN IBADAN, NIGERIA.

Daramola Funmilola Modupe is my name, I am a postgraduate student with specialization in Environmental Health in the Department of Epidemiology, medical statistics and environmental health, Faculty of Public Health, University of Ibadan. I am presently carrying out a research titled "Environmental Risk Factors with Potentials for Influencing the Occurrence of Asthma among Children". This research is purely for academic purpose. The findings of this research will be of immense benefit in reducing the occurence of asthma among children. Please be free to express your opinions and I assure you that your responses will be kept confidential. Thanks for your co operation.

Daramola, F.M

Instructions: Please answer the followin questions by ticking any of the alternative responses or filling the blank spaces, which applies to you as the case may be.

SECTION A: SOCIO-DEMOGRAPHIC INFORMATION

1. Age (in years).....
2. Sex: 1. Male[ ] 2. Female[ ].
3. Ethnic group 1. Yoruba[ ] 2. Ibo[ ] 3.Hausa[ ] 4.others (specify).....
4. Religion 1. Christianity[ ] 2.Islam[ ] 3. Traditional[ ] 4. Others(specify).....
5. Educational level 1.None[ ] 2.Nursery[ ] 3.Primary[ ] 4. Secondary[ ]
6. Educational status of father 1.None[ ] 2.Primary[ ]

3.Secondary[ ] 4.Tertiary[ ] 5. Non-formal[ ]

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7. Educational status of mother 1. None[ ] 2.Primary[ ] 3.Secondary[ ]
4. Tertiary[ ]
8. Occupation of father 1.Civil Servant[ ] 2.Business[ ] 3.Industrial worker[ ]
4. Self employed[ ] 5.Others (specify).....
9. Occupation of mother 1. Civil Servant[ ] 2. Business[ ] 3. Industrial worker[ ]
4. Self employed[ ] 5. Others(specify).....
10. Income of father.....
11. Income of mother.....
12. Family size.....
13. No of brothers.....
14. No of sisters.....

SECTION B: FAMILY AND SOCIAL HISTORY

15. Do your parents smoke? 1. Yes[ ] 2. No[ ]
16. If yes, which of your parents? 1. Father[ ] 2. Mother[ ]
17. Does any other member of your family smoke? 1. Yes[ ] 2. No[ ]
18. Did any of your parent smoke when you were born or were young?
1. Yes[ ] 2. No[ ]
19. Is there any history of asthma in your family? 1. Yes[ ] 2. No[ ]
20. If yes, who? 1. Father[ ] 2.Mother[ ] 3. Siblings[ ]
4. Grandparents 5.Others(specify).....

SECTION C: KNOWLEDGE ABOUT ASTHMA

21. What causes asthma?.....
22. What do you think enhances the occurrence of asthma?.....
23. Do you agree that smoke, dust, mold etc. causes asthma and other respiratory diseases?
1. Yes[ ] 2. No[ ]
24. Do you think asthma can be prevented? 1.Yes[ ] 2. No[ ]
25. If yes, how?.....
26. What are the symptoms of asthma?.....
27. Which age group is mostly affected with asthma? 1.Children <5 years[ ]
2.Children >5 years[ ] 3.Adolescents[ ] 4.Adults[ ] 5.Aged people[ ]
6. Others(specify).

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28. Can asthma be cured? 1. Yes[ ] 2.No[ ]

29. How can asthma be cured? 1.By using orthodox medicine 2.By using herbal medicine

3. Others(specify).....

SECTION D: ENVIRONMENTAL HEALTH CHARACTERISTICS

D1: HOUSING STATUS

30. Age of house.....

31. Type of House 1.Bungalow[ ] 2.Duplex[ ] 3. Storey building 4. Others(specify).....

32. No of rooms.....

33. No of persons per room.....

34. No of windows.....

35. Duration in residence.....

36. What are your floors covered with? 1.Carpet[ ] 2.Rugs[ ] 3. Wood[ ]
4. Tiles[ ] 5.Not covered[ ] 6. Others(specify).

37. Indicate which room has which type of covering.

Room	Type of covering
Living room	.....
Master's bedroom	.....
Children's room	.....
Visitor's room	.....

38. How often do you clean the floors? 1. Once daily[ ] 2.Twice daily[ ]
3.Thrice a week[ ] 4. Once a week[ ] 5. Once a month[ ]
6. Others(specify).....

39. How do you clean the floors? 1.By sweeping[ ] 2.By scrubbing[ ]
3.By mopping with water[ ] 4. By sweeping and mopping[ ]
5.Others (specify).....

40. What safety precautions do you carry out when cleaning? 1. Wear nose masks[ ]
2. Wet the floor[ ] 3.Open the windows[ ] 4. No precaution is taken[ ]
5. Others(specify).....

41. Who does the cleaning? 1. Mother[ ] 2. Father [ ] 3. Children[ ]
4. House help[ ] 5. Self[ ] 6. Others(specify).....

42. At what time of the day do you do the cleaning? 1. Morning[ ] 2. Afternoon [ ]
3.Evenings[ ] 4. Anytime of the day[ ].

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D2: INDOOOR AIR QUALITY

43. What type of cooking fuel do you use in your house? 1. Kerosene 2.Firewood
3. Coal [ ] 4. Gas [ ] 5.Others(specify).....

44. Where do you normally do your cooking? 1. In the kitchen inside the house[ ]
2.In the kitchen outside the house[ ] 3.In the verandah[ ]
4. In the room [ ]
5.Others(specify).....

45. How many times do you cook in a day? 1.> 3 times[ ] 2.Three times[ ]
3. Two times[ ] 4. Once in a day[ ] 5.Others(specify).....

46. Is your kitchen roofed? 1. Yes [ ] 2. No[ ]

47. Apart from cooking do you use cooking fuel to heat your house during harmattan?
1. Yes[ ] 2. No[ ]

48. Is your sleeping room far from your cooking area? 1. Yes[ ] 2. No[ ]

49. Do you experience mold growth in your home? 1. Yes[ ] 2. No[ ]

50. If yes, where do you normally experience it? 1. On the walls[ ]
2. On the furniture[ ] 3.On the clothes[ ] 4.others(specify).....

51. How often do you experience it? 1. Always[ ] 2. Seasonal[ ]
3. Others(specify).....

52. Do you use electricity generating sets? 1.Yes[ ] 2. No[ ]

53. If yes, how long have you been using it? 1. <5 years 2. >5 years
3.Others(specify).....

54. Does any other person use electricity generating set in the neighborhoods?
1. Yes[ ] 2. No[ ]

55. Do you have a farm/garden in your residential premises? 1. Yes[ ] 2.No[ ]

56. If yes, how long have you been using it? .....

57. What type of plants do you grow? 1. Vegetables[ ] 2.Fruits[ ]
3.Tubers[ ] 4. Grains[ ] 5.Flowers[ ] 6.Others(specify).....

58.If vegetables or flowers, what type?.....

59. Do you have problems with dusts in the house apart from during the harmattan?
1. Yes[ ] 2. No[ ]

D3: OUTDOOR AIR QUALITY

60. Do you have any industry located close to the house? 1. Yes 2 No [ ]

61. Is there a farm located close to the house? 1.Yes[ ] 2.No[ ]

62. Does your house face major road? 1. Yes [ ] 2. No[ ]

63. If yes, is the road tarred? 1. Yes[ ] 2.No [ ]

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D4: EXPOSURE TO HOUSEHOLD ANIMALS AND VECTORS

64. Which of these pets have you had in your house in the past 12 months? 1.Cat[ ] 2. Dog[ ] 3.Goats[ ] 4.Poultry[ ] 5.None[ ] 6.others(specify).....

65. Do you keep any of these pets presently? 1.Yes[ ] 2.No[ ]

66. If yes, which animals? 1.Cat[ ] 2. Dog[ ] 3.Goats[ ] 4.Poultry[ ] 5.None[ ] 6.others(specify).....

67. Where do you keep them? 1.in a separate building[ ] 2. In one of the rooms in the house[ ] 3. In a cage[ ] 4.Left free[ ] 5.Others(specify).....

68. Are you sensitive to the odor of pets mentioned above? 1. Yes[ ] 2. No[ ]

69.If yes, which of the animals are you sensitive to? .....

70. Do you experience cockroaches infestation? 1. Yes[ ] 2. No[ ]

71.If yes, are you sensitive to their odor? 1. Yes[ ] 2.No[ ]

72. Do you experience rats infestation? 1.Yes[ ] 2.No[ ]

73. Are you sensitive to their odor? 1. Yes[ ] 2. No[ ]

74. Are you sensitive to their hair/fur? 1. Yes[ ] 2. No[ ]

75. If you have experienced any of the following, please indicate state how long.

Problems	In the past 3 months	In the past six months	Others(specify)
a.Difficulty with breathing	[ ]	[ ]	_____
b. Running nose	[ ]	[ ]	_____
c. Coughing	[ ]	[ ]	_____
d. Cold/catarrh	[ ]	[ ]	_____
e. Chest pain	[ ]	[ ]	_____
f. Rashes	[ ]	[ ]	_____
g. Any other	[ ]	[ ]	_____

76. Have you ever seen a doctor regarding any of the problem above? 1. Yes[ ] 2. No[ ]

77.If yes, which of the problems?.....

78. Have you ever been diagnosed of asthma ? 1.Yes[ ]

79. At what age were you diagnosed of Asthma? .....

80. How many times were you admitted or hospitalized in the last one year because of Asthma? .....

81. Do you have asthma presently? 1. Yes[ ] 2. No[ ]

82. Are you currently taking medication for asthma? 1. Yes[ ] 2. No[ ]

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83. Which of these medication are you currently placed on? 1.Beta agonist e.g. Salbutamol[]
2. Inhaled steroid[ ] 3. Flixotide[ ] 4.Other(specify).....

84. How many times did you have attack in the last one year?.....

85. Did you attend school during those periods? 1.Yes[ ] 2. No[ ]

86. When do you normally have asthma attack? 1. During the rainy season[ ]
2.During dry season[ ] 3. Anytime of the year[ ]
4. Others(specify).....

87.How long does the attack last? .....days.

88. How do you feel anytime you have attack? 1. Anxious[ ] 2. Angry[ ]
3.Depressed[ ] 4.Others(specify).....

89. Which of the following are you allergic to? 1. Dust[ ] 2.Smoke[ ] 3.Pollen[ ]
4.Cold[ ] 5.Others(specify).....

90. How often do you eat fresh vegetables? 1.Twice per day [ ]
2.Three times a week[ ]
3. > Three times a week[ ] 4.Everyday[ ] 5.Others(specify).....

91. How often do you take fruits? 1.Daily[ ] 2.Three times a week[ ] 3.> Three times a week [ ] 4.Others(specify).....

92. How often do you take any of these proteinous foods (beans, milk, egg etc.)?
1.Never or Occasionally[ ] 2.Once or twice per week[ ] 3.Three or more times a week[ ] 4. Others(specify).....

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APPENDIX 2

OBSERVATIONAL CHECKLIST

Features	Present	Absent	Remarks
Mold			
Farmlands/gardens			
Pets			
Vectors			
Electricity generating set			
Dusts			
Major road			
Smoke			

Distance of the cooking area to the respondents' bedroom.....

No of persons per household.....

No of windows.....

Area of the windows.....

Type of floor coverings.....

Kinds of cooking fuel used.....

Type of housing.....

Age of housing.....

State of housing.....

Types of roof used.....

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APPENDIX 3

ENVIRONMENTAL MONITORING FORM

INDOOR			
	Living room	Bedroom	Kitchen
SO2			
NO2			
CO			
SPM			
OUTDOOR			
SO2			
NO2			
CO			

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APPENDIX 4

INFORMED CONSENT

My name is Daramola, Funmilola Modupe. I am a student of the Department od Epidemiology, Medical Statistics and environmental Health, Faculty of Public health, University of Ibadan, Ibadan. I am carrying out a research in ibadan in order to find out the environmental risk factors which have potentials for influencing the occurrence of asthma among children. I wil need to ask you some questions, please note that your answers will be kept confidential. You will be given a number and your name will not be written on the form so that your name will never be used in connection with any information you give.

During this exercise, you will be given some questionnaires to fill, environmental monitoring will also be carried out in your homes usiong some equipments. Your honest answer to the questions will help us have better understanding of the causes of asthma diseases in the environment.

You are free to refuse to take part in this programme. You have a right to withdraw at any time if you choose to. We will greatly appreciate your help in responding to the survey and taking part in this study.

CONSENT: Now that the study has been well explained to me and I fully understand the content of the study process. I will be willing to take part in the programme.

.....

Signature.

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IBEERE LATI WADI OUN NIPA AYIKA TO LE JE KI AWON IKO OMO WEERE NI ILU IBADAN TI ORILE EDE NAIJIRIA

Oremi Tooto,

Moje omo akeko ti eka Ajakale-arun, Igbelewon ati Ilera-ayika, ekun ti Ilera Eda ni Unifasity Ibadan.

Mowa lati beere awon ibeere lowo yin si iwadi lori ise iwadi-ijinle mi. Eyi jamo lati wadi oun ayinka to leje ki awon omode ni iko semi-semi. Awon idahun yin yio je ipamo. E jowo eka awon ibeere naa yekeyeke ki esi daun awon ibeere naa ni sise n tele.

Ese fun ifowo sowopo yin.

Emi tiyi,

DARAMOLA F.M.

Ayewo oun ayika to le mun iko semi-semi larin omo were towa ni ilu Ibadan. Ejowo e daun awon ibeere wonyi nipa mi maki tabi ko eroyin si awon aaye ofifo ti oje mo o tabi bose ye o si.

Noomba..... Agbegbe---------- Ojo----------

IPA. A **EKA IDAMO**

1	Ojo ori (ni odun)?	
2	Eda/Ibiwo niyin?	1.Okunri 2. Obinmri
3	Eya won niyin?	1.Yoruba 2. Ibo 3. House 4. Eya miran (se alaye)----------
4	Esin wo le nsin?	1. Kiristeni 2. Musulumi, 3. Abalaye 4. Esin miran (se alaye)----------
5.	Iwe ti e ka?	1. Nkolo rara 2. Jele osimi 3. Alakobere 4. Girama
6	Iwe ti Baba ka?	1. Nkolo rara 2. Alako bere 3. Girama 4. Iwe Giga 5. Eko-ile
7	Iwe ti Momo yin ka?	
8	Ise Baba yin	1. Ise ijoba 2. Ntaja 3. Osise ile ise 4. Alaadani 5. Omiran salaye_____
9	Ise Momo/Iya yin	_____

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10	Owo ti oun wole fun Baba yin	_____
11	Owo ti oun wole fun Momo yin	_____
12	Iye ebi yin	
13	Iye Egbon kunri yin	
14	Iye egbon binrin yin	

IPA B: IWADI EBI ATI ISESI

Se awon obi re nmun siga bi?

15. Nje awon obi yin ma nmun siga bi ?

16. Tobaje beeni, tani ninu won? 1. Baba 2. Iya

17. Nje enikekeni miran ninu obi re nmum siga? 1. Beeni 2. Beeko

18. Nje ikankan ninu obi re ma n mun siga nigba ti won bi o tabi, owa ni kekere bi ? 1. Beeni 2. Beeko

19. Nje itan iko semisemi jeyo ninu ebi yin ri bi? 1. Beeni 2. Beeko

20. Tobaje beeni, taani oni iko naa ri? 1. Baba 2. Iya 3. Awon yokure 4. Obi baba ati iya re 5. Omiran (Se alaye)----------

IPA D: IMO NIPA IKO SEMI SEMI

21. Kinni e ro pe o ma nfa iko semisemi ?----------

22. Kinni e ro pe o nfa ki iko semisemi mun eniyan?----------

23. Se e fowosi pe siga, eruku, irun ti ma nhu lara ile ma nfa iko semisemi ati ipenija gbogbo nipa mimi bi? 1. Beeni 2. Beeko

24. Se iko semisemi naa se demo bi? 1. Beeni 2. Beeko

25. Tobaje beeni, bawo ----------

26. Kinni awon ifian iko semisemi ----------

27. Iru ojo ori wo ni iko naa saba ma nmun? 1.Omo to kere si odun marun 2.Ju odun marun lo

3. Mewa si metadinlogun 4. Mejidinlogun soke 5. Ogbon 6. Omiran (salaye)----------

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28. Se iko semisemi naa se wo bi? 1. Beeni 2. Beeko

29. Bawo ni won se ma n wo 1. Nipa isegun oyinbo 2. Nipan lewe-legbo

3. Omiran (salaye)----------

IPA E: ILERA NIPA AYIKA

E1

30	Odun ile yin	
31	Iru ile yin	Ile Alabode 2. Alakowe/Olowo 3. Alaja/Ofisia 4. Irumiran (salaye)----------
32	Iye yara towa ni ibe	
33	Iye eniyan towa ni yara kan	

33	Iye eniyan towa ni yara kan	
34	Iye ferese towa ni ibe	
35	Odun ti eti ngbe ile yi	

36. Kinni wo fi te ile ile yin?

1. Kapeti 2. Domun-doun 3. Igi 4.Awo 5.Won ko bo rara 6. Omiran (salaye)----------

37. So oun ti efi bo yara wonyi

YARA	IRU IFESILE
Yara mo palo	
Yara okunrin	
Yara awon omo	
Yara Alejo	

38. Igba melo ni ema ntun ile se? 1. Ekan lojo 2. Emeji lojo 3. Emeta lose 4. Ekan lose

5. Ekan losu 6. Omiran (salaye) ----------

39. Bawo lese ma n tun ile se? 1. Gbigba 2. Gbigbon 3 .Ninun 4. Gbigba ati ninun

5. Omiran (Salaye)_____

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40. Kinni ona oun abo si ijamba ti e ma nwo ti eba ntun ile ile yin se? 1. Ma nwo abo imun

2. Ma nwan omi sile 3.Ma nsi awon ferese sile 4.Akise nkankan

5. Omiran (salaye)----------

41. tani o ma ntun ile yin se? 1.Mama 2.Baba 3.Omo 4. Oluran-lowo ile 5. Emi

6. Omiran (salaye)

42. Igbawo ni ojo ni ema ntun ile se? 1. Aaro 2. Osan 3. Ale 4. Igba kigba

E2 BI ATEGUN SE N FE SI NI INUN ILE

43. Iru epo idana wo ni ema nfi dana? 1. Karosin 2. Igi 3. Eedu 4.Afefe-gaasi

5.Iferun igi gige 6. Omiran (salaye)

44. Nibo ni eti ma ndana? 1. Kisinni inu ile 2. Kisinni ni ita 3. Ni ekule 4. Inu yara

5. Omiran(salaye)

45. Igba melo ni ema n dana ni ojo? 1. Okere si emeta 2.Emeta 3. Emeji 4. Ekan ni ojo

5. Omiran (salaye)

46. Se edi aja yara idana yin bi? 1. Beeni 2. Beeko

47. Yato si didana, se ema nlo epo idana lati mu ile yin gbona nigba oye bi?

1. Beeni 2. Beeko

48. Se yara yin jina si ibi idana bi? 1. Beeni 2. Beeko

49 Se e sakiyesi bi irun ara ile ti n hu ni ile yin bi? 1. Beeni 2. Beeko

50. Tobaje beeni, ibo ni e ti nkofiri ihu re ninu ile?

1. Lara igana 2.Ara nkan igi nile 3. Ara aso

4. Omiran (Salaye)_____

51. Oto awon igba melo ti ekofiri re? 1. Igba gbagbogbo 2. Ekokan Lodun

3.Omiran (Salaye)_____

52. Se ema nlo ero amuna wa bi? 1. Beeni 2. Beeko

53. Tobaje beeni, oto igbawo ti eti nlo? 1. Kere si odun-marun 2.Ju odun-marun lo

3. Omiran (Salaye)_____

54. Nje elomiran nlo ero amuanawa ni agbegbe yin bi? 1. Beeni 2. Beeko

55. Nje eni ogba oko ni ile yin bi? 1. Beeni 2. Beeko

56. Tobaje beeni, oto igbawo ti eti nda oko naa?----------

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57. Iru oun ogbin wo ni ema ngbin ? 1. Ewebe 2. Eso 3. Isu 4.Oun oni oro/oka 5. Ododo

6. Omiran----------

58. Tobaje ewebe abi ododo iruwo ni ?----------

59. Nje ema nkofiri eruku ninu ile yin yato si igba oye bi? 1. Beeni 2. Beeko

IRU ATEGUN TI O NFE NI ITA ILE YIN

60. Se ile ise Kankan sunmo ile yin bi? 1. Beeni 2. beeko

61. Se oko /ogba Kankan sunmo ile yin bi? 1. Beeni 2. Beeko

62. Se ile yin koju si titi nla bi? 1. Beeni 2. Beeko

63. tobaje beeni se won da oda ona naa bi ? 1. Beeni 2. Beeko

AJOSEPO PELU ERANKO ILE / ERAN-OSIN ATI OUN TI O NFA AISAN

64. Iru eran osin wo ni oti wo ile yin wa ni arin odun kan seyin? 1. Ologbo 2. Aja

3. Ewure 4. Oun-abiye 5. Kosi aran-osin Kankan 6. Omiran (salaye)----------

65. Nje e nsin okan ninu eran-osin ile bayi ? 1. Beeni 2. Beeko

66. Tobaje beeni, iru eran-osin wo ni? 1. Awon ologbo 2. Awon aja 3. Awon ewure

4. Awon oun abiye

5. Omiran (salaye) _____

67. Ibo ni eti nsin won tabi kowon si? 1. Ile miran 2. Yara kan ninu ile ti e ngbe 3. Inu ile won.

4. Omiran (salaye) ----------

68. Se ema ngbo orun abi awon awon naa ma nrun siyin bi? 1. Beeni 2. Beeko

69. Tobaje beeni, iru osin wo ni? _____

70. Nje ema ngbo orun ayan bi? 1. Beeni 2. Beeko

71. To baje beeni, orun ayan ma nrun siyin? 1. Beeni 2. Beeko

72. Nje eku ma nrun siyin bi? 1. Beeni 2. Beeko

73. Se orun eku naa ma nkoyin ni imu bi? 1. Beeni 2. Beeko

74. Se irun won ma nrun siyin bi? 1. Beeni 2. Beeko

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IPA F : IROYIN SI AWON ONA ILERA

75. Ti eba ti koju eyikeyi ninu awon nkan wonyi ri, ki eso igba ti oti to seyin

ISORO	NI OSU META SEYIN	NI OSU MEFA SEYIN	OMIRAN (SALAYE)
Aile min dada			
b. Ofinkin			
d. Iko			
e. Otutu/Ofinkin			
e. Aya riro			
f. Kuruna/Orisis kokoro jejade ni lara			
g. Oun miran			

76. Nje eti ri Dokita si awon isoro oke naa ri bi? 1. Beeni 2. Beeko

77. Tobaje beeni, ewo ninu isoro oke yi ganni? _____

78. Nje won ti se ayewo iko semisemi ri funyin be? 1. Beeni 2. Beeko

79. Eto omo odun melo ni igba naa? _____

80. Oto igba melo ti won ti dayin daro si ile iwosan nitori iko semisemi ri?

_____

81. Se eni iko semisemi bayi? 1. Beeni 2. Beeko

82. Se e sin lo oogun si iko naa lowowo bi? 1. Beeni 2. Beeko

83. Iru oogun wo ninu iwonyi ni e nlo lowowo? 1. Beta agonist, apeere Salbutamol
2. Steroid fifa si imu, apeere Flixotide 3. Omiran (Salaye)_____

84. Emelo ni eti ni idojuko iko yi larin odun kan seyin? _____

85. Nje e ma nlo Ile-iwe nigba ti iko yi bade bi? 1. Beeni 2. Beeko

86. Igbawo ganni ema n ni iko semisemi gan? 1. Nigba ojo 2. Nigba eerun
3. Nigba kigba ninu odun 4. Omiran (Salaye) _____

87. Igbawo ni aisan iko yi ti to? _____ ojo

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88. Bawo ni oti ma nseyin nigba ti iko yi bade? 1. Oma ndun moyin 2. Oma nbiyin ninu
3. Oma ndayin gunle 4. Omiran (Salaye)_____

89. Iru ewo ninu iwonyi ni oun ti otete ma n mun iko semisemi yi wa? 1. Eruku 2. Eefin
3. Orun-ododo 4. Otutu 5. Omiran (Salaye)_____

90. Ewebe jije yin ma nto emelo bi? 1.Emeji lojo 2. Emeta ni ose 3. Kere si emeta lose
4.Gbogbo ojo 5. Omiran (Salaye) _____

91. Emelo ni ema nje eso? 1. Ni ojojumo 2. Emeta ni ose 3. Kere si emeta lose
4. Omiran (Salaye) _____

92 Emelo ni ema nje awon ounje asara loore yi (Ewa, Miliki, Eyin ati be be lo)?
1. Nkiije abi ekokan 2. Ekan si emeji lose 3. Emete si opo igba lose
4. Omiran (Salaye) _____

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