EFFECT OF WOOD SMOKE (BIOMASS) ON SOME LUNG FUNCTION TESTS, CARDIOVASCULAR PARAMETERS AND HAEMATOLOGICAL INDICES OF WOMEN IN ZARIA, NIGERIA

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DECLARATION

I declare that the work in this thesis entitled "THE EFFECT OF WORD SMOKE ON SOME LUNG FUNCTION TEST, CARDIOVASCULAR PARAMETERS AND HAEMATOLOGICAL INDICES OF WOMEN IN ZARIA, NIGERIA" has been performed by me in the Department of Human Physiology under the supervision of Prof. M. A. Ali, Prof. M. Mabrouk and Dr. A.B. Adelaiye. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this thesis was previously presented for another degree or diploma at any University.

....

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CERTIFICATION

This thesis entitled "THE EFFECT OF WOOD SMOKE (BIOMASS) ON SOME LUNG FUNCTION TESTS, CARDIOVASCULAR PARAMETERS AND HEMOTOLOGICAL INDICES OF WOMEN IN ZARIA, NIGERIA" by Akor-Dewu Maryam meets the regulations governing the award of the degree of Masters of Science in Human Physiology at Ahmadu Bello University and is approved for its contribution to knowledge and literary presentation.

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ABSTRACT

Wood smoke is a complex mixture of substance produced during the combustion of wood. The major emissions from wood stoves are carbon monoxide, organic gases, particulate matter, oxides of nitrogen and sulphur. Toxic compounds as well as carcinogenic substances are also produced like benzopyrenes, aldehydes, phenols, cresols etc.

Irritants of the lungs like oxides of nitrogen and sulphur, phenols, particulate particles cause inflammation of the air passages inevitably causing/leading to obstruction of the lungs and other more severe effects.

The aim of this study was to investigate the effect of wood smoke on some lung function test, cardiovascular parameters and haematological indices in women in Zaria, Nigeria.

This study was conducted on 60 female subjects grouped into 2, Group 1 (control group) n = 30 were women that used other sources of fuel for cooking. Group 2 (study group) n = 30 were women that work as cooks in restaurants and use wood as a source of fuel. They were all matched for age and height.

Cardiovascular parameters investigated were blood pressure, pulse rate and mean arterial blood pressure. The Mean $\pm$ SEM of systolic and diastolic blood pressure of the two group (gp 1 & 2) were 113.67$\pm$ 2.65 and 111.17$\pm$ 3.44 mmHg and 76.00$\pm$2.15 and 75.00 $\pm$ 2.46 mmHg respectively. Mean arterial blood pressure were 89.78$\pm$ 1.86 and 87.06$\pm$ 2.69 mmHg for the two groups and pulse rate was 75.60$\pm$ 1.29 and 77.93 $\pm$ 1.56 beats/men respectively. There was no significant statistical difference in cardiovascular parameters between the two groups ($P > 0.05$).

Haematological indices such as packed cell volume for the two groups were 41.53± 0.60 and 41.43 ± 0.76. Haemoglobin concentration 13.84±0.20 and 13.81± 0.25 for group 1 and 2 respectively. The mean ± SEM of Differential white blood cell count, Neutrophils, lymphocytes, monocytes and Eosinophils for the two groups were 59.43± 0.57 and 59.03± 0.55, 31.87±0.67 and 32.50± 0.79, 6.47± 0.21 and 6.47± 0.25, and 3.04± 0.26 and 3.10± 0.28 respectively. There were no significant difference found between the two groups (of the respective parameters) $P > 0.05$. The Mean ±SEM of Respiratory function tests such as peak expiratory flow rate (PEFR) predicted and actual were 3.96± 0.10 and 3.88± 0.04, 4.87± 0.13 and 4.43± 0.12 respectively for the two groups. The Mean ± SEM of forced expiratory volume in 1 second percent (FEV_1%) ratio was found to be 81.83± 1.33 and 78.39± 1.61 for group 1 and 2 respectively.

There was no significant statistical difference in predicted peak expiratory flow rate and forced expiratory volume in 1 second percent ratio between the two groups ($P > 0.05$). There was a significant difference in actual peak expiratory flow rate between the two groups ($P<0.05$).

This result indicates that women using biomass fuel (wood) are more liable to have reduced pulmonary functions than women using kerosine or gas as shown by the reduction in PEFR.

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LIST OF ABBREAVIATIONS

(1)	ALRI	-	Acute lower respiratory illness
(2)	ARI	-	Acute Respiratory Infection
(3)	COPD	-	Chronic Obstructive Pulmonary disease
(4)	FVC	-	Forced Vital Capacity
(5)	TET	-	Total Expiratory Time
(6)	SVC	-	Slow Vital Capacity
(7)	FEV_T	-	Forced Expiratory Volume Timed
(8)	FEV_1%	-	Forced Expiratory Volume in 1 second percentage
(9)	PEFR	-	Peak Expiratory Flow Rate
(10)	BSA	-	Body surface area
(11)	RV	-	Residual Volume
(12)	FRC	-	Forced residual capacity
(13)	TLC	-	Total Lung Capacity
(14)	GOLD	-	Global Initiative for Chronic Obstructive Lung Disease
(15)	ABP	-	Arterial Blood Pressure
(16)	SBP	-	Systolic blood Pressure
(17)	PP	-	Pulse Pressure
(18)	DBP	-	Diastolic Blood Pressure
(19)	MABP	-	Mean Arterial Blood Pressure
(20)	WBC	-	White Blood Cells
(21)	RBC	-	Red Blood Cells
(22)	PAH	-	Polycyclic Aromatic Hydrocarbons
(23)	CO	-	Carbon Monoxide
(24)	COHb	-	Carboxyhaemoglobin

(25) DNA - Deoxyribonucleic Acid

(26) NO_2 - Nitrogen dioxide

(27) AM - Alveolar Macrophage

(28) SPM - Suspended Particulate Matter

(29) CAPS - Concentrate air Particles

(30) COM - Coefficient of Haze

(31) ER - Emergency Room

(32) COA - Chronic airway obstruction

(33) PCs - Pulmonary C fibers

(34) DMTU- Dimetrylthiourea

(35) SIAHR- Smoke-induced airway hyper responsiveness

(36) PPEFR- Predicted Peak Expiratory Flow Rate

(37) OAD - Obstructive Airways Disease

(38) LPG - liquid Petroleum gas

(39) LNG - Liquefied Natural gas

(40) EAP - Environmental Protection Agency

(41) PCV - Packed Cell Volume

(42) ESR - Erythrocyte Sedimentation Rate

(43) CVS - Cardiovascular System

(44) BMI - Basal Metabolic Index

(45) APEFR- Actual Peak Expiratory Flow Rate

CHAPTER 1

INTRODUCTION

1.0 General Introduction

The smell of wood smoke evokes fond memories for many people, but for others it has become a danger signal (Ammann, 1986). Wood crop residues and animal dung (biomass) is used by approximately half of the world's population as cooking and /or heating fuel. These fuels are handled and combusted primarily by women, who are largely responsible for chores such as cooking and are often involved in any household industries which involve cooking such as food processing (Aristanti, 1996).

When we first think of negative health effects connected to biomass fuel use, we invariably think of smoke. Indeed smoke is a health problem, but is not only because biomass is being used, it is also due to the stoves used to combust it and often poorly ventilated environment where the combustion takes place. (Aristanti,1996).

The use of wood stove has increased greatly in the past decade, causing concern in many communities about the health effect of wood smoke. Wood smoke is known to contain such compounds as carbon monoxide, nitrogen oxides, sulfur oxides, aldehydes, polycyclic aromatic hydrocarbons and fine respirable particulate matters. All of these have been shown to cause deleterious physiologic responses in laboratory studies in humans. Some compounds found in woodsmoke include; benso (a) pyrene and formaldehyde are possible human carcinogens. Fine

particulate matter has been associated with decreased pulmonary function in children and with increased chronic lung disease in Nepal, where exposure to very high amounts of woodsmoke occurs in residences (Pierson *et al*, 1989).

The particles in woodsmoke are too small to be filtered by the nose and upper respiratory system so they wind up deep in the lungs. They can remain there for months causing structural damage and chemical changes. Poisonous and cancer causing chemicals often enter the lungs by adhering to tiny particulate matter (Ammann, 1986).

Woodsmoke exposure causes decrease in lung functions and increase in secondary existing lung disease (which increases in smoke concentration or exposure time) (Ammann,1986). It also aggravates heart problems, the occurrence of respiratory illness in children has been shown to increase with predisposition and exposure to woodsmoke (Lewis, 1988). Acute lower respiratory illness (ALRI) has also been associated with exposure to domestic smoke, while cooking with wood burning stoves was associated with higher indoor air concentration of respiratory particles and with an increased risk of ALRI in Navajo children (Robin *et al* 1996).

Woodsmoke aggravates bronchitis, bronchial asthma, emphysema and pneumonia. Long term exposure may lead to emphysema, chronic bronchitis, arteriosclerosis, nasal, throat, lung, blood and lymph tissue cancers (based on animal studies) (Ammann, 1986).

Acute Respiratory Infection (ARI) is one of the top killer of children in developing countries, about five million deaths/year. In many developing communities, it is the primary cause of infant death. (Aristanti,1996). Women are more biologically sensitive to carbon monoxide (CO) emissions, during pregnancy, excessive or prolonged exposure represents an even more significant hazard. Carbon monoxide interference with the oxygen carrying capacity of the blood contributes to increased rates of low birth weight or still births. (Smith,1996).

Chronic obstructive lung disease in adults, for which tobacco smoking is now the major risk factor in the developed countries, is known to result from excessive exposure to air pollution. Several studies have shown that non-smoking women who have cooked on biomass stoves for many years exhibit a higher prevalence of decreased lung volume and capacities and other chest diseases than women who have had less contact with biomass stoves. Indeed in an area of rural Nepal, nearly 15% of non-smoking women had chronic bronchitis , a high rate for non-smokers. Right-side heart failure has been found to be prevalent and to develop earlier than average in non-smoking women who cook with biomass in India and Nepal (Pandey, 1989).

As a result of the possible health hazards associated with smoke, it is surprising that there are few documented effects of woodsmoke on respiratory functions and haematological indices in Africa especially in Nigeria.

1.1 Justification

The precise correlation between the use of Biomass fuels and health consequences has rarely been pinpointed. This is due to large numbers of confounding factors and the modest resources available for such research (Broakman, 1996).

There are some evidence to suggest that exposure to indoor pollutants like tobacco smoke may increase morbidity from chronic obstructive pulmonary disease (COPD). However, no study has clearly demonstrated a similar relationship between "COPD" and exposure to various cooking fuels (biomass) (Behera,1995).

In door air pollution due to the burning of biomass fuels has been shown to be an important factor increasing the prevalence of acute respiratory infections in children, and chronic obstructive pulmonary disease (COPD) often leading to heart failure in adult women (Nathan,1996).

The total human exposure to pollutants is much more substantial in the homes of the poor in developing countries than in the outdoor air of the cities in the developed world, which has received the vast majority of attention in the form of air pollution research and control efforts (Aristanti,1996).

As a result, this study becomes very relevant since there are practically very few studies on the effects of woodsmoke on both pulmonary functions and haematological indices in Africa and especially in Nigeria (Peters *et al*, 1999).

1.2 Aims and Objectives

The aims of this research project was to investigate the effect of Biomass (with emphasis on wood smoke and crop residues) on some respiratory function and haematological variables by measuring ventilatory lung functions and haemotological indices amongst (1) women exposed to wood smoke and (2) those that were not exposed.

The objectives of this work were to find out if there were any differences between these two groups for:

A) Lung function tests such as

(i) Predicted Peak Expiratory Flow Rate (PPEFR)

(ii) Actual Peak Expiratory Flow Rate (APEFR)

(iii) Forced Vital Capacity (FVC)

(iv) Forced Expiratory Volume in 1 second (FEV_1)

(v) Forced Expiratory Volume Ratio % (FEV_1/FVC x 100)

B) Cardiovascular Parameters such as

i) Pulse rate

ii) Systolic Blood Pressure

iii) Diastolic Blood Pressure

iv) Mean Arterial Blood Pressure

C) Haematological Indices

i) Hemoglobin Concentration (Hb Conc.)

ii) Haematocrit Value/Packed Cell Volume (PCV)

iii) Differential Leucocyte Count

D) Anthropometric Indices such as

i) Age in years

ii) Weight in kilogram

iii) Height in meters

iv) Body Mass Index (BMI) = weight (kg)/Height (m^2) was calculated.

These parameters were all measured and presented in tables.

CHAPTER 2

LITERATURE REVIEW

Injury or illness can result in a significant degree of pulmonary dysfunction. This dysfunction can severely limit the length or quality of an individual's life. In acute forms, pulmonary disorders can cause death within minutes e.g pulmonary thrombosis. Chronic disorders can take years before resulting in an individual's death e.g bronchial asthma, but many of those years may be spent with serious disability and poor quality of life. To be best managed, pulmonary disorders must be detected and treated at the earliest possible time (Madama, 1998).

2.1.0 Significance of Pulmonary Function Tests

The pulmonary function tests are performed as they:

a) Provide an objective assessment of pulmonary disability, which no other investigation can provide.

b) Are used to assess the integrated function of the structures that comprise the pulmonary system.

c) Aid in resolving whether such symptoms and signs as dyspnea, cough, cyanosis and polycythaemia are of respiratory origin

d) Are helpful in managing patients, already recognised as having pulmonary diseases.

e) Are carried out in epidemiological surveys on population groups suspected of having acquired lung disease through dust, smoke and exposure (Jardins, 2002).

2.1.1 Pulmonary Function Tests

Ventilatory function tests enable a better understanding of pulmonary physiology in subjects of all ages, sex, profession and occupation groups. They provide an understanding of functional changes in the lungs and their significance from the viewpoint of diagnosis, more so in chronic obstructive pulmonary disease (Ali, 1983).

2.1.2 Pulmonary Mechanic Measurements

(1) Forced vital capacity (FVC)

The FVC is the maximum volume of air that can be exhaled as forcefully and rapidly as possible after a maximal inspiration. The FVC is the most commonly performed pulmonary function measurement. In the normal individual, the total expiratory time (TET) required to completely exhale the FVC is 4 to 6 second. In obstructive lung disease the TET increases, TETs greater than 10 seconds have been reported in these patients. In the normal individual, the FVC and the Slow Vital Capacity (SVC) are usually equal. In the patient with obstructive lung disease, SVC is often normal and the FVC is usually decreased because of air trapping. The FVC is also decreased in restrictive lung disorder (e.g.

pulmonary fibrosis, adult respiratory distress syndromes etc), due to low vital capacity associated with restrictive disorders. The TET needed to exhale the FVC in a restrictive disorder, however is usually normal or even lower than normal, because the elasticity of the lung is high (low compliance) in restrictive disorders (Jardins,2002).

(2) Forced Expiratory Volume Timed (FEV_T)

The FEV_T is the maximum volume of air that can be exhaled within a specific time period. This measurement is obtained from an FVC, the most frequently used time period are 1, 2 & 3 second. The results determined for the FEV_T provide an indicator to the average flow rate over a time interval for the as FEV_T becomes longer (e.g FEV_2, FEV_3) flow rates later in expiration and through smaller airways are being measured. Patients with obstructive pulmonary disease have a decreased FEV_T. Patients with restrictive lung disease also have a decreased FEV_T, primarily due to the low vital capacity associated with such disease. The FEV_T decreases with age (Madama 1998; Jardins,2002).

(3) Forced Expiratory Volume 1 sec/Forced Vital Capacity Ratio (FEV_1/FVC Ratio) /Forced Expiratory Volume in 1 second percentage (FEV_1%).

The FEV_1/FVC ratio is the comparison of the amount of air exhaled in 1 second to the total amount exhaled during an FVC maneuver. Because the FEV_1/FVC, ratio is expressed as a percentage, it is commonly referred as a forced expiratory volume in 1 second percentage ($FEV_{1\%}$). As mentioned previously, the normal adult exhales 83 percent or more of the FVC in 1 second (FEV_1). Thus,

under normal conditions the patient's FEV_1% should also be 83 percent or greater. Clinically, however, an FEV1% of 65 percent or more is often used as an acceptable value in older patients.

Collectively FEV_1, FEV_2, and the FEV_1% are the most commonly used pulmonary function measurement to:

(a) Determine the severity of a patient's obstructive pulmonary disease and

(b) Distinguish between an obstructive and restrictive lung disorder. The key pulmonary function differences between an obstructive and restrictive lung disorder are as follows: In obstructive lung disorders, both the FEV_1 and the FEV_1% are decreased. In restrictive lung disorders, the FEV_1 is decreased, but the FEV_1% is normal or increased. (Jardins2002).

(4) Peak Expiratory Flow Rate (PEFR)

The PEFR (also known as peak flow rate) is the maximum flow rate that can be achieved during an FVC maneuver. The PEFR is most commonly measured at the bedside using a small, hand-held flow-sensing device called a Peak Flow Meter. The PEFR reflects initial flows originating from the lung airways during the first part of an FVC maneuver. Thus the greater the patient effort, the higher the PEFR value. The average PEFR for normal healthy men aged 20-30 years is about 10L/sec (600 L/min) and for women of the same age, about 7.5L/sec (450 L/min). The PEFR decrease with age and also in obstructive lung disease (Jardins,2002).

2.1.3 Predicted Normal Values For Pulmonary Function

Assessment of pulmonary function is based on comparing a subjects test result against the predicted normal values for that subject. Predicted normal pulmonary function values make it possible to determine whether a given subject's test results indicate the presence of an abnormality. Normal pulmonary values are affected by the physical characteristics of the subject. Among subjects of the same sex, height is the single greatest factor that affects pulmonary function. Taller subjects have, over all, greater pulmonary function values. This is especially true for lung volumes and diffusing capacity values. The weight of the subject is sometimes taken into consideration along with height. This is often done by using the values for the subject's body surface area (BSA). The effect of weight can be seen when comparing lung volume values for adult subjects of the same height. Beginning with lesser values of normal weight for a given height, there is an increase in the lung volumes as weight increases. This increase is related to the effects of greater muscularity. As weight continues beyond the normal range, however, there begins to be a decrease in lung volumes. This decrease is related to the effects of obesity (Madama,1998).

The age of the subject has an effect on the normal predicted values used for pulmonary function parameters. With adult subjects, especially over the age of 25, advancing age tends to have a deteriorating effect on normal pulmonary function values. As age increases, normal values decrease for

- Lung volumes (exceptions are RV and FRC)

- Expiratory flow rates
- Diffusing capacity

A complicating factor is the loss of height that normally occurs with age. Aging alone does affect the lungs in a way that reduces pulmonary function. The sex of the subject affects predicted normal pulmonary function values. Male subjects tend to demonstrate greater lung volumes, expiratory flow rates, and diffusing capacities than female subjects of same height and age (Madama, 1998).

There are secondary characteristics that also affect predicted values for pulmonary function. The race or ethnic origin of the subject has some effect on normal predicted values. Black and Oriental subjects tend to demonstrate smaller predicted normal values for a given height and age than subjects of European origin. For Black subjects, many laboratories reduce the values that are predicted for normal lung volumes (TLC, FVC, etc). The values are generally reduced to between 85% and 90% of the values normally predicted for subjects of European descent. It should be noted however, that values for predicted expiratory flow rates are the same for both Black and European descent subjects who have the same predicted FVC values (Madama, 1998). The effects of altitude and other environmental factors on predicted normal values for pulmonary function are not well established. Air pollution and other environmental factors (e.g. rural versus urban living) may have some effect on predicted normal values. Pollution caused by high levels of “reducing” type agents (by-products of high-sulfur coal

combustion) has been demonstrated to cause deterioration in pulmonary function (Madama 1998).

Example of Predictive Equation of PEFR

Male (0.0567x H) – (0.024 x A) + 0.225

Female (0.0354x H) – (0.018 x A) + 1.130 for predicted values of peak expiratory flow rate (Madama,1998).

2.1.4 Chronic Obstructive Pulmonary Disease (COPD)

Definition of COPD

The recently published and widely accepted definition from the Global Initiative for Chronic Obstructive Lung Disease (GOLD) has classified COPD as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles and gases. The subtypes of COPD (asthmatic, bronchitis, and emphysema) may have both different etiologies and outcomes, along with different treatment strategies. (Mannino and McGonigle, 2003).

Air flow limitation is the slowing of expiratory air-flow as measured by a spirometry, with a persistently low FEV_1 and a low FEV_1/FVC ratio despite treatment. The GOLD definition for air flow limitation is an FEV_1/FVC ratio of < 70%. The GOLD definition of COPD classified reversibility as an FEV_1 increase of 200ml and 12% improvement above baseline FEV_1 (Mannino and

McGonigle, 2003).

Epidemiology of COPD

In 2000 an estimated 10 million US adults reported physician-diagnosed COPD. Data from the Third National and Nutritional Examination Survey (NHAPES III), however, estimate that among 11 million US adults with evidence of low lung function, < 40% reported a diagnosis of COPD or asthma, suggesting that COPD is underdiagnosed. In 2000, COPD was responsible for 8 million physician – office and hospital outpatient visits, 1.5 million emergency department visits, 726,000 hospitalizations, and 119,000 deaths. The most dramatic change over the 21 – year period analyzed in the Centers for Disease Control Report was the increase in the COPD death rate for women, from 20.1 per 100,000 in 1980 to 56.7 per 100,000 in 2000 (Mannino and McGonigle,2003).

Smoking is the primary risk factor for the development and progression of COPD: however, < 25% of the smokers develop COPD and about 15% of COPD –related mortality occurs in non smokers, who had never smoked suggesting that other factors are important. Identified factors other than smoking that are important in COPD development and progression include asthmas and bronchial responsiveness, occupation, genetic factors, air pollution, sex, socio-economic status, nutrition and childhood exposures. Understanding how these factors work together to cause diminished lung function in never-smokers may improve our understanding of and treatment

options for COPD in the general population. The majority of COPD in the developing world probably occurs in never-smokers (Mannino and McGonigle 2003).

High-dose irritant exposures (i.e. firesmoke, etc) that cause a life – threatening acute pulmonary toxicity may result in reactive airways dysfunction syndrome or brocnhiolitis. Both outdoor and indoor air pollutants can cause exacerbations of existing lung disease. The primary outdoor air pollutants of interest include ozone, particulate matter, and sulfur dioxide; important indoor pollutants include environmental tobacco smoke, wood smoke and nitrogen oxides. Exposure of indoor air pollutants can frequently result in higher exposures than one would obtain from outdoor exposures. Tobacco smoke, wood smoke, and cooking fumes have all been associated with the development of COPD. As was demonstrated with outdoor exposures, indoor exposures also result in more COPD among people with genetic risk factors (Mannino and McGonigle, 2003).

2.2.0 Normal Arterial Blood Pressure

The blood pressure in the brachial artery in young adults in sitting or lying position at rest is approximately 120/70 mmHg. It is appreciably lower at night and is lower in women than in men. Since the arterial pressure is the product of the cardiac output and the peripheral resistance, it is affected by conditions that affect either or both of these factors. Emotion, for example increases the cardiac output, and it may be difficult to obtain a truely resting blood pressure in an excited or tense individual. In

general, increases in cardiac output increases the systolic pressure, whereas increases in peripheral resistance increase the diastolic pressure. There is controversy about where to draw the line between normal and elevated blood pressure levels particularly in older patients. However, the evidence seems incontrovertible that in apparently healthy humans both systolic and the diastolic pressure rise with age. An important cause of the systolic pressure rise is decrease distensibility of the arteries; at the same level of cardiac out put, the systolic pressure is higher in old subjects than in young ones because there is less increase in the volume of the arterial system during systole to accommodate the same amount of blood (Ganong, 2003).

2.2.1 Factors Determining Blood Pressure

In order to have a blood pressure there must be a cardiac output and a resistance to blood flow in the systemic circulation. This resistance is termed the peripheral resistance.

Blood pressure = Cardiac Output x Peripheral Resistance

This resistance to blood flow lies mainly in the small arteries of the body which are termed arterioles. It is these small diameter vessels that offer the greatest resistance to blood flow. The capillaries are even smaller vessels than the arterioles, but although each individual capillary will offer a higher resistance than an arteriole, there is a larger number of capillaries in parallel supplied by each arteriole As a result there is a large number of alternative pathway for blood to take in its passage from an arteriole through to the veins, and because of this the capillary network does not offer such a resistance to blood flow as the arteriole supplying it (Green, 2002).

By decreasing the caliber of the outflow tubing, the resistance against which the heart pumps (peripheral resistance) can be increased. When the peripheral resistance is increased, the heart puts out less blood than it receives for several beats. The resistance of blood flow is determined not only by the radius of the blood vessels (Vascular hindrance) but also by the viscosity of the blood. Plasma is about 1.8 times as viscous as water, whereas whole blood is 3 -4 times as viscous as water (Ganong,2003).

2.2.2 Viscosity of Blood

The resistance offered by an arteriole of a given size depends on the viscosity of the blood. Blood is a sticky viscous fluid which offers two to three times more resistance than plain water or saline. The viscosity of blood depends partly on the plasma and partly on the number of red cells present, (Green, 2002).

The viscosity of blood is usually constant but it will be reduced if large quantities of saline are given. Plasma substitutes, are viscous. A reduction in the circulating red cells (anaemia) has some effect on the viscosity but it will be increased in polycythaemia (high red cell count). A low blood viscosity will be associated with a low blood pressure and a high viscosity with a high blood pressure (Green, 2002).

2.2.3 Cardiovascular Regulatory Mechanisms

In humans and other mammals, multiple cardiovascular regulatory

mechanisms have evolved. These mechanisms increase the blood supply to active tissues and increase or decrease heat loss from the body by redistributing the blood. In the face of changes such as hemorrhage, they maintain the blood flow to the heart and brain. When the challenge faced is severe, flow to these vital organs is maintained at the expense of the circulation to the rest of the body (Ganong 2003).

Circulatory adjustments are effected by altering the output of the pump (the heart), changing the diameter of the resistance vessels (primarily the arterioles) or altering the amount of blood pooled in the capacitance vessels (the veins). The caliber of the arterioles is adjusted in part by autoregulation. It is also increased in active tissues by locally produced vasodilator metabolites. It is also affected by substances secreted by the endothelium, and is regulated systematically by circulating vasoactive substances and the nerve that innervate the arterioles. The caliber of the capacitance vessels is also affected by circulating vasoactive substances and by vasomotor nerves. The systemic regulatory mechanisms synergize with the local mechanisms and adjust vascular responses throughout the body (Ganong, 2003).

2.2.4 Blood Pressure

The Arterial Blood Pressure (ABP): is the lateral pressure exerted by the column of blood on the arterial wall.

The Systolic Blood Pressure (SBP): is the maximum level of arterial Blood Pressure, that is reached during the rapid ejection phase of ventricular systole. For a normal adult male it is about 120 mmHg (normal range is 110-140mm Hg). Ref. (Sembulingam and Sembulingam, 2000; Abu-Shita et al 2003).

The Pulse Pressure (PP) is the difference between the systolic and the diastolic blood pressure values (SBP – DBP). It is the cause of arterial pulsation. For a normal adult male the pulse pressure = 120 – 80 = 40 mmHg.

The Diastolic Blood Pressure (DBP); is the minimum of arterial blood pressure, that is reached during the isometric contraction phase of ventricular systole. For a normal adult, it is about 80mmHg (normal range is 60-80mm Hg). Ref. (Sembulingam and Sembulingam, 2000; Abu-Shita et al 2003).

The Mean Arterial Blood Pressure (MABP) is calculated by integrating the arterial pressure value overtime. Because the diastolic time is longer than the systolic the mean arterial pressure is closer to the diastolic than to the systolic pressure level. It equals; the diastolic blood pressure + $^{1}/3$ the pulse pressure

i.e (DBP + $^{1}/3$ PP) or approximately 93 mmHg.

The heart of a normal adult male beats automatically and regularly at a rate of 75 beats/min during rest. The normal range of heart rate is between 60 and 100 beats/min. A heart rate higher than 100 beats/min is called tachycardia. A rate lower than 60 beats/min is called bradycardia (Sembulingam and Sembulingam, 2000; Abu-shita *et al* 2003).

2.2.5 Blood

Blood consists of numerous specialised cells that are suspended in a liquid substance called plasma. The cells in the plasma include the erythrocytes (red blood cells), leukocytes (white blood cells) and thrombocytes (or platelets) (Jardins 2002)

A) **Erythrocytes**

Erythrocytes constitute the major portion of the blood cells. In the healthy adult there are about 5 million red blood cells a range at 4.0 to 5.5 million RBC in each cubic millimeter of blood (mm^3). The percentage of RBCs in relation to the total blood volume is known as the hematocrit. The normal hematocrit is approximately 40-54 percent in the adult man and 35 – 47 percentage in the adult woman (Sembulingam and Sembulingam 2000; Abu-Shita *et al* 2003).

In the normal newborn, the hematocrit ranges between 45 and 60 percent. (Jardins 2002). The range of haemoglobin concentration is within 12.0 – 16.0g/dl (Flemings, 1976).

B) **Leukocytes**

The primary function of the leukocytes or white blood cells (WBCs) is to protect the body against bacteria, viruses, parasites, toxins, and tumors. The leukocytes are far less numerous than RBCs, averaging between 4000 and 11,000 cells/mm^3. Leukocytes are grouped into two major categories on the

basis of structural and chemical characteristics; Granulocytes (neutrophils, eosinophils and basophils) contain specialized membrane – bound cytoplasmic granules; agranulocytes (lymphocytes and monocytes) lack granules. Because the general function of the leukocytes is to combat inflammation and infection, the clinical diagnosis of an injury or infection is often assisted by a differential count, which is the determination of the percentage of each type of white cell (Jardins 2002).

1) **Granulocytes**

Neutrophils

Are the most numerous of the WBCs. They typically account for half or more of the WBC population. Neutrophils are active phagocytes that are twice the size of erythrocytes and contain small granules that produce potent antibotic – like proteins called defensins They have a diameter of 9-12um. They are especially found at inflammation sites caused by bacteria and some fungi, which they ingest and destroy. The number of neutrophils increases dramatically during bacterial infections. (Saladin and Porth, 1996; Jardins, 2002).

Easinophils

Have a diameter of 10-14um and have larger coarse granules that stain the acidic dye eosin. Eosinophils are phagocytosis of antigen-antibody complexes, allergens and inflammatory chemicals. They aggregate near parasites such as

worms and release enzymes that weaken or destroy them. Differential count increases in asthmatic patients, parasitic infections allergies etc (Saladin and Porth 1996; Jardins, 2002).

Basophils

Have a U or S shaped nucleus, but it is obscured from view by coarse cytoplasmic granules that stain dark purple with basic dyes. They have a diameter of 8 – 10um. The granules contain histamine as an inflammatory substance that causes vasodilation and attracts other WBCs to the inflamed site. They also secrete heparin which promotes mobility of other WBCs by preventing clotting (Saladin and Porth, 1996; Jardins, 2002).

(ii) Agranulocytes

Lymphocytes

They are sometimes classified by size as small (5-8um), medium (10-12 um) and large (14-17um) lymphocytes. They are the second most numerous leukocytes in the blood. Although large numbers of lymphocytes exist in the body, only a small amount are found in the blood stream. The medium and large lymphocytes are usually seen in the connective tissue and only occasionally in the circulating blood. Natural killer lymphocytes attack cells of the body that are infected with viruses or that have turned cancerous. B lymphocyte "present" antigens and activate other cells of immune system, differentiate into plasma cells that secrete antibodies, and serve as memory

cells in humoral community. T lymphocytes destroy foreign cells, coordinate action of other immune system cells, limit immune response and serve as memory cells in cellular immunity (Saladin and Porth, 1996; Jardins, 2002).

Monocytes

They are the largest of the formed elements about twice the size of an erythrocyte with a diameter of 12-15um. Their nuclei are quite variable in shape, round, oval, lobed, kidney-shaped or C-shaped. The cytoplasm is abundant and relatively clear. In the tissue, monocytes differentiate into highly mobile macrophage with large appetites. In chronic infections, such as tuberculosis, the macrophages increase in number and are actively phagocytic. Monocytes are also effective against viruses and contain intracellular parasites (Saladin and Porth, 1996; Jardins 2002).

Adult reference ranges of differential white blood cell count is as follows. (Reference Baker *et al* 2001)

White cell type	% age	Absolute (x 10^a/L
Neutrophils	40 – 75	2.0 – 7.5
Lymphocytes	20 – 45	1.5 – 4.0
Monocytes	2 – 10	0.2 – 0.8
Eosinophils	1 – 6	0.04 – 0.4
Basophils	<1	<0.1

- Absolute numbers can be calculated by multiplying the percentage of each white cell type by the total white cell type (Baker *et al* 2001).

C) Thrombocytes

Otherwise known as blood platelets are the smallest of the formed elements in the plasma. The normal platelet count ranges from 250,000 to 500,000/mm^3 of blood. The function of the platelets is to prevent blood loss from a traumatized area of the body involving the smallest blood vessels. They do this by virtue of an activator substance called platelet factor, which causes blood clotting at traumatized site. The platelets also contain serotonin when released causes smooth –muscle contraction and reduced blood flow (Jardins, 2002)

2.3.0 Wood smoke

Many organic compounds are produced by combustion of wood, some burn completely, some are changed chemically, and some leave the stove without burning. Some of these compounds deposit in the Chimney as creosote, some condense as very tiny particles of smoke, and some are released into the air as gases. Some of these organic compounds are poisonous, irritate the respiratory tract, and cause cancer or mutations (Washington State Department of Ecology, 1997).

The toxicity of wood smoke is well known. It contains many carcinogenic

substances such as aldehydes, dioxin, polycyclic aromatic hydrocarbons (PAH), carbon monoxide and ultra fine particulate matter i.e. particles that are less than 2.5 microns in diameter (PH 2.5). The problem with these ultra fine particles is that when they are less than 2.5 microns in diameter they are retained in the human lungs and have been shown to cause increases in morbidity and mortality. Also in developing countries, wood smoke has had a serious effect on human health as some wood stoves emit smoke to indoor air (Bowes 1998).

The chemical composition of wood smoke is listed below. (Environmental Protection Agency Report, 1993).

Chemical Composition of Wood Smoke

Species	g/kg wood
Carbon Monoxide	80-370
Methane	14-25
VOCs (c2-c7)	7-27
Aldehydes	
Formaldehyde	0.1-0.7
Acrolein	0.02-0.1
Propinaldehyde	0.1-03

Butrayldehyde	0.01-1.7
Acetaldehyde	0.03-0.6
Furfural	0.2-1.6 1.6
Substituted Furans	0.15-1.7
Benzene	0.6-4.0
Alkyl Benezenes	1-6
Toluene	0.15-1.0
Acetic Acid	0.15-1.0
Formic Acid	0.06-0.08
Nitrogen oxides (NO, No2)	0.2-0.9
Sulfur Dioxide	0.13-0.24
Methyl chloride	0.01-0.4
Napthalene	0.24-1.6
Substituted Napthalenes	0.3-2.1
Oxygenated Monoaromatics	1-7
Guaiacol (and derivatives)	0.4-1.6
Phenol (and derivatives)	0.2-0.8
Syringol (and derivatives)	0.7-2.7

Catechol (and derivatives)	02-0.8
Total Particle Mass	7-30
Particulate Organic Carbon	2-20
Oxygenated PAHs	*0.15-1*
Polycyclic Aromatic Hydrocarbons (PAH)	
Fluorene	4x10-5 - 1.7x10-2
Phenanthrene	2x10-5 – 3.4x10-2
Anthracene	5x10-5 – 2.1x10-5
Methylanthracenes	7x10-5 – 8x10-5
Fluoranthene	7x10-4 – 4.2x10-2
Pyrene	8x10-4 – 3.1x10-2
Benzo (a) anthracene	4x10-4 – 2x10-3
Chrysene	5x104 – 1x10-2
Benzofluoranthenes	6x10-4 – 5x10-3
Benzo (e) pyrene	2x10-4 – 4x10-3
Benzo (a) pyrene	3x10-4 – 5x10-3
Perylene	5x10-5 – 3x10-3
Ideno (1, 2,3-cd) pyrene	2x10-4 – 1.3x10-2

Benz (ghi) perylene	3x10-5 – 1.1x10-2
Coronene	8x10-4 – 3x10-3
Dibenzo (a,h) pyrene	3x104 – 1x10-3
Retene	7x10-3 – 3x10-2
Dibenz (a,h) anthracene	2x10-5 – 2x10-3

Trace Elements

Na	3x10-3 – 1.8x10-2
Mg	2x10-4 – 3x10-3
A1	1x10-4 2.4x10-2
Si	3x10-4 – 3.1x10-2
S	1x10-3 – 2.9x10-2
Cl	7x10-4 – 2.1x10-2
K	3x10-3 – 8.6x10-2
Ca	9x10-4 – 1.8x10-2
Ti	4x10-5 – 3x10-3
V	2x10-5 – 4x10-3
Cr	2x10-5 – 3x10-3
Mn	7x10-5 – 4x10-3

Fe	3x10-4 – 5x10-3
Ni	1x10-6 – 1x10-3
Cu	2x10-4 – 9x10-4
Zn	7x10-4 – 8x10-3
Br	7x10-5 – 9x10-4
Pb	1x10-4 – 3x10-3
Particulate Elemental Carbon	0.3 – 5
Normal alkanes (c24-c30)	1x10-3 – 6x10-3
Cyclic di-and triterpenoids	
Dehydroabietic acid	0.01 – 0.05
Isopimarci acid	0.02 – 0.10
Lupenone	2x10-3 – 8x10-3
Friedelin	4x10-6 – 2x10-5
Clorinated dioxine	1x10-5 – 4x10-5
Particulate Acidity	7x10-3 – 7x10-2

Reference: Environmental Protection Agency (1993)

2.3.1 Carbon Monoxide

Carbon monoxide is a gas formed from burning fuels with an insufficient

supply of oxygen. Vehicles are the greatest source by far in the UK but combustion of wood and coal can produce high concentration close to the source. It is difficult to achieve a low emission of carbon monoxide with most solid domestic fuels. The smoke stream from a burning log, for example can contain a carbon monoxide concentration in excess of 10,000 milligrams per cubic meter. It is easy to achieve a very low emission with most gaseous fuels as long as the oxygen supply to the flame is adequate, but if oxygen is inadequate for these rapid burning, pressurized systems then very high CO levels are produced which have resulted in many CO related deaths and injuries. The main, persistent but relatively low level sources of carbon monoxide are tobacco smoking, environmental tobacco smoke (ETS), vehicle exhausts and the burning of wood, coal and charcoal. The residency time in air is about one month but dispersal and dilution means that ambient airway from sources is harmless most of the time (Department of Environmental, Transport and the Regions, 1997).

After breathing in, carbon monoxide interferes with the blood Hb by readily combining with hemoglobin to form carboxyhaemoglobin (COHb). The percentage of COHb in the blood determines the harmful effect to the body by causing and exacerbating cardiovascular disease. A 2.5% COHb due to from CO in air is now acknowledged to be definitely harmful to health but it is probable that a 1% COHb due to CO may be as much as the human body can

harmlessly take in addition to its endogenous (internally generated) production. (Department of Environmental, Transport and the Regions, 1997).

Behera et al 2001 measured the carboxyhaemoglobin in blood to find the extent of air pollution in each subject. The values were not very high 4.1% for asthmatic women that use liquefied petroleum gas (LPG) and 3.5% for asthmatic women that use biomass, they were still raised. This relatively low values were due to the fact that blood samples were taken 4 –5 hours after cooking and COHb has a short half-life.

It has also been noted that cigarette smokers and wood burners have equally high levels of carbon monoxide (CO) in their blood. Constricting blood vessels thereby restricting the flow of oxygen to the muscles, thus the burners feel cold and burn more. Carbon monoxide vented from wood stoves can cause fatigue, chest pain, irregular heartbeats, dizziness, weakness, nausea and disorientation. (Rozenberg, 1994).

Ozturk *et al* (2002) in a prospective study investigated the genotoxic effect of acute overexposure to combustion products originating from coal and wood stoves in patients presenting with acute carbon monoxide intoxication. They suggested that acute exposure to combustion products of wood or coal is genotoxic to DNA. Potential causes of genotoxicity include known mutagenic compounds present in coal or wood smoke and ash, oxygen radicals formed during combustion as well as hypoxic and reperfusion injury mechanisms initiated by carbon monoxide

intoxication.

2.3.2 Oxides (Nitrogen and Sulfur)

Irritants in woodsmoke (such as phenols, aldehydes, quinones, nitrogen oxides and sulphur oxides) contribute to health problems in the respiratory tract. (Ammann,1986).

Nitrogen dioxide (NO_2), a by product of oxidation and combustion, is a primary outdoor and indoor air pollutant, because of its oxidative potential and limited solubility, NO_2 is an alveolar irritant, and accidental exposures to high concentrations can cause acute pneumonia and death. NO_2 interacts with the lung epithelial lining fluid and epithelial cell membranes, with local production of reactive oxygen and nitrogen species. In door NO_2 concentrations are often greater than those found outdoors, with peak levels exceeding 2.0 ppm in homes with unvented sources of combustion. (Frampton *et al* 2002).

Human clinical studies have generally found no effect of NO_2 exposure on pulmonary function at concentrations < 2.0 ppm. However, exposures to 1.5 – 2.0 ppm for 1 –3h increased nonspecific airway responsiveness, and recent studies suggest that exposures as low as 0.26ppm NO_2 for 30min at rest induce increased responsiveness to specific allergen challenge in patients with asthma. Exposures to 2.0 ppm for 6h with intermitted exercise caused a very mild airway inflammatory response in healthy subjects, with no changes in lung function or alveolar macrophage (AM) phenotype. These data suggest that there are dangerous effects

on airway epithelium at concentrations below those associated with pulmonary function changes or inflammation (Frampton *et al* 2002).

Damji and Ricters 1989 found alterations in circulating and splenic lymphocytes, subsets after exposure to NO_2 for 8h at levels as low as 4 ppm. These findings suggest that NO_2 exposure may alter both local and systemic host defenses. However, clinical studies have been inconclusive.

The health effects of NO_2 exposure may therefore result both from direct oxidant effects of the pollutant and from increasing airway susceptibility to other challenges, including respiratory virus infection. It was hypothesized that NO_2 causes a cascade of events, beginning with injury and inflammation of the distal airway epithelium, recruitment of T lymphocytes from blood to the airways, and increased susceptibility of the injured epithelial cells to viral infection (Frampton *et al* 2002).

The effects of wood burning stoves on indoor air quality was investigated in a rural community of southern Brazil, during the winter season of 1991. The concentrations of polycyclic aromatic hydrocarbons (PAHs), nitrogen dioxide (NO_2) and suspended particulate matter (SPM) were assessed in houses with wood stoves and the results compared with levels found in houses with gas stoves. Higher levels of PAHs, and much higher levels of SPM were found in the kitchens with wood stoves. In contrast NO_2 concentrations in the kitchen as well as in personal exposure, were found to be slightly higher in houses with gas stoves. All

these differences were minimally affected by smoking, outdoor air pollution or other emissions from indoor combustion products. These findings appear to support the hypothesis that domestic wood burning stoves are risk factors for some upper digestive and respiratory tract cancers in Brazil. (Hamada *et al* 1992).

Morgan *et al* 1998 examined the effects of outdoor air pollutants in Sydney, Australia on daily mortality. They found an increase in the daily mean nitrogen dioxide concentration from the 10^{th} to the 90^{th} centile was associated with an increase of 7.71% (0.34 to 6.40) in respiratory mortality, and nitrogen dioxide on respiratory mortality are independent of the effects of the other pollutants.

Chronic exposure to biomass fuel combustion produces significant bronchial hyperesponsiveness. Further, air pollutants can act as potential triggers resulting in transient airway narrowing, or can have direct irritant effect leading to airway inflammation. Thus, they may increase the risk of developing bronchial asthma or can cause exacerbations of preexisting disease. Sulfur dioxide has been shown to produce bronchoconstriction when the concentration exceeds 1 ppm. (Behera *et al* 2001).

2.3.3 Particulate Particles

Rozenberg (1994) measured tiny air borne particles smaller than 2.5 microns, using a nephelometer, from anything that could produce/make dust, smoke or soot. She found that emissions from a clean and efficient fuel like gas had no detectable

emission of PM 2.5, burning anything that was solid immediately gives very high readings. The particles that were measured were so small that 30 would fit on a red blood cell. When something solid disappears into the air it's still there in this deadly form. "Matter is neither created or destroyed it just changes form".

Particles smaller than 10 microns (PM 10), are associated with asthma, cancer, and lung disease. New research links ingestion of PM 10 to sudden elderly heart attack, hyertensive heart disease. Sudden Infant Death Syndrome (SIDS), and altered immune defense mechanisms. The last compromises resistance to bacterial infections and cell proliferation. Repeated exposures permanently change the structural integrity of the cells of nasal and respiratory passages inhibiting the flow of mucous. There was a consensus that once ingested, these particles cannot get back out. They are digested or encapsulated in the body. In fibrocystic lung disease the scarred lung can weigh three times that of the normal lung. (Rozenberg, 1994).

There is extensive epidemiological evidence that increased levels of the inhalable particulate fraction of air pollution (PM 10) are associated with increased morbidity and mortality. The mechanisms of these effects are unknown, and the exact types and sizes of particles responsible are a matter of intense dispute. Aerodynamic diameters revealed that 96% of the particles had diameters less than 2.5. This data indicates that human lung parenchyma effectively retains PM 2.5, suggesting that attempts to determine the particles responsible for chronic

particulate pollutant effects should concentrate on this size range. (Churg and Braver, 1997).

Epidemiologic studies have found increased mortality associated with particulate air pollution. To test the biologic plausibility of this association Godleski et al 1996 used normal, rats with monocrotyline – induced pulmonary inflammation (50mg/kg SC), and rats with SO_2 induced chronic bronchitis (250 ppm SO_2, 6 wks) were exposed to concentrated air particles (CAPS) or filtered air for 3 consecutive days, 6 hours/day. Death occurred during exposure, inflammation was found in groups with disease, but animals exposed to CAPS exhibited increases in inflammatory parameters and bronchoconstriction. Animals with chronic bronchitis had the most evidence of airway constriction. Bronchoconstriction was significantly increased in the disease groups, thus ambient particle inhalation can cause death in rats with pulmonary disease, and inflammatory airway constriction also appear to be important in this response.

This investigation examined whether there was a relationship between ambient air pollutant in Santa Clara country, California and Emergency Room (ER) visits for asthma during the winters of 1988 – 1989 through 1991 – 1992. Air monitoring data included daily coefficient of haze (COH) and every-other-day particulate matter with aerodynamic diameter equal to or less than 10 microns (PM 10, 24 hr average). Daily COH measurements were used to predict values for missing days of PM 10 to develop a complete PM 10 time series. In time-series

analysis using Poisson regression, consistent relationships were found between ER visits for asthma and PM 10. This demonstrates an association between ambient winter time PM 10 and exacerbations of asthma in an area where one of the principal sources of PM 10 is residential wood combustion (Lipsett *et al* 1997).

Due to the fact that the use of wood stoves has increased greatly in the past decade, causing concern in many communities about the health effects of wood smoke. Fine particulate matter has been associated with decreased pulmonary function in children and with increased chronic lung disease in Nepal, where exposure of very high amounts of wood smoke occurs in these residential areas (Pierson *et al* 1989).

A 61-yr-old woman was evaluated for dyspnea on exertion and interstitial lung disease. A unique association between inhaled particles from wood burning and interstitial pneumonitis was demonstrated. Bronchoalveolar lavage revealed numerous particulates and fibers, as well as cellular and immunoglobin abnormalities. The particulate source was traced to a malfunctioning wood-burning heater in the patient home. (Ramage *et al* 1988).

Autopsies have shown that particles less than 2.5 microns in diameter (PM25) are retained in human lungs. Larger particles are not retained (Bowes, 1998).

2.3.4 Effect of Wood smoke and Passive Smoking (Secondary) on lung function and COPD

Lal *et al* 1993 had exposed rats to repeated, intermittent exposure to smoke generated from combustion of 1g wood/15min, total period for 75 min daily under dynamic exposure conditions, over a period of 15, 30 and 45 days. First 15 days exposure caused mild bronchiolitis, hyperplasia and hypertrophy of bronchiolar epithelial lining cells, some necrosed lining cells desquamated into lumens, congestion of parachymatous blood vessels, edema, hyperplasia of lymphoid follicles, peribronchiolar and perivascular infiltration of polymorphonuclear cells, and mild emphysema. These lesions progressed further during 30 and 45 days of exposure, though emphysematous changes remain constant. By 30 days and 45 days, hyperplastic and hypertropic changes of bronchioles became quite marked, with mononuclear cells infiltration and alveolar septa thickening.

Animal toxicology studies showed that wood smoke exposure could disrupt cellular membranes, depress macrophage activity, destroy ciliated and secretory respiratory epithelial cells and cause aberrations in biochemical enzymes levels (Larson *et al* 1994).

Betchley *et al* 1997 evaluated effects on respiratory health of forest firefighters exposed to high concentrations of smoke during their work shift, 76 subjects were studied for cross-shift and 63 for cross-season. On average the cross-season data were collected 77.7 days after the last occupational smoke exposure. The cross-shift analysis identified significant mean individual declines in FVC, FEV_1 and FEF (25- 75%). The pre/shift to mid/shift decreases were

0.089L, 0.190L and 0.439L/ sec respectively, with pre/shift to post/shift declines of 0.065L, 0.150L and 0.496L/sec. Mean individual declines for FVC, FEV_1 and FEF (25 –75) of 0.033L, 0.104 L and 0.275 L/sec respectively, also were noted in the cross-season analysis. The FEV_1 changed significantly, the use of wood for indoor heat also was associated with the declines in FEV_1. Although annual lung function changes for a small subset (n = 10) indicated reversibility of effect, this study suggested a concern for potential adverse respiratory effects in forest firefighters.

Spontaneous inhalation of wood smoke via a tracheotomy immediately triggered either a slowing of respiration or an augmented inspiration in 83 anesthetized Sprague- Dawley rats. Results suggested that increase in hydroxyl radical burden following smoke inhalation is actively involved in evoking the acute irritant effects of wood smoke on breathing in rat (Kou *et al* 1997).

A case –control study was performed in women older than 40 years of age to evaluate the risk of cooking with traditional wood stoves for chronic bronchitis and chronic airway obstruction (CAO). 127 patients with chronic bronchitis or CAO of which, 63 had chronic bronchitis alone, 23 had CAO alone (FEV_1 less than 75% of predicted) and 41 had both chronic bronchitis and CAO were selected. Four control groups were selected: 83 patients with pulmonary tuberculosis, 100 patients with interstitial lung disease, 97 patients with ear, nose and throat ailments, and 95 healthy visitors to the hospital (controls). Exposure to

wood smoke, assessed as any or none, and as hours-years (years of exposure multiplied by average hours of exposure per day) was significantly higher in cases than in controls The risk of chronic bronchitis alone and chronic bronchitis with CAO increased linearly with hour – years of cooking with a wood stove. The findings supported a causal role of domestic wood smoke exposure in chronic bronchitis and chronic airflow obstruction (Perez – Padilla *et al* 1996).

Whittemore *et al* (1995) determined the prevalence of COPD (self-reports of doctor-diagnosed chronic bronchitis or emphysema) among 12,980 lifelong non-smokers aged 18 – 74 years. Overall, 3.7% of men and 5.1% of women reported physician- diagnosed COPD, and the prevalence increased with age and among the economically disadvantaged. These results provide evidence that COPD is not uncommon among non-smokers and that risk factors other than active cigarette smoking, such as environmental tobacco smoke (ETS), may contribute to the development of COPD in non-smoking adults.

Forest firefighters in the USA had decreased lung function after fighting fires and the use of woodstoves by these firefighters also affected their lung function. Eight out of twelve homes were also shown to have an increase in indoor mutagenicity (Bowes, 1998).

In a laboratory study, mice were subjected to either wood smoke, oil furnace fumes or clean air for 6 hours. They were then challenged with a streptococcus bacterium and within two weeks, 21% of the mice exposed to

wood smoke were dead compared with 5% of the mice exposed to the oil furnace fumes or clean air. (Bowes, 1998).

Park *et al* (2004) worked on the assessment of oxidative stress in lungs in sheep after inhalation of wood smoke. It evaluated antioxidant status and the extent of pulmonary injury in sheep after graded exposure to smoke. Adult, male sheep (n = 4-5 per group) were anesthetized and received 0, 5, 10 or 16 units of cooled western pine bark smoke, corresponding to 0, 175, 350 and 560, respectively of smoke dwell time in the airways and lung. Taken together these data show that few indices of oxidative stress responded in a dose dependent manner to graded dose of smoke inhalation, although most of the indices measured in the lungs were affected by the highest dose of smoke.

Saini *et al* 2003 in a case report wrote that "there are reports of patients developing interstitial lung disease following prolonged exposure to chronic domestic wood smoke inhalation. In both the reports the patients were mostly females, over 60 years of age with a long - standing and intense indoor wood smoke exposure. Dyspnea and cough were the main complaints and the chest radiograph showed a diffuse bilateral reticulonodular pattern. Histopathology showed fibrosis and inflammatory focal thickening of the alveolar septa as well as diffuse parenchymal anthracotic deposits. It was

suggested that the carbonaceous particles produced by the wood burning have a fibre-like character and iron coating, that enables them to incite chronic inflammation. Probably these patients represented one end of the spectrum of the wood smoke inhalation associated lung disease.

A medical evaluation of Mexican women who regularly cook over open wood fires revealed ravaged lungs and Pulmonary Arterial Hypertension, more severe than tobacco -related Chronic Obstructive Pulmonary Disease. (Sandoval *et al* 1993).

Controlled exposures of rabbits to white pine wood smoke, an animal model of smoke inhalation was created. Light and electron microscopic examinations of injured respiratory tissues from the animals revealed a reproducible, necrotizing tacheobronchial epithelial cell injury. Six hours after injury, the epithelium remains largely intact but was infiltrated by inflammatory cells; by 24 hours its ciliated and secretory lining cells were largely destroyed, the inflammatory reaction was maximal, but basal epithelial cells retained their normal structural appearances; by 72 hours, its surfaces were largely covered by a nonciliated, stratified reparative epithelium, apparently derived from proliferating and migrating basal cells. (Thorning *et al* 1982).

Lai and Kou 1998 investigated the stimulation of vagal pulmonary C fibers (PCs) by wood smoke. They recorded impulses from PCs in 58 anesthetized,

open-chest, and artificially ventilated rats, delivered 6 ml of wood smoke into the lungs. Within 1 or 2 seconds after the smoke delivery, an intense and non phasic burst of discharge was evoked in 60 of the 68 PCs studied and lasted for 4 - 8s. This immediate stimulation was usually followed by a delayed and more sustained increase in C- fiber activity. The overall stimulation was not influenced by removal of smoke particles or by pretreatment with dimethylthiourea (DMTU: a hydroxyl radical Scavenger) or indomethacrin (Indo; a cyclooxygenase inhibitor). The immediate – phase stimulation was not affected by pretreatment with Indo but was largely attenuated by pretreatment with DMTU or by a combined treatment with DMTU and Indo. Conversely, the delayed-phase stimulation was partially suppressed either by DMTU or by Indo but was totally abolished by DMTU and Indo. These results suggest that (1) stimulation of PCs is linked to the gas phase of wood smoke and (2) hydroxyl radical, not cycloxygenase products is involved in the immediate - phase stimulation, whereas both metabolites are responsible for evoking the delayed-phase stimulation.

Tesfaigzi *et al* 2002 worked with Brown Norway rats that were exposed to air for 3h/day, 5days/week for 4 or 12 weeks to air as control, or to 1 or 10 mg/ m^3 concentration of wood smoke particles from pinus edulis (a specie of wood). The wood smoke consisted of fine particles. Pulmonary function, specifically carbon monoxide - diffusing capacity and pulmonary resistance, was somewhat affected in the high-exposure group. Mild chronic inflammation and squamous metaplasia were observed in the larynx of the exposed groups. The severity of alveolar

macrophages hyperplasia and pigmentation increased with smoke concentration and length of exposure, and alveolar septae were slightly thickened. Together these observations suggest that exposure to wood smoke caused minor but significant changes in Brown Norway rats.

Work was done on the mediator mechanisms of delayed smoke - induced lung injury in 126 anesthetized and artificially ventilated guinea pigs who received challenges of either air or 40 tidal breaths of wood smoke. Two hours after inhalation, wood smoke produced various injurious responses, including increases in alveolar - capillary permeability, microvascular permeabilities and histological injury scores, in airway and parenmchymal tissues (Lin *et al* 2001).

Two smoke challenges (each 10 ml) separated by 30 min were delivered into the lungs of anesthetized guinea pigs by a respirator to investigate the time course of, and the contribution of other chemical mediators to smoke - induced airway hyper responsiveness (SIAHR). In the control animals, the SIAHR was evident by the broncho-constrictive response to the second smoke challenge (SM2) which was approximately 5.2 fold greater than that of the first challenge (SM1). This SIAHR was alleviated by shortening the elapsed time between SM1 and SM2 to 10 min or by expanding it to 60 min, and was abolished by extending it to 120min. (Hsu *et al* 2000).

Ho and Kou, 2002 investigated the airway responses evoked by wood smoke delivered through the nasal cavity in anesthetized Sprague - Dawley

rats. Wood smoke (5ml, 1.4ml/s) was delivered into an isolated nasal cavity while animals breathed spontaneously. In study 1, nasal wood smoke triggered either an apneic response (n= 26) or a sniff-like response (n= 16) within 1 sec after smoke exposure in 42 normal rats. Both airway responses were abolished by trigeminal nerve deneroction and by nasal application of a local anesthetic or a hydroxyl radical scavenger, but they were not significantly affected by removal of smoke particulates or nasal application of a saline vehicle. In study 2, nasal wood smoke only triggered a mild apneic response in two rats neonatally treated with capsaicin and had no effect on breathing in the other six. In contrast, wood smoke through the nasal cavity evoked an apneic response in six rats neo-natally treated with the vehicle of capsaicin and elicited a sniff -like response in the other two.

Lin and Kou, 2000 studied the mechanisms underlying wood smoke induced acute airway injury in 120 anesthetized guinea pigs. Five minutes after airway exposure, various doses of wood smoke produced a dose - dependent increase in Evans blue dye contents at all airway levels measured. Additionally inhaled wood smoke produced submucosal edema of the trachea, bronchus, and peribronchial edema.

The short term respiratory effects of heavy occupation wood smoke exposure among traditional charcoal production workers was investigated. A total

of 22 charcoal workers were studied and compared with a control group of 35 farmers residing in Perama, Rethymnan and Crete. The charcoal workers were exposed to wood smoke for an average of 14 hour/day during a mean of 23.7days required for the burning of kilns. The workers under study were found to have significantly more cough, sputum production, wheezing, dyspnea and hemoptysis than the control. The prevalence of respiratory symptoms such as cough, sputum production, wheezing and dyspnea in the charcoal workers were significantly elevated during the exposure period. The mean+/-SD percent of predicted values of FVC, FEV_1, FEV_1/FVC ratio and forced expiratory flow at 25 to 75% of FVC during the exposure period were significantly lower than those before exposure. The mean +/- SD value of peak expiratory flow at midday and in the evening during the exposure were significantly lower than before 524+/-131L/min vs 548+/-108L/min, P = 0.03; and 521+/-135 L/min vs 547+/ - 131 L/min, P = 0.02 respectively. Suggesting that wood smoke exposure in charcoal workers is associated with increased respiratory symptoms and decreased pulmonary function. (Tzanakis *et al* 2001).

Peters et al 1999 determined the lung function status of some Nigerian men and women chronically exposed to fish drying using burning firewood. There were 183 male and 192 females aged 20 to 45 years who had been exposed for a minimum of five years. The control group comprised sex matched male (142) and female (152) Nigerians from the same area who were not exposed to any known pollutant. Lung function indices were significantly lower in the men engaged in

firewood fish dying than in their control. Similarly lung function indices were lower in females in the fishing industry than in their controls; FVC 2.42 (0.17) Vs 3.02 (0.24); P<0.001]; FEV1 [1.70 (0.19) Vs 2.55 (0.21), P<0.001]; FEV1% [72.9 (3.2) Vs 84.4 (6.7), P<0.001]; and PEFR [298 (22) Vs 418 (34), P< 0.001]. All the lung function indices (except FEV_1%) of the fishermen and women decline significantly (P<0.001) with their duration of exposure. The results showed a predominantly mixed pattern of respiratory defect. There were higher prevalences of respiratory and other symptoms among the cases than the controls.

Dennis *et al* 1996 investigated if exposure to firewood smoke and other indoor pollutants is a potential risk factor for Obstructive Airways Disease (OAD) among women in Bogota in whom cigarette smoking and other known risk factors may not be the most frequent. A hospital-based case-control study to identify risk factors for OAD among women in Bogota was conducted. 104 OAD cases with 104 controls matched by hospital frequency matched by age was compared. Univariable analysis showed that tobacco use, wood use for cooking passive smoking and gasoline use for cooking were associated with OAD. Trends for years of tobacco use and years of wood cooking were present (P<0.05). (After multivariate analysis, variables remained significant except gasoline use). This study showed that among elderly women of low socio-economic status in Bogota, wood smoke exposure was associated with development of OAD and may help explain around 50% of all OAD cases.

Pulmonary function studies were carried out in 3318 healthy, nonsmoking,

asymptomatic housewives to evaluate the role of different cooking fuels in domestic use. The women used four different types of cooking fuels; biomass fuel, Liquified Petroleum Gas (LPG), kerosine used in stoves, and a combination of two or more of these (mixed). Three parameters of ventilatory function (FVC, FEV_1, PEFR) were evaluated. A positive correlation was observed between all these parameters except PEFR with that of height, but a negative correlation was observed between the age, duration of cooking and exposure index. Mixed fuels and biomass fuels affected FVC values more adversely. Similar trend was observed for FEV_1 also. Users of biomass fuel had the lowest mean value for PEFR. In users of mixed fuels, there was a decline in FVC, FEV_1 and PEFR, as the exposure increased. Thus mixed fuel has more deleterious effects on pulmonary function than other fuels. (Behera, 1997).

Lung function parameters, Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV_1), Peak Expiratory Flow Rate (PEFR) were measured in 3318 non smoking Indian women using four different types of cooking fuels (biomass, liquified petroleum gas, kerosine and mixed). Biomass fuel users had FVC values less than 75% predicted (73.42+/-0.90; mean+/-SE) whereas in other groups it was more than 75% of predicted, though less than 80% of the predicted values. However, FEV_1, FEV_1/FEV (%) and PEFR were within normal limits in all the four groups. The absolute values of all the three parameters of lung functions were the lowest in the biomass and mixed fuel users. A negative correlation was observed of cooking and exposure index. Thus this study showed

that, lung function, particularly FVC, is affected by indoor air pollution due to domestic cooking more so with biomass fuel. (Behera *et al* 1994).

Dutt *et al* 1996 studied the effects of exposure to indoor air pollution from the use of cooking fuels on lung functions and respiratory symptoms in women aged 15 - 60 years. The participants were 105 women using biofuel, 105 using kerosine and 105 using liquid petroleum gas (LPG), selected from among 1117 women aged 15 - 60 years, by a stratified random sampling technique. Lung functions were assessed by measuring forced vital capacity (FVC), forced expiratory volume in the first second (FEV_1) and peak expiratory flow rate (PEFR). Women using biofuels experienced more respiratory symptoms (23%) than those using kerosine (13%; $P>0.05$) or LNG (8%; $P<0.05$). Lung functions - FVC, FEV_1, FEV_1% and PEFR - were significantly lower in biofuel users compared with both kerosine ($P<0.01$) and LPG users ($P<0.001$). Lung functions in kerosine users also were significantly poorer when compared with LPG users ($P<0.01$). Predicted pulmonary functions using multiple regression equations, derived from the data of the present study, indicated that women using biofuels were more liable to have reduced pulmonary functions than women using kerosine or LPG.

2.3.5 Wood smoke and Carcinogenic Substances

Wood smoke is estimated to be 12 times more carcinogenic than an equal concentration of cigarette smoke (Bowes, 1998).

Environmental Protection Agency (EPA) estimates that the lifetime cancer risk from wood stove smoke is twelve times greater than that from an equal volume of second hand tabacco smoke. Burning two cords of wood produces the same amount of mutagenic particles as driving 13 gasoline powdered cars 10,000 miles each at 20 miles/gallon or driving 2 diesel powdered cars 10,000 miles each at 30 miles/gallon (Lewtas, 1991).

Current ambient measurements, surveys and model predictors indicate winter respirable (<2mm) emissions from residential wood combustions can easily exceed all other sources. Both the chemical potency and deliver-ability of the emissions from the source are of concern. The emissions are almost entirely in the inhalable size range, and contain toxic and priority pollutants, carcinogen, co-carcinogens cilia toxic, mucus coagulating agents, and other respiratory irritants such as phenols, aldehydes etc (Cooper, 1980).

Smoke samples, in both gas and particulate matter (PM) phases, of the three domestic stoves were collected using U.S. EPA modified method 5 and were analyzed for 17 PAH, acute toxicity and mutagenicity. The gas phase of smoke contributed ≥ 96% of toxicity, and ≥60% of mutagenicity. The highest emission factor of 17PAH was from sawdust but the highest emission of 11 genotoxic PAH was from kerosine. The total toxicity emission factor was the highest from sawdust, followed by kerosene and wood fuel. The higher mutagenicity emission factor was from wood fuel and the lower from sawdust (Kim *et al* 2002).

2.3.6 Blood

Since the total white blood cell count (WBC) has been related to both death from coronary heart disease and the levels of lung function, the relationship between these parameters was examined in subjects from the Busselton Health surveys. Questionnaires regarding respiratory and cardiac illness, and smoking habits were administered and total WBC, Forced Expiratory Volume in one second (FEV_1) and Forced Vital Capacity were measured in 2,105 males and 2,186 females. Multiple linear regression showed that smoking, increasing age, reduced FEV_1 (% predicted) and a history of bronchitis were associated with increased WBC. Reduction of FEV_1 (% predicted) by 20%, a history of dyspnoea and an increase in WBC of 1,300 cells x ML (-1) were predictive of increased mortality from all causes of coronary heart disease by approximately 20,100, and 10% respectively, independent of smoking. Removing WBC from the regression model did not significantly change the relationship between FEV_1 and mortality. The study showed that white blood cell count, forced expiratory volume in one second and dyspnoea are independently related to mortality in both males and females and that the effect of forced expiratory volume in one second on mortality is not explained by the white blood cell count (James *et al* 1999).

Lal *et al* 1993 examined the histomorphological changes in lungs of rats following exposure to wood smoke he found that hematological studies

showed marginal alterations in hemoglobin levels, ESR and PCV during 15 days, whereas significant changes in eosinophils were observed during 30 and 45 days, and ESR during 45 days only.

Tesfaigzi *et al* (1998) investigated the effects of subchronic exposure to wood smoke on the respiratory tract of rats, the effects of wood smoke on the immune system were also investigated by analyzing the proliferation of T cells from the lung – associated lymph nodes and from concanavalin A (Con A). T cells from spleens from both the 1 mg/m^3 and $10mg/m^3$ rats had reduced incorporation of 3H – thymoline in response to activation with 0.3 and 1 mg/well of Con A. Similarly T cells from lung associated lymph nodes had reduced proliferation in response to Con A. These results indicate that not only the T cells associated with the lung, but also circulating T cells were affected by the wood smoke exposure. No significant differences were observed in b-glucuronidase or the numbers of macrophages, neutrophils lymphocytes or eosinophils. This may at least in part explain why respiratory tract infections are more common in populations using wood for cooking and heating.

Neufeld *et al* 2004 did a study to determine whether, among women who burn biomass fuels for cooking indoors, the use of "smokey" fires is associated with elevated hemoglobin concentration in comparison to women using "smokeless" stoves, i.e stoves that are designed to reduce indoor air pollution. Multiple linear regression analyses were used to investigate the relationship

between exposure (smokeless stove or smoky fire) and hemoglobin concentration. No effect of exposure on hemoglobin concentration was found in univariate or multivariate analysis.

CHAPTER 3

MATERIALS AND METHODS

This study was carried out on sixty healthy, non-smoking adult women with normal blood pressure and with no lung problems like bronchitis emphysema, asthma etc or metabolic diseases like diabetes. They had no disabilities, not infected with any kind of infection that will have serious effects on their WBC and also they were not on any antibiotics. Their ages ranged between 30 – 45 years.

The subjects were divided into two groups based on the kind of fuel used for cooking as well as the extent of exposure to wood smoke. The women that were studied (study group) were 30 subjects who worked for a living in restaurants (cooks) and used wood as a constant source of cooking fuel, for no less than 10 years. The control group were also 30 subjects in number, who used other kinds of fuel to cook like kerosine, gas or electric within the same age group. They also used wood to cook but much less frequently.

Clearance to carry out this research was obtained from the Scientific and Ethical Committee on Human Research, Ahmadu Bello University Teaching Hospital, Zaria.

3.1 Materials

1. Vitalograph (S-Model Spirometer) Serial No. 48917, Vitalograph Limited Buckingham, England.

2. Peak flow monitor
3. Vitalograph charts
4. Disposable mouth pieces
5. Airtight plastic container with Heparin
6. Needles and syringes

3.2 Methods

Peak Expiratory Flow Rate

The peak expiratory flow rate of these subjects were determined using the Peak Flow Monitor. The peak expiratory flow rate is the maximum velocity of airflow that can be produced during forced expiration.

For each subject, at least three readings were taken and recorded as PEFR. The test was done by having the subjects perform a maximal inspiration followed by a short, maximally forceful expiration through the PFFR monitoring device. The subjects were told not to cough and the expiratory efforts lasted only for one to two seconds (Madama, 1998).

Vitalograph

The Vitalograph was used to measure the Forced Expiratory Volume in one second (FEV_1) and Forced Vital Capacity (FVC). The subjects were instructed accordingly.

The recording switch at the right hand side of the Vitalograph was depressed

and the white lamp lit up. The subject was encouraged strongly to perform a maximal inspiration and continued the inspiratory effort at the full Total Lung Capacity (TLC) level for one to two seconds. A maximally forceful and complete expiration to the residual volume (RV) level was then performed. The forced expiration was continued for a minimum of six seconds. At least three trials/ maneuvers were performed by the subjects (Vitalograph limited, 1977).

Hematological Indices

Hematological indices was carried out by the methods explained by Darcie and Lewis 1991.

Collection of Blood

A tourniquet was tied around the upper left arm of the subjects. The antecubital vein was located on the arm and the skin was cleaned with 70% alcohol (methylated spirit), allowed to dry before being pieced by means of a disposable syringe. The piston of the syringe was withdrawn slowly, no attempt was made to withdraw blood faster than the vein was filling. The arm was then elevated after withdrawal of the needle and pressure applied to the site for several minutes. The blood was carefully delivered from the syringe to the plastic container containing heparin, it was then promptly and thoroughly but gently mixed with the anticoagulant.

Packed Cell Volume (PCV) (Haemtocrit Value)

The Micro Hematorit method was used as explained by Darcie and Lewis, 1991.

Capillary tubes 75mm in length and having an internal diameter of about 1mm, coated with heparin internally was used.

The blood that was already collected from the subjects was allowed to enter the tube by capillarity leaving at least 15mm unfilled. The tube was then sealed by heating the dry end of the tube rapidly in a fine flame. It was then centrifuged 1000 cycles for 5 minutes. The PCV was measured using a haematocrit reader.

Differential white Blood cell count

Fixing and staining of the film to distinguish the cells clearly was done using Leishman's stain. Sufficient Leishman's stain was poured to cover the dry film and allowed to stand for about 2 minutes. The methyl alcohol in the stain fixes the cells, after two minutes, the stain on the slide is mixed with buffered distilled water and left to stand for about 5 – 10 minutes. It was then rocked gently to aid mixing. It was then washed in a stream of buffered water until the film had a pinkish tinge (2 minutes). The back of the slide was cleaned and then set upright to dry.

A detailed examination of the morphology was examined under the microscope using the microscope and lens of x 100 with oil Immersion on movable stage.

The film was examined systematically as each leukocyte enters the field of view it was identified according to its kind and counted. A total of about 200 leucocytes were counted and the different kinds of leucocytes were calculated.

(Neutrophils, lymphocyte, Eosinophils, monocytes and Basophilic were identified and counted).

Identification of differential white blood cells according to Baker & Silverton, 2001.

Neutrophils

When stained they show a lobed nucleus, which stains a purple-violet colour. The older the cells the more lobes they have. Most of the cells have 2 or 3 lobes but it is possible to see as many as 7 lobes. The cytoplasm stains a light pink colour and contains small violet or pinkish staining, dust-like granules.

Eosinophils

They usually, have only 2 lobes to their nucleus, often in a 'spectacle' arrangement. The nucleus stains a little paler than the neutrophil and the cytoplasm contains many large, round or oval, deep orange-pink granules.

Basophils

Their nucleus is usually kidney-shaped and the cytoplasm contains a mass of large, deep purple staining granules which frequently obscure the nucleus.

Lymphocytes

Small lymphocytes nucleus stains a deep purple which occupies most of the cell so that the cytoplasm, which stains a pale blue colour can be seen only as a rim around the nucleus.

Large lymphocytes the nucleus stains a little paler than the small lymphocytes. The cytoplasm is more plentiful, staining a pale blue colour. The lymphocytes sometimes show a few reddish granules in the cytoplasm.

Monocytes

Monocytes have one large nucleus, which is usually centrally placed within the cell and often kidney shaped. This nucleus has a stranded appearance, like a skein of wool, and when stained, is a paler violet colour. The copious cytoplasm, staining a pale greyish –blue, contains in numerable dust like granules of reddish-blue.

Hemoglobin Concentration

The AO Hb –Meter was used. It is a specialized type of colorimeter for the convenient evaluation of the hemoglobin content of the blood. This instrument compares the absorption of light by the haemoglobin in a layer of haemolyzed blood of carefully defined depth, with the absorption of a standardized glass wedge.

A drop of blood was placed on the H-shaped moat, the blood is then haemolysed using a haemolysis applicator. A cover plate is placed on top of the H-shaped moat and clip is placed to hold them tightly together by touching only the edges.

The complete chamber was then inserted into the slot on the left side of the instrument. The instrument was held to the eye with the left hand in such a manner that the left thumb rests on the light switch button on the bottom of the instrument when the light is switched on a green split field appears in the instrument. The right hand is then free to move the slide button on the right side of the instrument until the two halves of the field are equally lit and appear as a single field. At this position the index mark on the slider knob indicates the haemoglobin concentration. Hb-Concentration was measured in g./dl.

Sahli (Acid haematin) Method

Hemoglobin is converted to acid haematin by the action of hydrochloric acid.

Apparatus and Reagent

(1) Sahli graduated tube and standard.

(2) N/10 Hcl

(3) 0.02 ml Pipette.

Technique

Sahli's graduated tube is filled to the 20 mark with N/10 Hcl. 0.02ml of blood is added then mixed well and allowed to stand for 5 – 10 N/10Hcl is added drop by drop, mixing between each addition, until the colour matches the standard. The amount of solution in the graduate tube is read. The calibrations give the haemoglobin concentration as a percentage.

The hemoglobin content, in g per 100ml of blood is calculated as follows.

The standard tube will state the number of grams per 100ml of hemoglobin that is equivalent to 100 percent. Some of the older Sahli standards use 17.2g per 100 ml as 100 percent haemoglobin. Using this apparatus, if a haemoglobin value is 90 percent the haemoglobin content in g per 100 ml is

90/100 x 17.2 = 15.48g per 100ml. As reported by Baker and Silverton, 2001.

3.3 Statistical Analysis

The sampling techniques used was simple random sampling.

All results are shown as mean $\pm$ S.E.M. Differences between measurements were analyzed statistically by means of Z test between the 2 groups. Percentage changes were also calculated between the various groups. Pearson correlation between parameters of each group using sigmastat 2.0 for windows (Jandel, Scientific, 1995). Differences between the two groups were considered significant when $P < 0.05$ (Singha, 2002).

CHAPTER 4

RESULT

4.0 Results

Sixty (60) female subjects were studied, they were all non-smoking women and were grouped into two groups. Group 1 were women that do not use wood to cook, so are therefore less exposed to wood smoke (control group), while group 2 were women that use wood to cook and are therefore highly exposed to wood smoke (experimental group).

Their age, height, weight, respiratory function test, CVS parameters, haematological indices were measured and the results are presented below.

4.1 **Arthropometric Parameters**

The mean ± SEM are of age, height, weight and BMI of the two groups are shown in table 1 and figures 1, 2, 3, 4. The mean ages, height and BMI of group 1 and 2 were 38.40 ± 0.83 and 36.90± 0.69yrs, 1.62± 0.01 and 1.62± 0.07 meters and 29.49± 1.00 and 26.78± 0.99 kg/m^2 respectively. There were no significant statistical difference between the two groups. The mean weight of group 1 and 2 were 77.27± 2.44 and 66.55± 3.10Kg respectively. There was significant statistical difference between the two groups (P<0.05).

Table 1: The mean and SEM of age, weight, height and BMI of women that do not use wood to cook (group 1), and women that use wood to cook (group 2).

Parameters	Control Group Group 1 (n =30)	Study Group Group 2 (n =30)	Percentage changes (%)	Z -test	P-value
Age (yrs)	38.40± 0.83	36.90 ± 0.69	3.91	1.40	P>0.05
Weight (kg)	77.27 ± 2.44	66.55 ± 3.10	13.87	2.72	P<0.05*
Height (m)	1.62 ± 0.01	1.62 ± 0.01	1.25	0	P>0.05
Body Mass Index (kg/m^2)	29.49 ± 1.00	26.78 ± 0.99	9.19	1.92	P>0.05

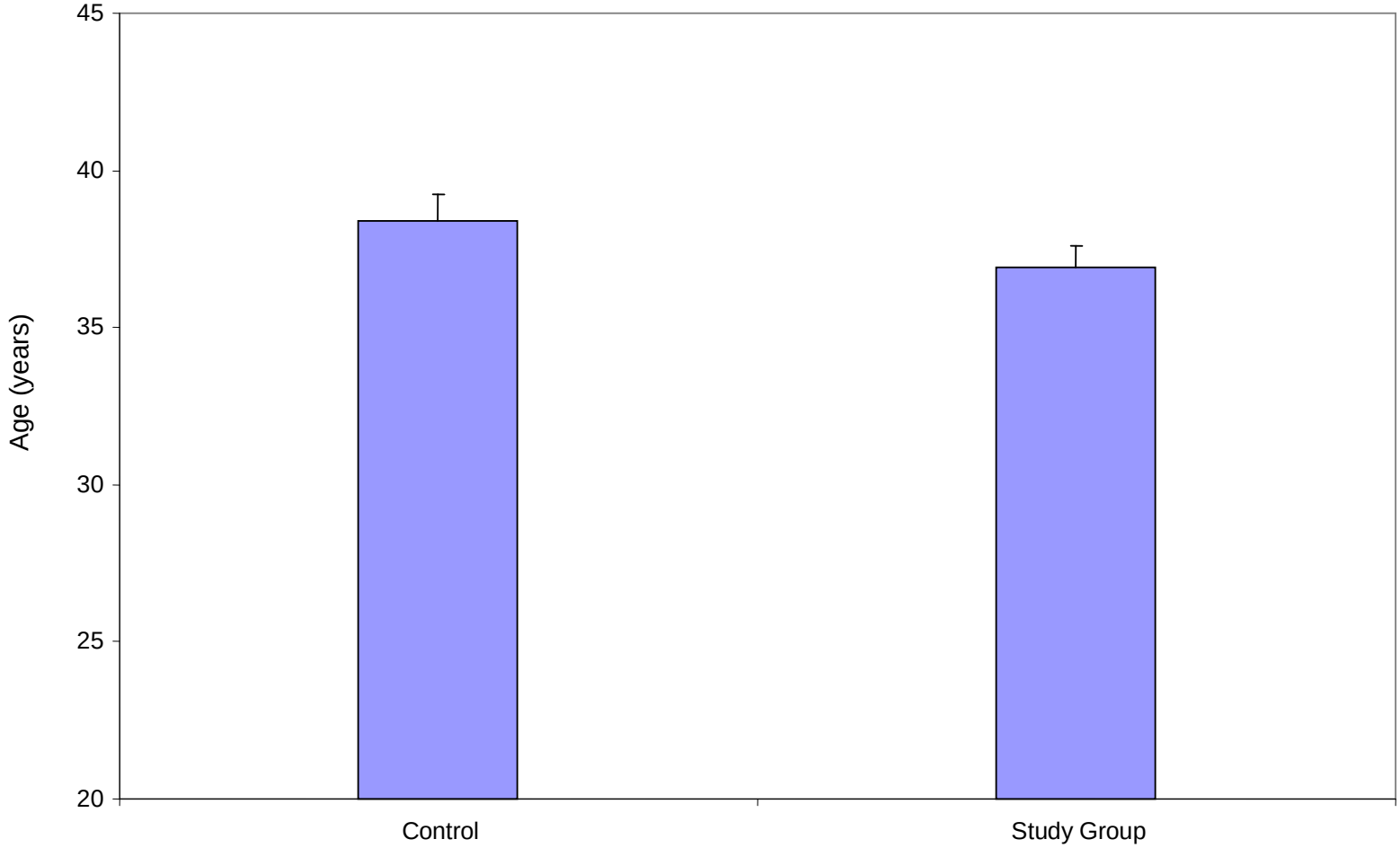


Fig. 1: Barchart showing mean ± SEM of age of control and study group (Control n=30, study group n=30) P>0.05

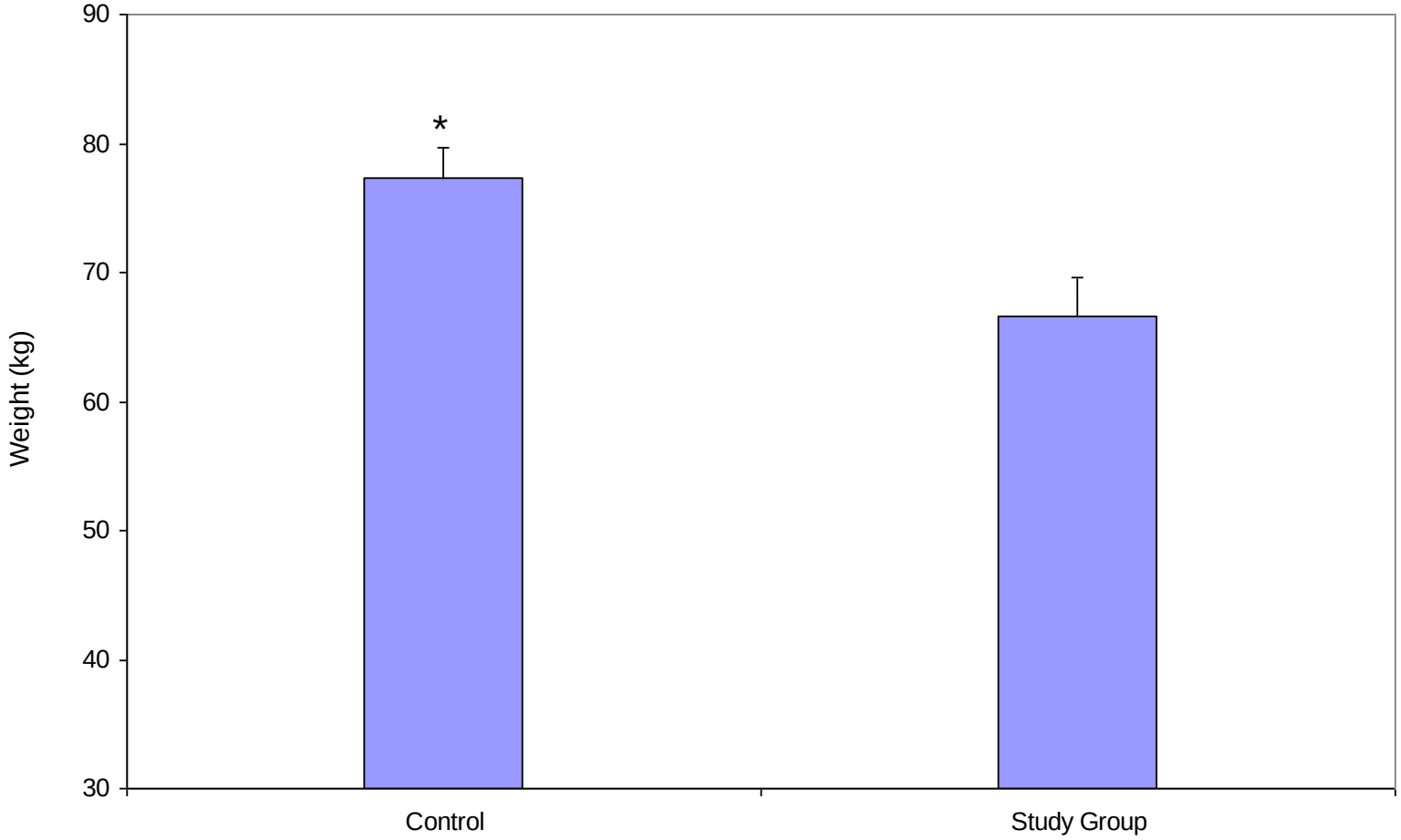


Fig. 2: Barchart showing mean ± SEM of weight of control and study group * P < 0.05 (Control n=30, study group n=30)

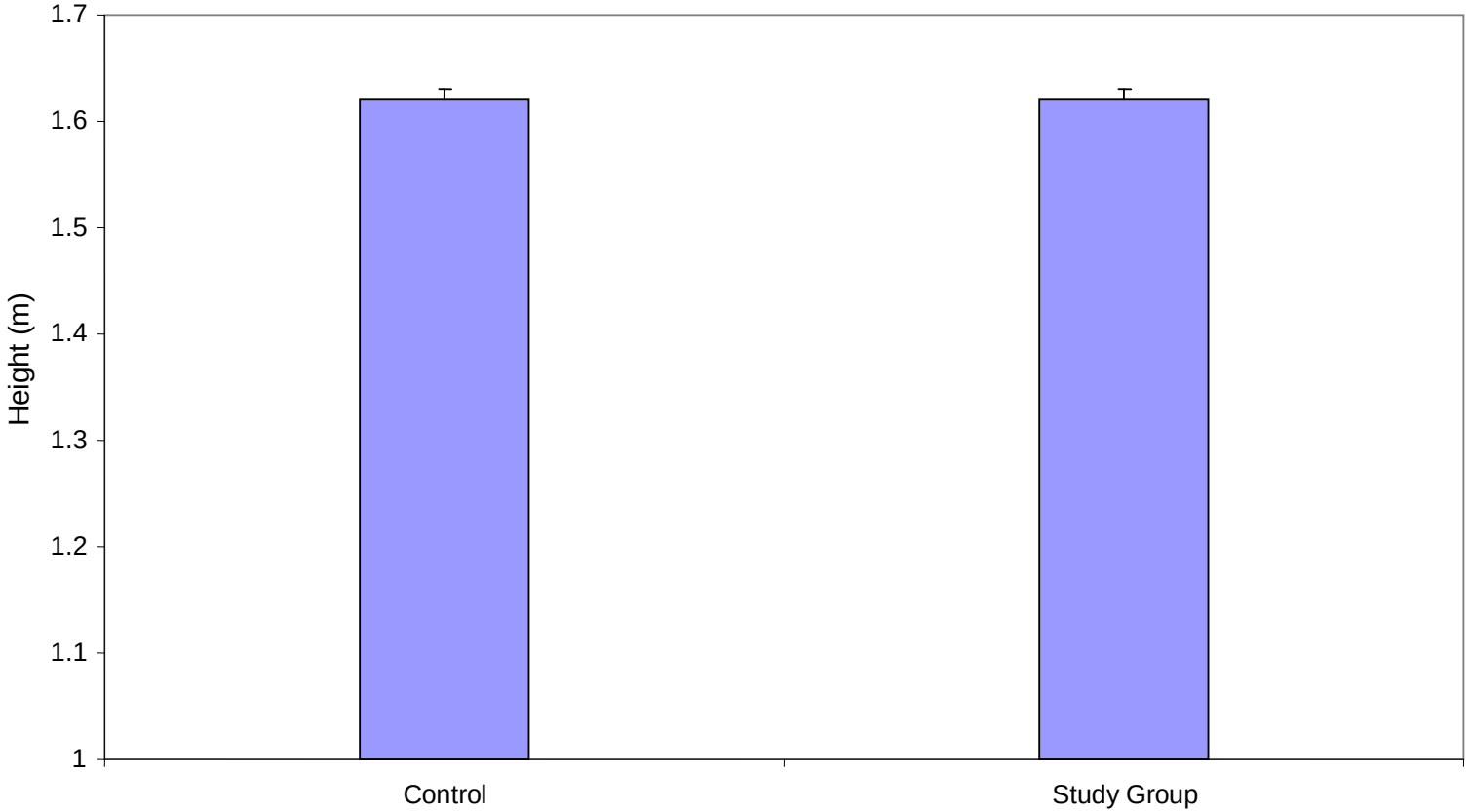


Fig. 3: Barchart showing Mean ± SEM of height of control and study group (Control n=30, study group n=30) p>0.05

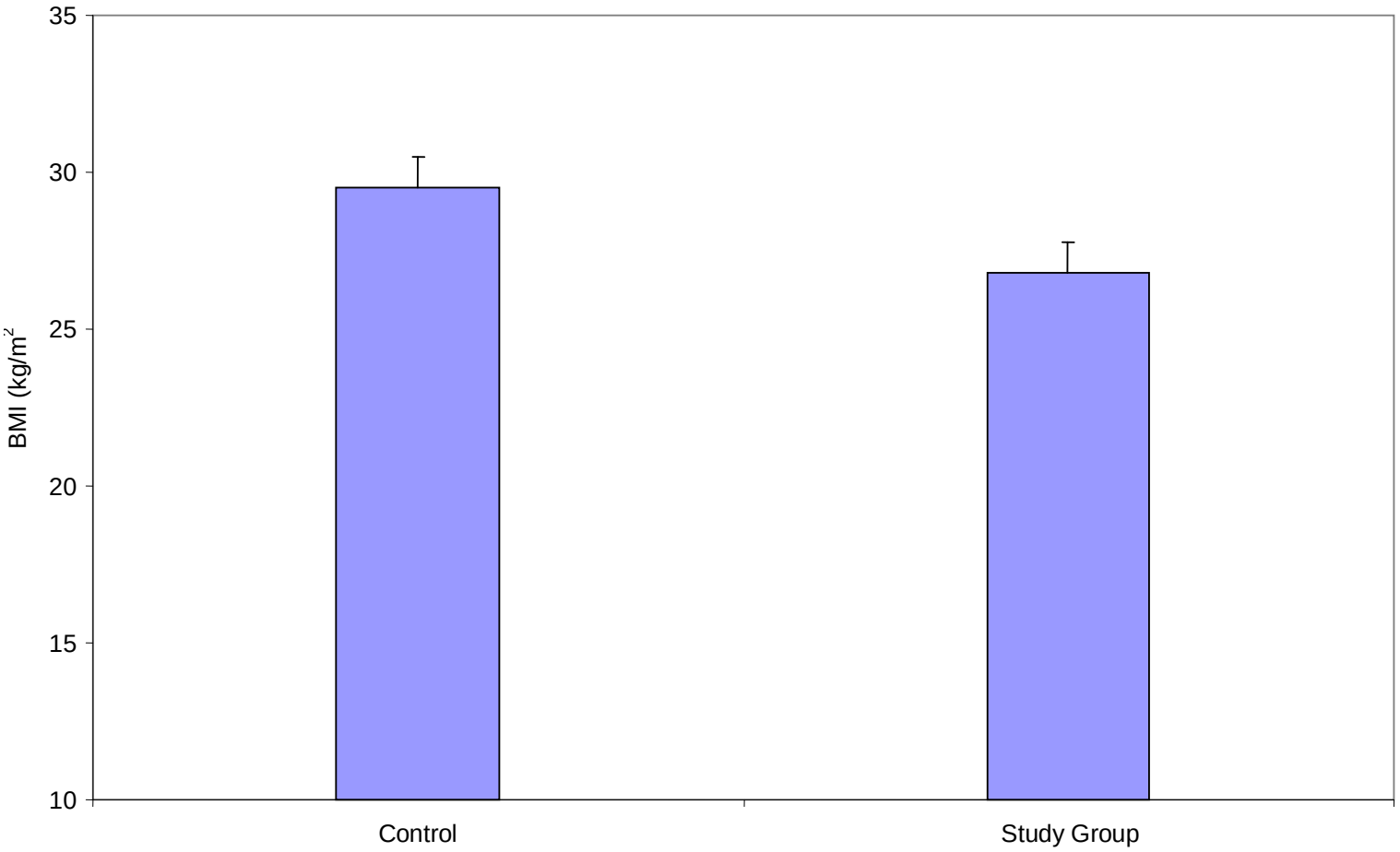


Fig. 4: Barchart showing Mean ± SEM of BMI of control and study group (Control n=30, study group n=30) p>0.05

4.2 Cardiovascular (CVS) Parameters

The mean ± SEM of systolic blood pressure, diastolic blood pressure, mean arterial blood pressure and pulse rate of the two groups are shown in table 2 and figure 5, 6 and 11. The mean systolic B.P, mean arterial B.P and pulse rate of groups 1 and 2 were 113.67± 2.65 and 111.17± 3.44 mmHg, 76.00 ± 2.15 and 75.00 ± 2.46 mmHg, 89.78 ± 1.86 and 87.06 ± 2.69 mmHg and 76.60 ± 1.29 and 77.93 ± 1.56 beats/ min respectively. There were no significant statistical difference found between the two groups (of the respective paramenters) ($P > 0.05$).

Table 2: The mean and SEM of pulse rate, systolic, diastolic and Mean arterial blood pressure of women that do not use wood to cook (group 1) and women that use wood to cook (group 2).

Parameters	Control Group Group 1 (n =30)	Study Group Group 2 (n =30)	Percentage changes	Z -test	P-value
Pulse rate (beats/min	75.60± 1.29	77.93 ± 1.56	3.08	-1.50	P>0.05
Systolic B.P (mmHg)	113.67 ± 2.65	111.17 ± 3.44	2.20	0.46	P>0.05
Diastolic B.P (mmHg)	76.00 ± 2.15	75.00 ± 2.46	1.32	0.31	P>0.05
Mean Arterial B.P.	89.78 ± 1.86	87.06 ± 2.69	3.03	0.83	P>0.05

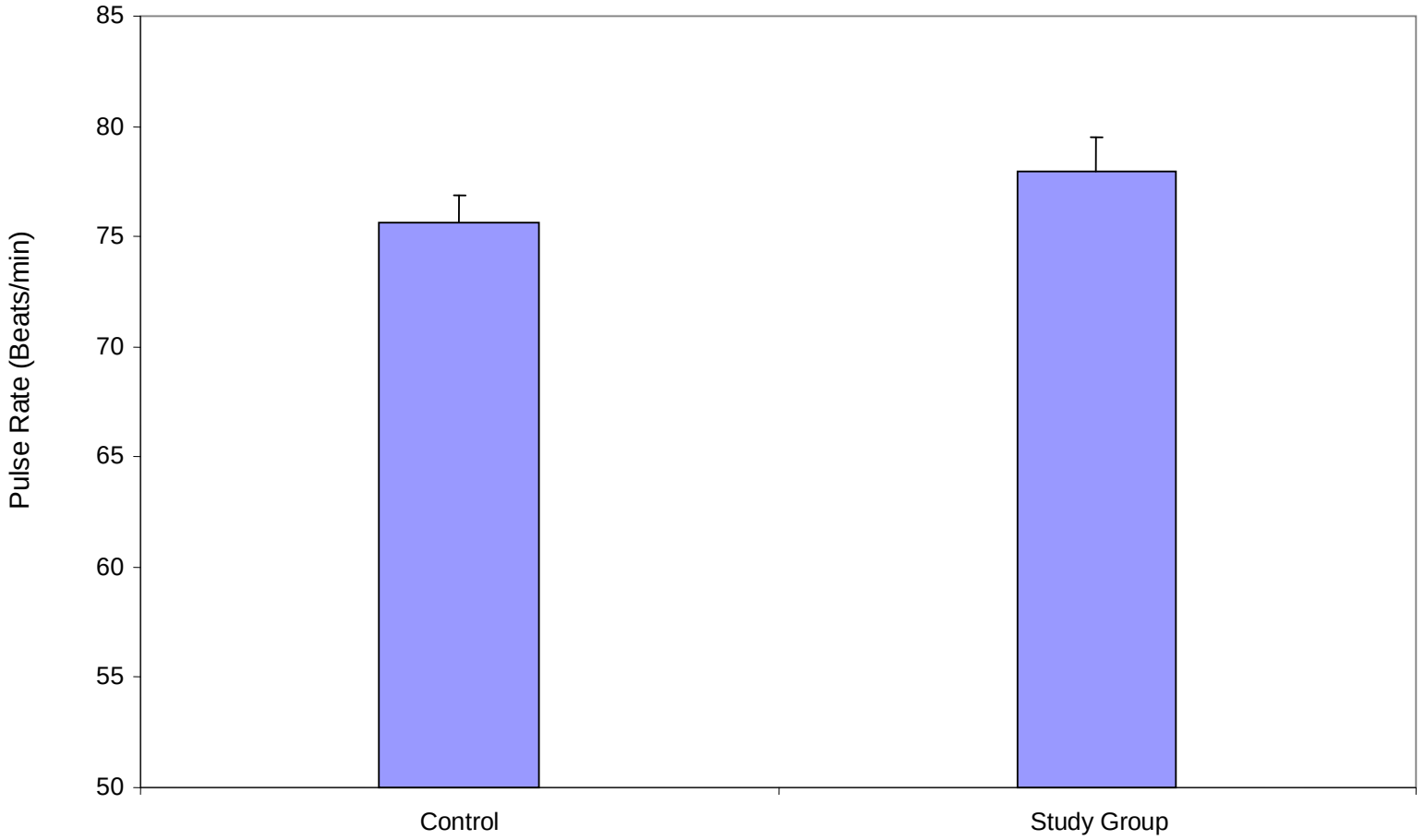


Fig. 5: Barchart showing mean ± SEM of BMI of control and study group (Control n=30, study group n=30) p>0.05

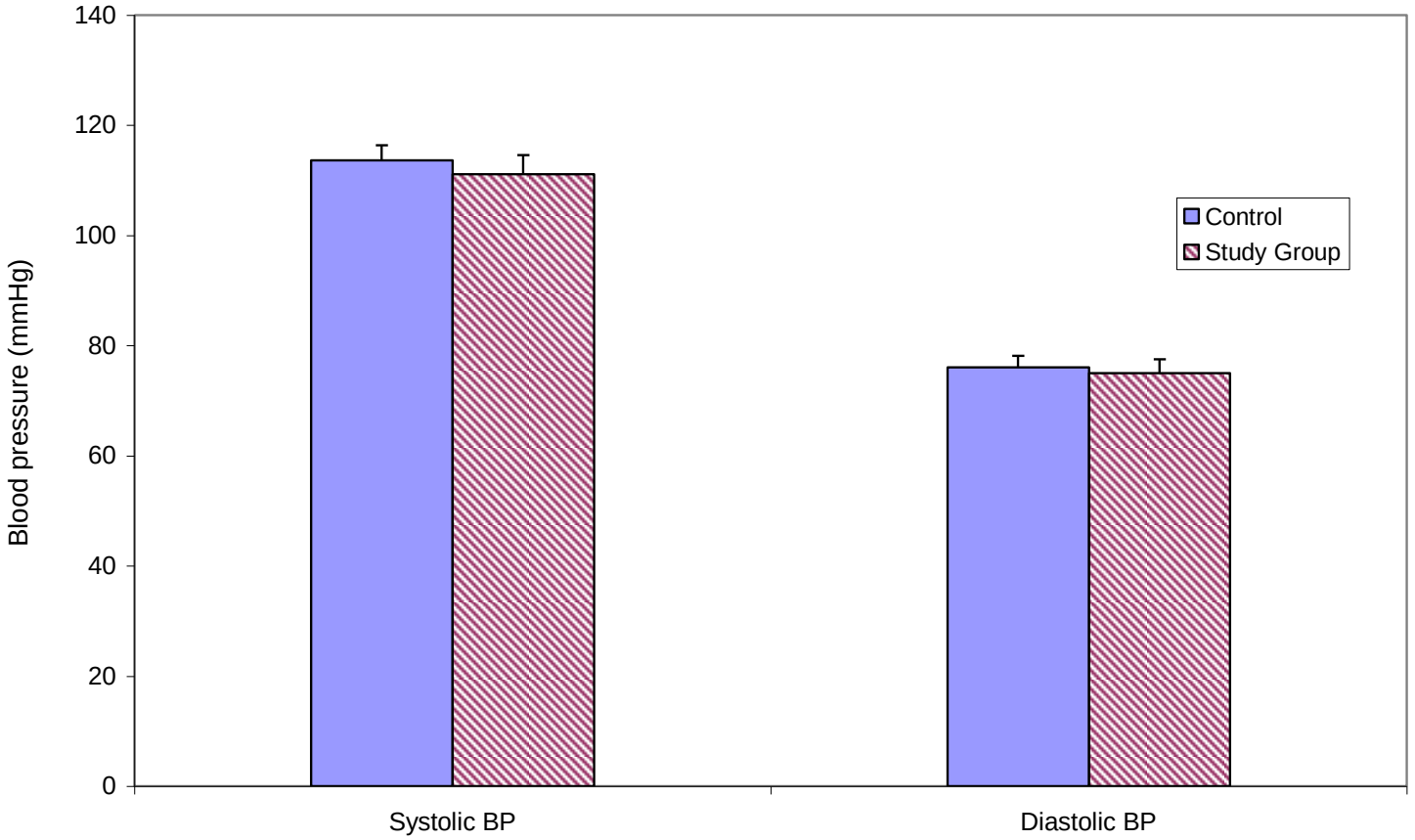


Fig. 6: Barchart showing mean ± SEM of blood pressure of control and study group (control n=30, study group n=30) P>0.05

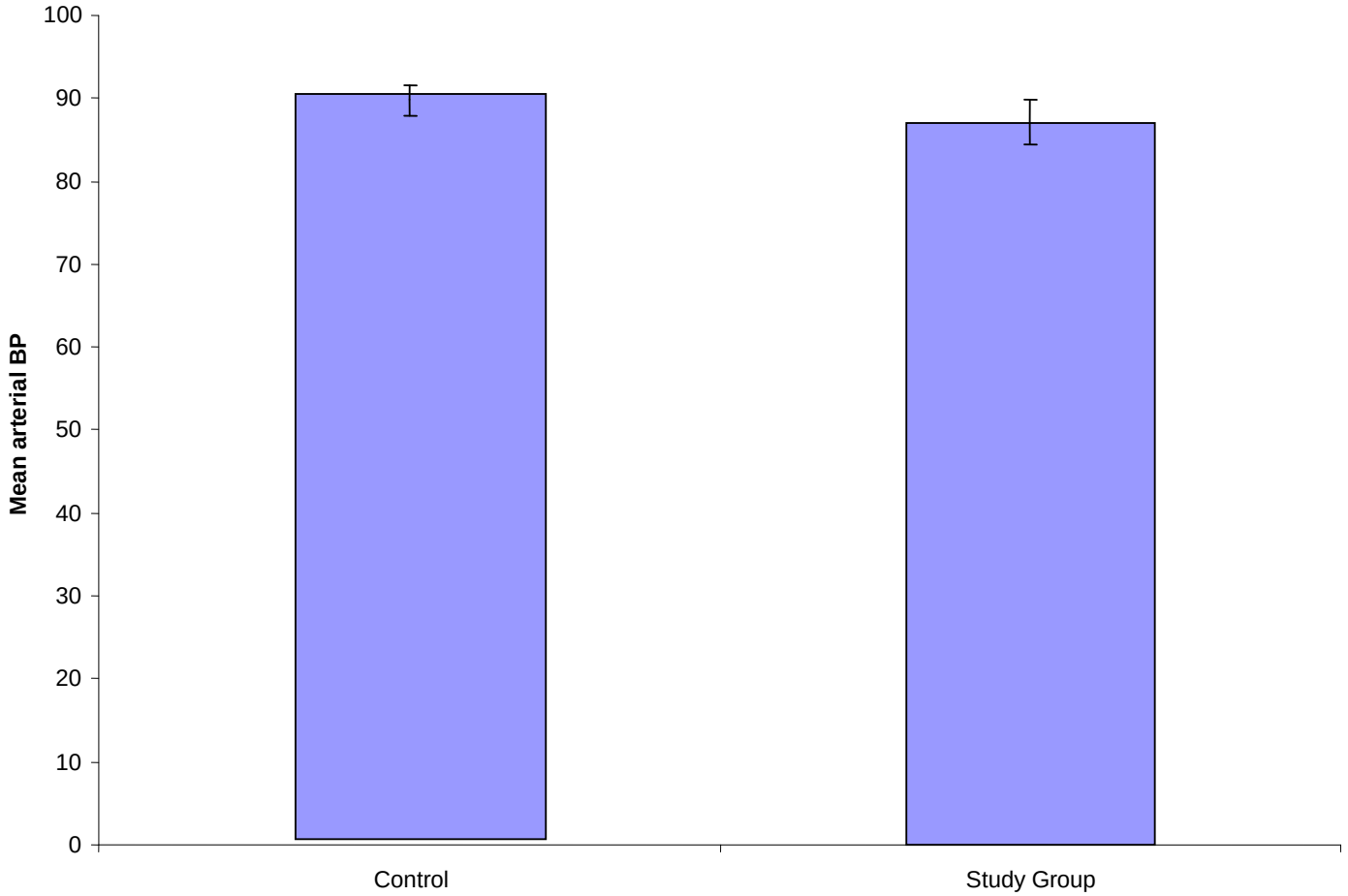


Fig. 11 Barchart showing Mean ± SEM of mean arterial Blood pressure control and study group (control n=30, study group n=30) p>0.05

4.3 Haematological Parameter

The mean ± SEM of Hb concentration, PCV Neutrophils, Lymphcytes, Monocytes and Esinophils of the two groups are shown in table 3 and figures 7.8 and 10. The mean Hb concentration, PCV, Neutrophils, lymphocytes, monocytes and eosinophils were 13.84 ± 0.20 and 13.81 ± 0.25, 41.53 ± 0.60 g/dl and 41.43 ± 0.76%, 59.43 ± 0.57 and 59.03 ± 0.55, 31.87 ± 0.67 and 32.50 ± 0.79%, 6.47 ± 0.21 and 6.47 ± 0.25, 3.04 ± 0.26 and 3.10 ± 0.28 respectively. There were no significant difference found between the two groups (of the respective parameters).

Table 3: The mean and SEM of neutrophils, lymphocytes monocytes, eosinophils, haemoglobin concentration (Hb) packed cell volume (PCV) of women that do not use wood to cook (group 1), and women that use wood to cook (group 2).

Parameters	Control Group Group 1 (n =30) Mean + SEM	Study Group Group 2 (n =30) Mean + Sem	Percentage changes %	Z -test	P-value
Hb Conc. G/dl	13.84± 0.20	13.81 ± 0.25	0.22	0.105	P>0.05
PCV (%)	41.53 ± 0.60	41.43 ± 0.76	0.24	0.103	P>0.05
Differential					
Leucocyte count					
Neutrophils (%)	59.43 ± 0.57	59.03 ± 0.55	0.67	0.50	P>0.05
Lymphocytes (%)	31.87± 0.67	32.50±0.79	1.97	0.61	P>0.05
Monocytes (%)	6.47± 0.21	6.47± 0.25	-	0	P>0.05
Eosinophils (%)	3.04 ± 0.26	3.10 ± 0.28	1.97	0.156	P>0.05
Basophils	0	0	0	0	P >0.05

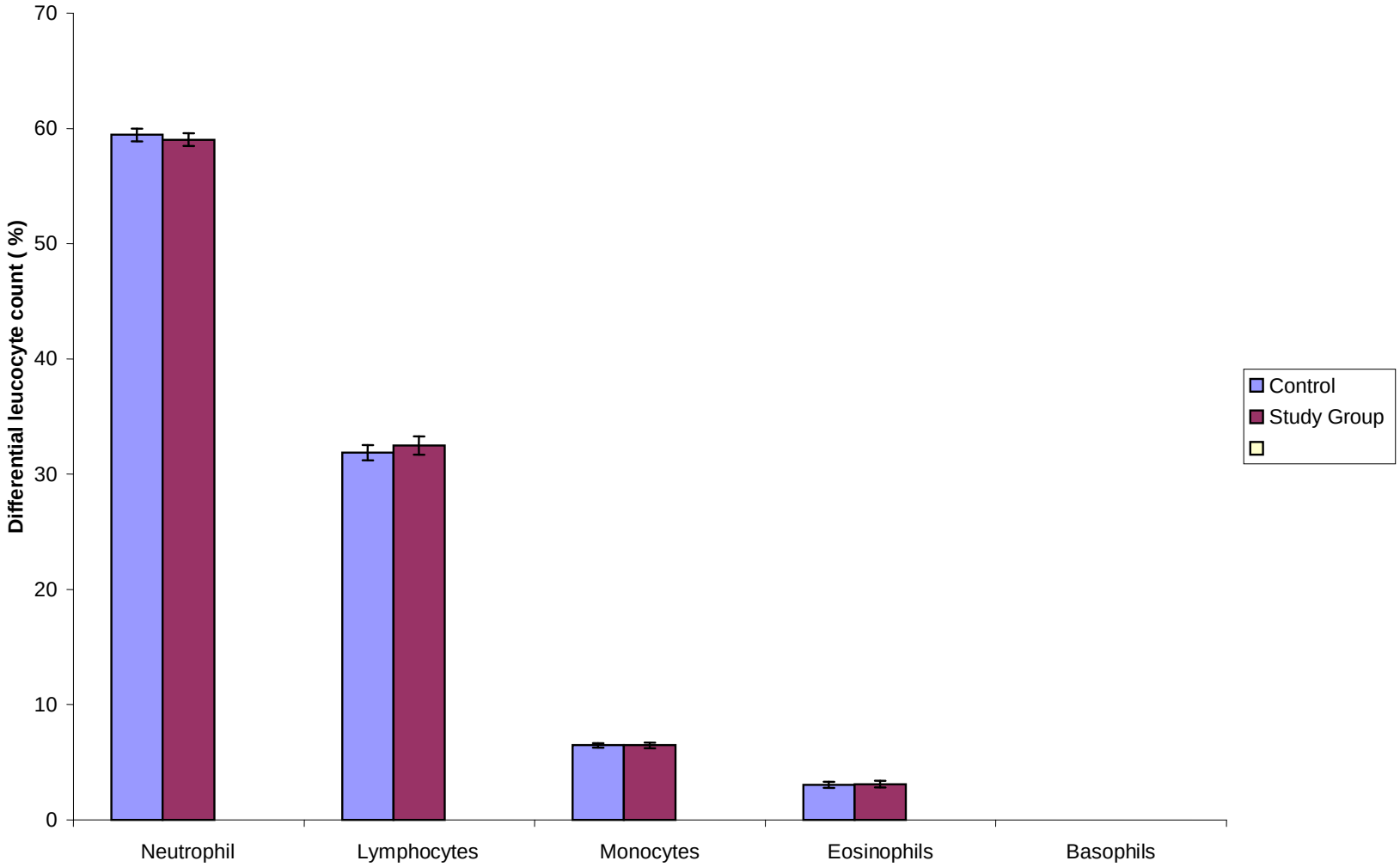


Fig 7: Bar chart showing mean SEM of differential leucocyte count of control and study group (control n=30, study group =30) p>0.05

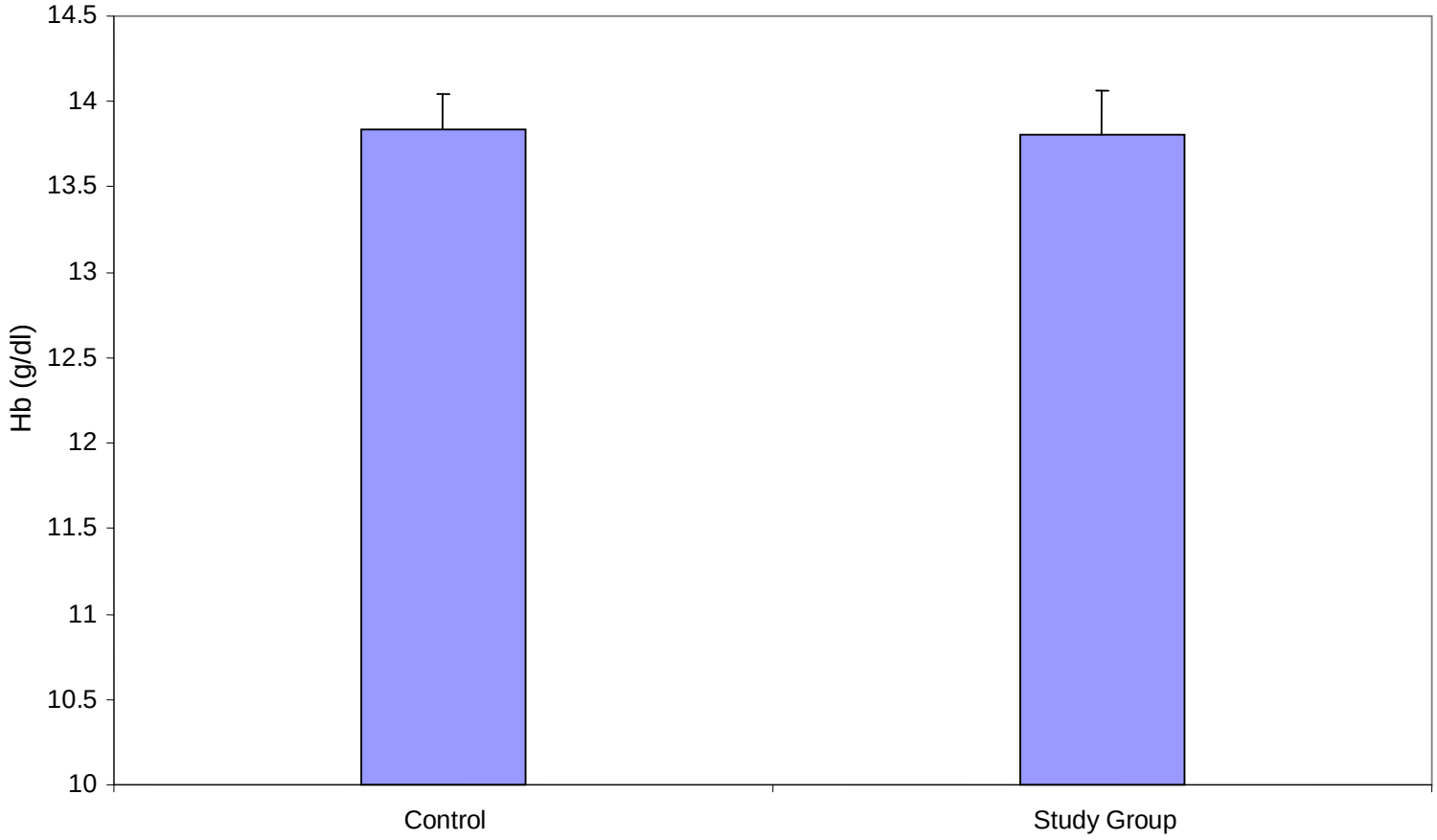


Fig. 8: Barchart showing mean ± SEM of Hb concentration of control and study group (Control n=30, study group n=30) p>0.05

4.4 Respiratory Function Test Parameters

The mean ± SEM were shown on table 4 and figures 9 and10 for predicted peak expiratory flow rate (PPEFR), actual peak expiratory flow rate (APEFR) and forced expiratory volume percentage (FEV%). The mean ± SEM of predicted peak expiration flow rate and forced expiratory volume percentage were 3.96 ± 0.10 and 3.88 ± 0.0l/sec and 81.83 ± 1.33 and 78.39 ± 1.61% respectively. There were no significant statistical difference between the two groups, while there were significant statistical difference ($P < 0.05$) for the actual peak expiratory flow rate (APEFR) between the two groups mean ± SEM = 4.87 ± 0.13 and 4.43 ± 0.12 for group 1 and 2 respectively.

When the predicted peak expiratory flow rate (PFEFR) and actual peak expiratory flow rate (APEFR) were compared in the control group there was a significant statistical difference ($P < 0.05$). The same was observed for the study group being found that ($P < 0.05$) there was a significant statistical difference between PPEFR AND APEFR.

Figures 1 to 10 shows the bar charts of the mean ± SEM of the different parameters that were studied of the study group and control group for graphical understanding.

No correlation was found between any of the parameters that were studied.

Table 4: The mean, SEM, percentage changes Z test and P value of predicted peak expiratory flow rate (PPEFR), Actual peak expiratory flow rate (APEFR), forced expiratory volume percentage (FEV %), forced vital capacity, forced expiratory volume in one second (FEV1).

Parameters	Control Group Group 1 (n =30) Mean + SEM	Study Group Group 2 (n =30) Mean + SEM	Percentage changes %	Z-Value	P-value
PPEFR L/Sec	3.96± 0.10	3.88 ± 0.04	2.02	0.715	P>0.05
APEFR L/Sec	4.87 ± 0.13	4.43 ± 0.12	9	2.488	P<0.05*
FEV_1%	81.83 ± 1.33	78.39 ± 1.61	4.2	1.639	P>0.05

% changes = $\frac{GP\ 1 - GP\ 2}{GP1 \times 100}$

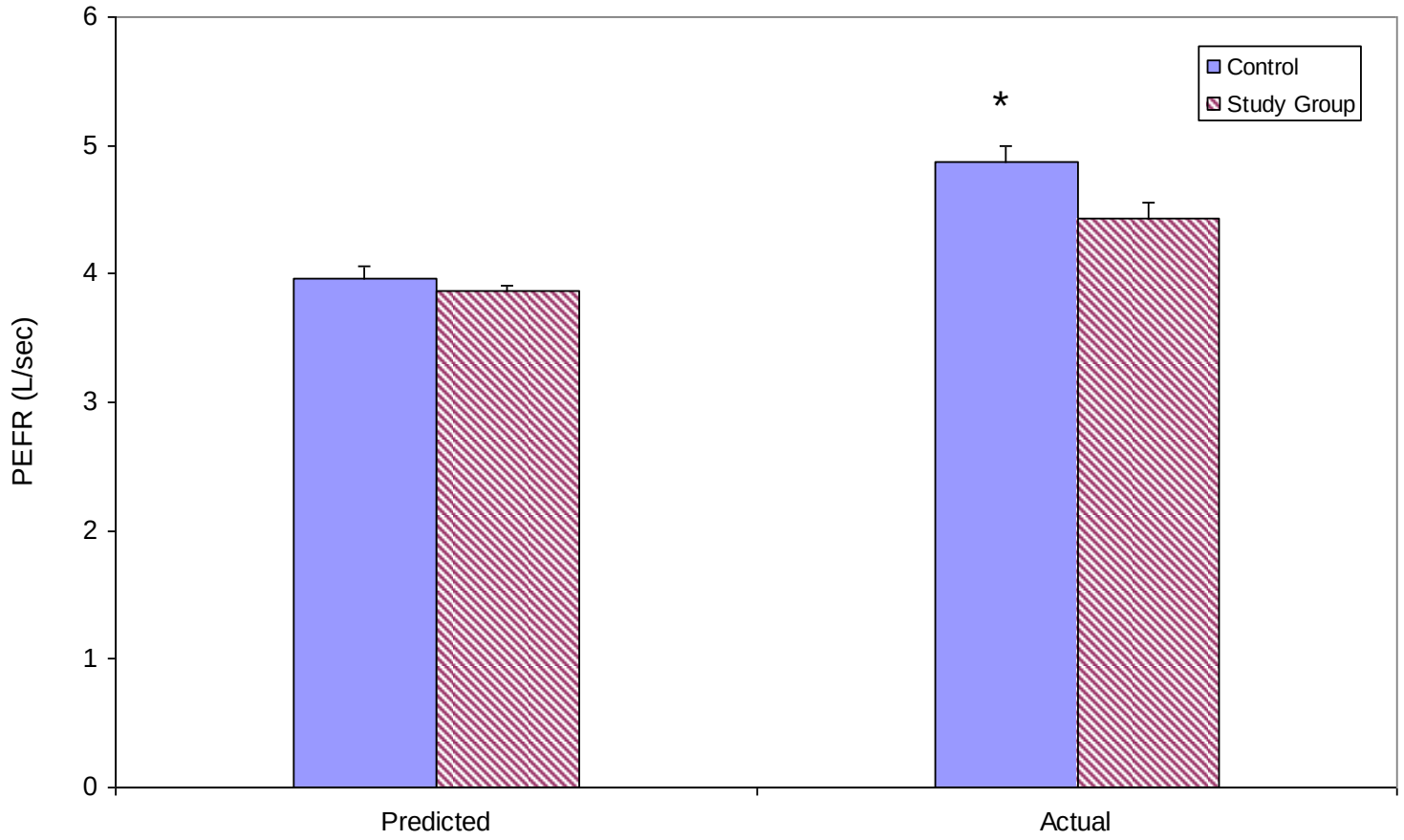


Fig. 9: Barchart showing mean ± SEM of predicted and actual PEFR of control and study group * P < 0.05 (Control n=30, study group n=30)

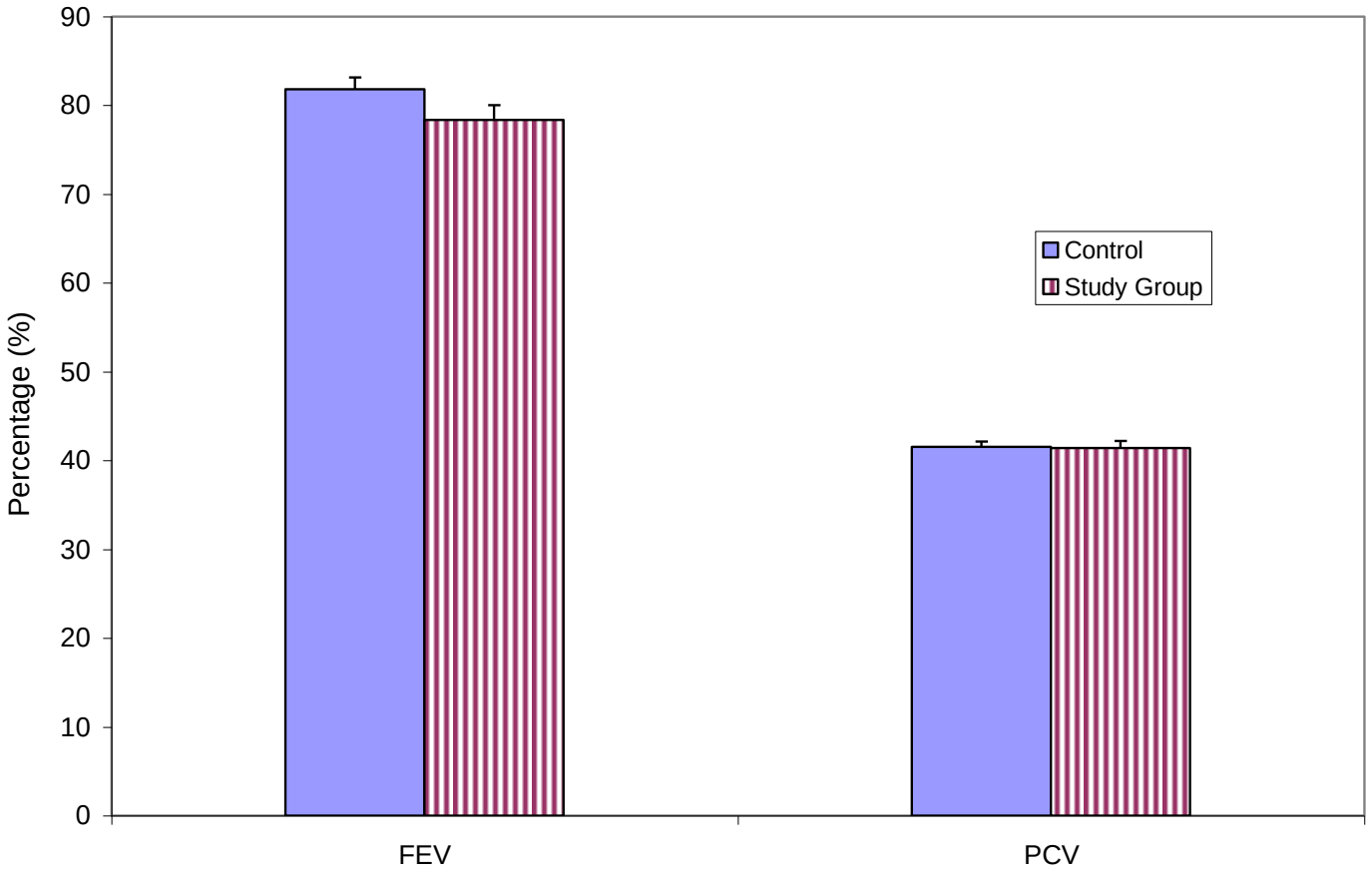


Fig. 10: Barchart showing mean ± SEM of FEV % and PCV of control and study group (control n=30, study group n=30) P>0.05

CHAPTER 5

DISCUSSION

Despite the important role of biomass fuels in the economy of developing countries, there are no occupational health standards for domestic cooks (Pandey 1997). Some of the highest exposures to air pollution in the developing countries occur inside homes where biofuels are used for daily cooking (Dutt *et al* 1996). Exposure to domestic cooking fuels has been implicated as a cause of respiratory symptoms like chronic brochitis. Pulmonary function studies also revealed that various parameters are affected by domestic cooking fuels both in adults and children. Health effects of such exposure will depend upon several factors, such as location of kitchen, type of fuel used and adequacy of ventilation provided in the kitchen. As a majority of kitchens are located within fairly confined spaces with inadequate ventilatory facilities, the levels of indoor pollutants are likely to be very high (Behera, 2001). Significant alterations of lung function or hematological indices in women using biomass fuel is clinically undesirable. These adverse side effects of biomass fuel has led to more research into alternatives with less physiological consequences.

This study was carried out to investigate if exposure to firewood smoke (biomass) and other sources of fuel (kerosene, liquefied natural gas (LNG), electricity or two or more or all the above (Mixed fuel) is a potential risk factor for chronic obstructive pulmonary disease (COPD) and related cardiovascular disease

among women in Zaria, Nigeria in whom cigarette smoking and other known risk factors may not be the most frequent.

The results obtained from the study indicates that anthropometric indices/measurement of the control group were not significantly different from the study group (Table1, figure 1,2,3 and 4) except for weight which was found significantly lower in the study group ($P< 0.05$, Table1). This difference could be attributed to the difference in diet or socioeconomic status since the study group were from a lower socioeconomic status when compared to the control group. It was observed that women that use wood to cook use this source of fuel because it is cheaper, more economical as well as affordable, as compared to kerosene, gas or electricity.

The cardiovascular parameters investigated in the study were pulse rate, diastolic and systolic blood pressure and mean arterial blood pressure. The results obtained indicate that there were no significant differences between the two groups ($P>0.05$) (Table 2, Figure 5,6 and 11). All the values were within normal physiological ranges reported by Sembulingam and Sembulingam (2000); Guyton and Hall (2001) and Abu-Shita *et al* (2003).

Hematological parameters that were studied include hemoglobin concentration, packed cell volume and differencial white blood cells, (neutrophils, lymphocytes, monocytes, and eosinophils). The results show that there were no significant difference between the control group and study group ($P>0.05$ Table 3,

Figure 7, 8 and 10). Rutgers *et al* (2002) found that patients with COPD had a higher percentage of sputum neutrophils and eosinophils than healthy controls. They observed that there was an increase in eosinophils in airways of COPD patients.

Balzono *et al* (1999) observed that eosinophil and neutrophil count were significantly higher in asthmatic patients and COPD patients when compared with healthy control. Also Lacoste *et al* (1993) suggested that neutrophils may play a role in COPD suggesting a part in chronic airflow limitations.

Tesfaigzi *et al* (1998) and Neufeld *et al* (2004) found no significant difference in the number of macrophages, neutrophils, lymphocytes or eosinophils or on hemoglobin concentration (smokeless stove or smoky fire) in univariate or multivariate analysis respectively.

Lal *et al* 1993 worked on histomorphological changes in lungs of rats following exposure to wood smoke, they found that hematological studies showed marginal alterations in hemoglobin levels, ESR and PCV during 15 days. While significant changes in eosinophils were observed during 30 and 45 days.

The respiratory parameters that were investigated were actual peak expiratory flow rate (APEFR) predicted peak expiratory flow rate (PPEFR) and forced expiratory volume in 1 second percentage ratio (FEV_1%). The results indicated that there were no significant difference in predicted peak expiratory flow rate and forced expiratory volume in 1 second percentage ratio between the

two groups (P> 0.05) (Table 4, figure 9 and 10). There was a significant difference in actual peak expiratory flow rate between the control group and the study group (p> 0.05). The mean FEV_1% ratio was within normal physiological range.

Actual PEFR for both groups were higher than the calculated value of predicted PEFR. Studies have shown that factors like weight, height, age, BMI, sex, race or ethnic origin affects the predicted PEFR (Madama, 1998). Although, this study did not reveal any significant correlation between PEFR or any of the various parameters investigated including BMI, the difference observed may be due to any of the above stated variables especially weight, race, BMI or ethnic origin.

The significant difference obtained in actual PEFR between the control group and study group is in agreement with the results obtained by other researchers. Peters *et al* 1999 determined the lung function status of some Nigerian men and women chronically exposed to fish drying using burning firewood. There were 182 male and 192 females aged 20-45 years who had been exposed for a maximum of five years. The control group comprised sex matched males (142) and females (152) Nigerian from the same area who were not exposed to any known pollutant. Lung function indices were significantly lower in the men and women engaged in firewood fish dying/industry than in the control (FVC, FEV_1 and PEFR). All the lung function indices (except FEV_1%) of the fishermen

and women declined significantly ($P<0.05$) with their duration of exposure.

Dutt *et al* 1996 studied the effect of exposure to indoor air pollution from the use of cooking fuels on lung functions and respiratory symptoms in women aged 15-60 years. The participants were 105 women using biofuel, 105 using kerosine and 105 using liquid petroleum gas (LPG), selected from among 1117 women aged 15-60 years. Lung functions were assessed by measuring forced vital capacity (FVC), forced expiratory volume in the first, second (FEV_1) and peak expiratory flow rate (PEFR) women using biofuel experienced more respiration symptoms (23%) than those using kerosine (13%; $P>0.05$) or (8%; $P<0.05$). Lung functions FVC, FEV_1, FEV_1% and PEFR were significantly lower in biofuel users compared with both kerosine and LPG users. Lung functions in kerosine users also were significantly poorer when compared with LPG users ($P< 0.01$)

Behera (1997) also worked with women using for different types of cooking fuels; biomass fuel, Liquified Petroleum Gas (LPG), kerosine used in stoves and a combination of two or more of these (mixed). Three parameters of ventilatory function (FVC, FEV_1, PEFR) were evaluated. Mixed fuels and biomass fuels affected FVC values more adversely, similar trend was observed for FEV_1 as well. Users of biomass fuel had the lowest mean value for PEFR. In users of mixed fuels there was a decline in FVC, FEV_1 and PEFR as exposure increased.

This study demonstrates that wood smoke users (biomass) actually had a lower actual PEFR than the control group. In summary it could be said that wood

smoke actually decreased actual PEFR of the women who used wood as a source fuel.

CHAPTER 6

CONCLUSION/RECOMMENDATION

It may be interesting to conduct a study on long term effects of wood smoke on:

a) Measurements of carbon monoxide in the blood

b) Various lung function parameters such as FVC, FEV_1, FEV_1% and PEFR

c) The size of particulate particles in the various kitchens.

d) Haematological parameters such as differential white blood cells count, packed cell volume, hemoglobin concentration.

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APPENDIX I

Questionnaire

Age……..

Weight…….

Height…….

No disease like

Bronchitis, Asthma, Pneumonia, Diabetes	Yes/No
Heart condition/Problems	Yes/No

Not having any form of interaction

No disability

Pulse rate	Beats/min
Blood Pressure — S.B.P	
D.B.P	mm Hg

Complete Blood Count

White Blood cell count

Neutrophils	/mm3
Lymphocytes	/mm3
Monocytes	/mm3
Eosinophils	/mm3
Basophilis	/mm3
Haemoglobin concentration	g/100ml

Kitchen location…….....

Type of job…….

Duration or how many hours a day are spent by the fire side?.....

Lung Function

1. PEFR 1^{st}

2^{nd}

3^{rd}

Time Vital capacity

FEV_1 PVC

PCV

APPENDIX 2

WOOD SMOKE

Age	Weight	Height	BMI	Pulse Rate	Systolic	Diastolic	Neutr	Lymp	Mono	Eosin	Hb	PPEFR	APEFR	FEV	PCV
40	54.4	1.575	21.93	72	120	90	60	37	3	-	16.7	3.73	5.12	85.5	50
40	77.2	1.625	29.24	84	140	90	62	28	7	3	16	3.9	3.95	77.1	48
38	88.3	1.675	31.47	87	70	40	59	30	7	4	14.3	4.12	4.67	75.6	43
35	57.7	1.55	24.02	90	110	75	60	33	4	3	16	3.73	4.45	81.6	48
39	114.8	1.75	37.06	79	120	70	60	30	7	3	13.3	4.36	3.38	55.6	40
45	61.5	1.625	23.29	76	100	80	58	36	4	2	11	3.81	4.5	83.3	33
32	62.6	1.575	25.24	65	90	70	58	34	6	2	14.3	3.87	3.83	73.7	43
36	88.1	1.6	34.41	60	130	90	58	33	7	2	13.7	3.89	4.95	76.1	41
37	90.1	1.625	34.12	90	120	75	64	28	7	2	13.7	3.96	4.28	75	41
35	67.5	1.6	26.37	88	120	70	60	32	6	-	15	3.9	3.95	75.9	45
35	66.8	1.625	25.3	84	120	70	60	28	8	4	15	3.99	5.38	88.7	45
43	103.1	1.55	42.91	84	100	70	57	33	8	4	14	3.58	4.17	78	42
36	80	1.58	32.25	88	130	90	66	24	6	3	12	3.8	3.38	85.7	36
32	62.5	1.65	22.96	84	140	90	63	30	7	-	14.3	4.14	4.78	80.8	43
40	53.3	1.55	22.27	70	130	90	55	40	7	-	12	3.64	3.83	75	36
38	51.3	1.6	20.04	76	130	90	50	45	5	-	13.3	3.85	5.05	75.5	40
40	71.5	1.575	28.82	80	120	70	60	26	5	6	13	3.58	4.22	83	39
30	75.2	1.6	26.33	80	90	60	60	35	8	-	15	3.99	3.45	89.8	45
42	76.1	1.7	26.33	68	90	70	55	37	5	-	12	4.13	3.55	82.5	36
30	59.6	1.575	24.03	80	90	75	60	30	8	3	14	3.91	4.55	89.8	42
33	50.6	1.755	20.4	64	90	60	60	30	7	3	14	3.85	4.78	86.3	42
37	62.3	1.575	25.12	72	120	80	58	30	7	6	13	3.78	4.33	81.1	39
39	69.7	1.675	24.84	72	120	80	55	37	6	-	14	4.1	3.33	62.3	42
39	45.8	1.42	22.71	88	115	75	55	35	8	2	15.7	3.2	4.61	81.3	47
42	64.9	1.625	24.58	80	120	80	60	30	8	2	12.7	3.87	4.95	84.6	38
37	57.8	1.75	18.87	86	120	80	60	35	8	2	11.7	4.4	5.22	79.7	35
33	70.5	1.7	24.39	80	70	40	60	30	6	4	12.7	3.49	4.67	85	38
35	60.7	1.6	23.71	76	120	90	60	35	5	-	15.3	3.9	5.62	78.6	46
34	67.1	1.55	27.93	70	110	80	60	30	8	-	13.3	3.75	4.17	52.5	40
35	88.6	1.65	32.54	65	90	60	58	34	6	2	13.3	4.08	5.87	72.3	40

APPENDIX 3

PPEFR	APEFR	FEV	PCV
3.73	5.12	85.5	50
3.9	3.95	77.1	48
4.12	4.67	75.6	43
3.73	4.45	81.6	48
4.36	3.38	55.6	40
3.81	4.5	83.3	33
3.87	3.83	73.7	43
3.89	4.95	76.1	41
3.96	4.28	75	41
3.9	3.95	75.9	45
3.99	5.38	88.7	45
3.58	4.17	78	42
3.8	3.38	85.7	36
4.14	4.78	80.8	43
3.64	3.83	75	36
3.85	5.05	75.5	40
3.58	4.22	83	39
3.99	3.45	89.8	45
4.13	3.55	82.5	36
3.91	4.55	89.8	42
3.85	4.78	86.3	42
3.78	4.33	81.1	39
4.1	3.33	62.3	42
3.2	4.61	81.3	47
3.87	4.95	84.6	38
4.4	5.22	79.7	35
3.49	4.67	85	38
3.9	5.62	78.6	46
3.75	4.17	52.5	40
4.08	5.87	72.3	40

APPENDIX 4

NOT WOOD

Age	Weight	Height	BMI	Pulse Rate	Systolic	Diastolic	Neutr	Lymp	Mono	Eosin	Hb
38	87.3	1.675	31.1	76	110	90	55	35	6	4	13.3
35	66.3	1.6	25.9	72	120	90	60	33	7		14
38	95.1	1.65	34.9	76	100	70	60	30	8	2	14.3
45	67.3	1.6	26.3	64	120	80	58	37	5		14
38	84.3	1.55	35.1	76	130	95	60	30	7	3	15
35	68.1	1.725	22.9	80	120	90	55	35	5	5	12
43	69.2	1.7	23.95	65	110	70	58	34	6	2	11
43	98.9	1.625	37.4	65	120	80	63	27	8	2	16.3
42	61.6	1.67	20.1	76	120	80	55	35	8	2	13.7
31	74.4	1.6	29.1	68	110	80	52	40	8		14.3
37	88	1.7	30.5	80	120	80	53	40	7		13.7
35	76.4	1.65	28.1	84	130	80	60	30	8	2	14.3
35	79.5	1.75	26	74	110	70	58	35	7		13.3
40	55.6	1.525	23.9	78	70	40	60	30	7	3	12.3
42	92.6	1.67	33.2	68	120	80	60	30	7	3	13.3
33	113.1	1.575	45.6	80	100	70	60	30	6	4	14.3
44	89	1.575	35.9	65	140	90	58	35	7		12.7
45	85.9	1.57	34.6	68	110	80	65	25	8	2	14.7
43	85	1.625	32.2	76	100	70	60	30	8	2	12.7
33	74.4	1.7	25.7	86	100	65	63	30	5	2	13.3
40	71.6	1.6	28	82	90	60	60	30	6	4	14.3
44	65.7	1.575	26.5	82	120	80	60	30	6	4	14.7
34	71.3	1.625	27	72	110	70	60	35	5		13.7
38	85.9	1.625	32.5	88	130	95	65	29	5	1	15
30	63	1.575	25.4	68	110	70	60	30	6	4	15
44	81.9	1.525	35.2	72	140	80	60	35	5		14.7
35	71.2	1.625	27	80	120	80	60	30	6	4	12.7
42	68.3	1.525	29.4	88	100	70	60	30	7	3	13.3
32	74.3	1.55	30.9	75	110	60	60	30	4	6	14
38	52.8	1.6	20.6	84	120	65	65	26	6	3	15.3

APPENDIX 5

PPEFR	APEFR	FEV	PCV
4.12	5.9	85	40
3.9	4.38	75.6	42
3.22	3.88	70.7	43
3.72	5.22	84.6	42
3.67	4.33	84.8	45
4.35	6.12	100	36
4.11	5.85	75.4	33
3.85	6.28	86.4	49
4.03	4.78	86.5	41
3.36	4.45	81.4	43
4.22	4.83	85.3	41
4.08	3.83	80	43
4.44	5.12	70	40
3.55	3.78	85.7	37
4.04	5.28	82.5	40
3.85	5.78	74.4	43
3.65	4.88	86.8	38
3.64	4.62	78.1	44
3.8	4.45	78.4	38
4.29	4.72	76.2	40
3.81	5.45	83.3	43
3.65	4.38	73.2	44
4.01	4.83	72.2	41
3.94	5.62	85	45
3.9	4.78	98.1	45
3.48	4.44	90.7	44
3.99	4.38	87	38
6.51	4.33	80	40
3.78	4.05	73.7	42
3.85	5.5	83.9	46