EFFECT OF ANTIOXIDANTS ON CARDIOVASCULAR DISEASE

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INSTITUTE OF NUTRITION AND FOOD SCIENCE

UNIVERSITY OF DHAKA

April: 2012

EFFECT OF ANTIOXIDANTS ON CARDIOVASCULAR DISEASE

A DISSERTATION SUBMITTED TO UNIVERSITY OF DHAKA IN PARTIAL FULFILLMENT OF THE REQUIRMENT OF THE DEGREE OF MASTER OF PHILOSOPHY IN NUTRITION

SUBMITTED BY:

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SESSIONS: 2004-2005

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DECLARATION

This is to certify that the thesis entitled "**Effect of Antioxidants on Cardiovascular Disease"** by **Nahida Akter** in partial fulfillment of the requirement for the aware of **Master of Philosophy in Nutrition** has been completed under my supervision.

It is certified that **Nahida Akter** has fulfilled all conditions laid down in the Academic Ordinance with regard to the M.Phil course work and to the best of my knowledge; the thesis contains his original research.

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DECLARATION

I, hereby, humbly declare that this thesis entitled "**Effect of Antioxidants on Cardiovascular Disease"** based on works carried by me. No part of it has been presented for higher degree.

The research work was carried out in

1) Institute of Nutrition and food Science (INFS), University of Dhaka and
2) Department of Cardiology, Bangabandhu Sheikh Mujib Medical University Dhaka, Bangladesh.

Under the guidance of professor Dr.A.M.M Mokarram Hossain Institute of Nutrition and Food Science (INFS), University of Dhaka.

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ABSTRACT

An Intervention study was carried out among 150 cardiovascular disease patients to observe the Effect of Antioxidants on Cardiovascular Disease, which was carried out in indoor & outdoor patients Department of Cardiology of Bangabandhu Sheikh Mujib Medical University. The diseases upon which the experiment was carried out were on Myocardial infraction (50), Hypertension (50), Ischemic Heart Disease (50) patients. Data were collected with pre-tested and well designed questionnaires.

Most of the respondents (56%) age were 57-60 years, the educational level were mainly graduate (54.7%). In relation to occupation, business was the highest percentage (50%). Most of the families income were >25000 TK.

The main sign and symptoms were chest pain, palpitation, edema, breathing problem, & high BP. After antioxidant treatment the above clinical sign and symptoms were significantly reduced (P=0.000).

In terms of biochemical measurements such as- serum cholesterol, serum LDL and serum TG level were reduced significantly (P=0.000) after antioxidant treatment. On the other hand blood hemoglobin, serum HDL and serum homocystein were increased significantly (P=0.000) after antioxidant treatment.

Antioxidants activity lowering effects on sign and symptoms were much higher in hypertensive patients followed by MI and IHD. Where as antioxidant effects on BP (systolic and diastolic) were much higher in IHD patients followed by hypertensive and MI patients.

The antioxidants had higher percentage of increasing effect on serum folate in hypertensive patients and also hemoglobin levels were increased in MI patients. Serum cholesterol and serum LDL were decreased in hypertensive patients. Whereas TG level was decreased in IHD patients.

There were significant relationship among socio-demography (sex, education, occupation, and income), clinical sign & symptoms (chest pain, palpitation, edema, breathing problem and high BP) and biochemical investigations (Hemoglobin level, serum folic acid, and serum lipid profile).

Significant correlation among different variables (clinical and biochemical investigations) of the participants were observed.

On the basis of the analysis and interpretation of the findings it is necessary to take antioxidants as supplementations to reduce dysfunctional conditions of the heart (heart diseases) and vessels that supply oxygen to organs and tissues throughout the body.

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ABBREVIATIONS

FR	:	Free Radicals
CVD	:	Cardiovascular Diseases
AF	:	Antioxidant Foods
ADS	:	Antioxidant Dietary Supplements
AMI	:	Acute Myocardial Infarction
ACS	:	Acute Coronary Syndrome
IMR	:	Infant Mortality Rate
AP	:	Angina Pectoris
OS	:	Oxidative Stress
ANOVA	:	One Way Analysis of Variance
CI	:	Confidence Interval
STD	:	Standard Deviation
RDA	:	Recommended Dietary Allowances
MI	:	Myocardial Infraction
IHD	:	Ischemic Heart Disease
BP	:	Blood Pressure
HDL	:	High Density Lipoprotein
LDL	:	Low Density Lipoprotein
TG	:	Triglyceride
BMI	:	Body Mass Index
Hb	:	Heamoglobin

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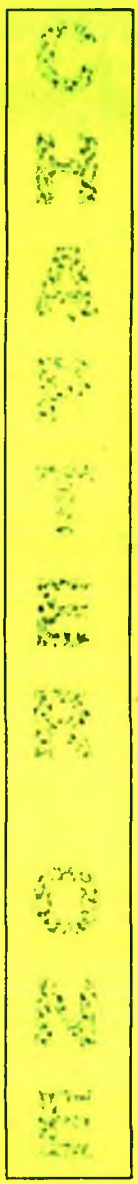

INTRODUCTION

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1.1 Introduction

1.1.1 Cardiovascular Diseases

Cardiovascular pertains to cardio (heart) and vascular (blood vessels). Thus, cardiovascular disease (CVD) refers to dysfunctional conditions of the heart (heart disease) and vessels (particularly the arteries) that supply oxygen to organs and tissues throughout the body, including the brain, heart, and other vital organs.[1] CVD includes arteriosclerosis, coronary artery disease, heart valve disease, arrhythmia, heart failure, hypertension, orthostatic hypotension, and shock, endocarditic, disorders of the peripheral vascular system, and congenital heart disease and stroke (WHO).

1.1.2 The Oxidative-Modification Hypothesis of Cardiovascular Disease

According to the oxidative-modification hypothesis, LDL initially accumulates in the extracellular subendothelial space of arteries and, through the action of resident vascular cells, is mildly oxidized to a form known as minimally modified LDL.[2] This minimally modified LDL induces local vascular cells to produce monocyte chemotactic protein 1 and granulocyte and macrophage colony-stimulating factors, which stimulate monocyte recruitment and differentiation to macrophages in arterial walls.[3] The accumulating monocytes and macrophages, stimulate further peroxidation of LDL. The products of this reaction make the protein component of LDL (apolipoprotein B-100) more negatively charged. By virtue of its increased negative charge, this completely oxidized LDL is recognized by scavenger receptors on macrophages and internalized to form so-called foam cells.[4] In contrast to the uptake of native (unoxidized) LDL by the LDL (apolipoproteins B and E) receptor on macrophages, the uptake of oxidized LDL by the scavenger-receptor pathway is not subject to negative-feedback regulation and thus results in massive uptake of cholesterol (from oxidized LDL) by the macrophages.

In addition to promoting the formation of foam cells, oxidized LDL has direct chemotactic activity for monocytes [5] and stimulates the binding of monocytes to the endothelium.[6] Once monocytes cross the endothelial layer, they become trapped in the subendothelial space, partly because oxidized LDL inhibits their egress from the arterial wall.[7] Oxidized LDL is also cytotoxic to vascular cells, [8, 9] thus promoting the release of

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lipids and lysosomal enzymes into the intimal extracellular space and enhancing the progression of atherosclerotic lesions[8].

The oxidative-modification hypothesis is supported by evidence that LDL oxidation occurs in vivo and contributes to the clinical manifestations of atherosclerosis. Antibodies raised against oxidized LDL react with atherosclerotic lesions but not with normal arterial segments[10]. LDL extracted from human atherosclerotic lesions, but not plasma-derived LDL, resembles LDL that has been oxidatively modified in vitro[11].

Thus, oxidative modification of LDL appears to have an important role in foam-cell formation and atherogenesis. This link between the oxidation of LDL and atherogenesis provides a convenient and simple rationale for the beneficial effect of antioxidants on the incidence of coronary artery disease. A number of clinical studies and studies in animals have explored this link between LDL oxidation and atherogenesis

1.1.3 What Are Antioxidants

Antioxidants are substances that are capable of counteracting the damaging, but normal, effects of the physiological process of **oxidation** in animal tissue. Antioxidants are **nutrients** (vitamins and minerals) as well as **enzymes** (proteins in your body that assist in chemical reactions)[12]. They are believed to play a role in preventing the development of such chronic diseases as cancer, heart disease, stroke, Alzheimer's disease, Rheumatoid arthritis, and cataracts.

1.1.4 Antioxidant Process: (Antioxidant Metabolites /Antioxidant: Pro-Oxidant Activities)

Antioxidants block the process of oxidation by **neutralizing** free radicals. In doing so, the antioxidants themselves become oxidized [13]. That is why there is a constant need to replenish our antioxidant resources in our body.

How they work can be classified in one of two ways:

i. Chain-breaking - When a free radical releases or steals an electron, a second radical is formed. This molecule then turns around and does the same thing to a third molecule, continuing to generate more unstable products[14]. The process continues until termination

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occurs either the radical is stabilized by a chain-breaking antioxidant such as beta-carotene and vitamins C and E, or it simply decays into a harmless product.

ii. Preventive - Antioxidant enzymes like superoxide dismutase, catalase and glutathione peroxidase prevent oxidation by reducing the rate of chain initiation. They can also prevent oxidation by stabilizing transition metal radicals such as copper and iron [15].

The effectiveness of any given antioxidant in the body depends on which free radical is involved, how and where it is generated, and where the target of damage is. Thus, while in one particular system an antioxidant may protect against free radicals, in other systems it could have no effect at all. Or, in certain circumstances, an antioxidant may even act as a "**pro-oxidant**" that generates toxic oxygen species.

1.1.5 Types of Antioxidants

i. Antioxidant Nutrients

Antioxidants from our diet appear to be of great importance in controlling damage by free radicals. Each nutrient is unique in terms of its structure and antioxidant function.

Vitamin E is actually a generic term that refers to all entities that exhibit biological activity of the isomer tocopherol (an isomer is one of two or more molecules that have the same chemical formula but different atomic arrangements). Alpha-tocopherol, the most widely available isomer, has the highest biopotency or strongest effect in the body, because it is fat-soluble. Alpha-tocopherol is in a unique position to safeguard cell membranes largely composed of fatty acids from damage by free radicals. Alpha-tocopherol also protects the fats in low-density lipoproteins (LDLs, or the "bad" cholesterol) from oxidation [16].

Vitamin C also known as ascorbic acid is a water-soluble vitamin. As such, it scavenges free radicals that are in an aqueous (watery) environment, such as inside your cells [16].

Beta-carotene also a water-soluble vitamin, it is thought to be the best quencher of singlet oxygen (an energized but uncharged form of oxygen that is toxic to cells). Beta-carotene is also especially excellent at scavenging free radicals in low oxygen concentration.

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Selenium is a trace element. It is a mineral that we need to consume in only very small quantities, but without which we could not survive. It forms the active site of several antioxidant enzymes including glutathione peroxidase[16].

Manganese and zinc, the minerals manganese and zinc are trace elements that form an essential part of various antioxidant enzymes[16].

ii. Antioxidant Enzymes/ Antioxidant Enzymes System

The antioxidant enzymes **superoxide dismutase** (SOD), **catalase** (CAT) and **glutathione peroxidase** (GPx) serve as your primary line of defense in destroying free radicals. SOD first reduces (adds an electron to) the radical superoxide ($O2^-$) to form hydrogen peroxide (H2O2) and oxygen (O2)[16, 17].

$2O2^- + 2H$ --SOD--> H2O2 + O2

Catalase and GPx then work simultaneously with the protein glutathione to reduce hydrogen peroxide and ultimately produce water (H2O).

2H2O2 --CAT--> H2O + O2

H2O2 + 2glutathione --GPx--> oxidized glutathione + 2H2O

Together, they repair oxidized DNA, degrade oxidized protein, and destroy oxidized lipids. Various other enzymes act as a secondary antioxidant defense mechanism to protect you from further damage.

iii. Other Antioxidants

Enzymes, vitamins, and minerals, there appear to be many other nutrients and compounds that have antioxidant properties. Among them is **coenzyme Q10** which is essential to energy production and can also protect the body from destructive free radicals[18]. Also, **uric acid**, a product of DNA metabolism, has become increasingly recognized as an important antioxidant. Additionally, substances in plants called **phytochemicals** are being investigated for their antioxidant activity and health-promoting potential.[18]

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1.1.6 Recommended Dietary Allowance

Since 1941, the Food and Nutrition Board has determined the types and quantities of nutrients that are needed for healthy diets by reviewing scientific literature, considering how nutrients protect against disease, and interpreting data on consumption of nutrients[19]. For each type of nutrient, the Board has established a Recommended Dietary Allowance (RDA)-a daily intake goal for nearly all (98 percent) healthy individuals, and a "tolerable upper intake level" (UL)-the maximum amount of a nutrient that healthy individuals can take each day without risking adverse health effects. In some cases, the Board has decided there isn't enough evidence to determine the amount at which a particular nutrient is essential or harmful to health.

The following recommendations were made for consumption of antioxidants in the 2000 report by American Heart Association.

Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids

Antioxidant	RDA (adults)	Upper Level (adults)	Comment
Vitamin E	15 mg	1,070 mg natural vitamin E 785 mg synthetic vitamin E	Higher amounts impair blood clotting, increasing likelihood of hemorrhage.
Vitamin C	Women: 75 mg Men: 90 mg	2,000 mg	Higher amounts could lead to diarrhea and other GI disturbances. Extremely high levels may lead to cancer, atherosclerosis, and kidney stones.
Beta-carotene	None	None	Chronic high doses turn your skin yellow-orange, but it is not toxic. However, research indicates it is unwise to consume doses of beta-carotene beyond what is in a multivitamin and your regular diet.
Selenium	55 micrograms	400 micrograms	Higher amounts could cause hair loss, skin rashes, fatigue, GI disturbances, and nervous system abnormalities.

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1.1.7 Source of Antioxidants

Below is a list of where to find specific antioxidants: [19]

Vitamin E is found in vegetable oils, walnuts, peanuts, almonds, seeds, olives, avocado, wheat germ, liver, and leafy green vegetables.

Vitamin C good sources for look to citrus fruits (like oranges and grapefruit), broccoli, leafy green vegetables, tomatoes, peppers, potatoes, cantaloupe, and strawberries.

Beta-carotene Common sources of include cantaloupe, mangoes, papaya, pumpkin, peppers, spinach, kale, squash, sweet potatoes, and apricots.

Selenium good sources of in seafood, beef, pork, chicken, Brazil nuts, brown rice, and whole wheat bread.

Phytochemicals are found in a variety of sources. Some phytochemicals that are currently under study for their antioxidant activity and ability to reduce disease risk.

Source of Phytochemical are listed below:

Phytochemical	**Food source**
Allyl sulfides	Onions, garlic, leeks, chives
Carotenoids	Tomatoes, carrots, watermelon, kale, spinach
Curcumin	Turmeric
Flavonoids	Grapes, blueberries, strawberries, cherries, apples, grapefruit, cranberries, raspberries, blackberries
Glutathione	Green leafy vegetables
Indoles	Broccoli, cauliflower, cabbage, Brussels
Isoflavones	Legumes (peas, soybeans)
Isothiocyanates	Broccoli, cauliflower, cabbage, Brussels sprouts, bok choy
Lignans	Seeds (flax seeds, sunflower seeds)
Monoterpenes	Citrus fruit peels, cherries, nuts
Phytic acid	Whole grains, legumes
Phenols, polyphenols, phenolic compounds	Grapes, blueberries, strawberries, cherries, grapefruit, cranberries, raspberries, blackberries, tea
Saponins	Beans, legumes

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1.1.8 Free Radical

It is ironic that oxygen, an element indispensable for life can, under certain situations, have severely deleterious effects on the human body. Most of the potentially harmful effects of oxygen are due to the formation and activity of a number of chemical compounds, known as reactive oxygen species, which have a tendency to donate oxygen to other substances. Many such reactive species are free radicals and have a surplus of one or more free-floating electrons rather than having matched pairs and are, therefore, unstable and highly reactive. Types of free radicals include the hydroxyl radical (OH.), the superoxide radical ($O._2$), the nitric oxide radical (NO.) and the lipid peroxyl radical (LOO.) [19].

1.1.9 Production of Free Radicals in the human body

Free radicals and other reactive oxygen species are derived either from normal essential metabolic processes in the human body (external source).

Free radical formation occurs continuously in the cells as a consequence of both enzymatic and non-enzymatic reactions (internal source). Enzymatic reactions which serve as sources of free radicals include those involved in the respiratory chain, in phagocytosis, in prostaglandin synthesis and in the cytochrome P450 system. Free radicals also arise in non-enzymatic reactions of oxygen with organic compounds as well as those initiated by ionizing radiations.

Some internally generated sources of free radicals are: [20]

- mitochondria
- phagocytes
- xanthine oxidase
- reactions involving iron and other transition metals
- arachidonate pathways
- peroxisomes
- exercise
- inflammation
- Ischaemia/reperfusion.

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Some externally generated sources of free radicals are: [20]

- cigarette smoke
- environmental pollutants
- radiation
- ultraviolet light
- certain drugs, pesticides, anaesthetics and industrial solvents
- Ozone.

1.1.10 Counteracting Free Radical Damage

The human body has several mechanisms to counteract damage by free radicals and other reactive oxygen species. These act on different oxidants as well as in different cellular compartments.

One important line of defence is a system of enzymes, including glutathione peroxidases, superoxide dismutases and catalase, which decrease concentrations of the most harmful oxidants in the tissues. Several essential minerals including selenium, copper, manganese and zinc are necessary for the formation or activity of this enzymes[21]. Hence; if the nutritional supply of these minerals is inadequate, enzymatic defences against free radicals may be impaired.

The second line of defence against free radical damage is the presence of antioxidants. An antioxidant is a molecule stable enough to donate an electron to a rampaging free radical and neutralize it, thus reducing its capacity to damage. Some such antioxidants, including glutathione, ubiquinol and uric acid, are produced during normal metabolism in the body. Other lighter antioxidants are found in the diet. Although about 4000 antioxidants have been identified, the best known are vitamin E, vitamin C and the carotenoids. Many other non-nutrient food substances, generally phenolic or polyphenolic compounds, display antioxidant properties and, thus, may be important for health[21].

Although a wide variety of antioxidants in foods contribute to disease prevention, the bulk of research has focused on three antioxidants which are essential nutrients or precursors of nutrients. These are vitamin E, vitamin C and the carotenoids. Each of these

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antioxidant nutrients has specific activities and they often work synergistically to enhance the overall antioxidant capability of the body.

1.1.11 Free Radical Diseases

The balance between the production of free radicals and the antioxidant defences in the body has important health implications. If there are too many free radicals produced and too few antioxidants, a condition of "oxidative stress" develops which may cause chronic damage. As mentioned above, free radicals have been implicated in several health problems are just a few examples:

i. Vitamin E is the collective name for eight compounds, four tocopherols and four tocotrienols, found in nature. It is a fat-soluble substance present in all cellular membranes and is mainly stored in adipose tissue, the liver and muscle. Vitamin E is a principal antioxidant in the body and protects polyunsaturated fatty acids in cell membranes from peroxidation. It is a singlet oxygen quencher, neutralizing these highly reactive and unstable singlet oxygen molecules. In fact, singlet oxygen can damage DNA and be mutagenic. Vitamin E also protects the double bonds of b-carotene from oxidation and thus exhibits a sparing effect. Due to the ability of vitamin E to work at higher oxygen pressures, free radicals are scavenged and tissue injury is minimized[18].

Vitamin E degenerative below these diseases:

a. Vitamin E and cancer

b. Vitamin E and cardiovascular disease

c. Vitamin E and eye disorders

d. Vitamin E and neurological disorders

e. Vitamin E and aging

ii. Vitamin C or ascorbic acid is a water-soluble vitamin. This vitamin is a free radical scavenger and interacts with free-radicals in the water compartment of cells as well as in

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the fluids between cells. It is considered to be one of the most important antioxidants in extra cellular fluids.

Vitamin C degenerative below these diseases:

a. Vitamin C and cancer

b. Vitamin C and cardiovascular disease

c. Vitamin C and cataracts

d. Vitamin C and other diseases

iii. Carotenoids are a group of red, orange and yellow pigments found in plant foods, particularly fruits and vegetables. Some carotenoids like b-carotene act as a precursor of vitamin A; others do not. B-carotene is an effective antioxidant as it is one of the most powerful singlet oxygen quenchers. It can dissipate the energy of singlet oxygen, thus preventing this active molecule from generating free radicals. Its other antioxidant properties include the scavenging of free radicals.

Carotenoids degenerative below these diseases:

a. Carotenoids and cancer

b. B-carotene and cardiovascular disease

1.1.12 Antioxidant Health Effects

i. Disease Treatment

The brain is uniquely vulnerable to oxidative injury, due to its high metabolic rate and elevated levels of polyunsaturated lipids, the target of lipid peroxidation. Consequently, antioxidants are commonly used as medications to treat various forms of brain injury. Here, superoxide dismutase mimetics, sodium thiopental and propofol are used to treat reperfusion injury and traumatic brain injury, while the experimental drug NXY-059 and ebselen are being applied in the treatment of stroke[22]. These compounds appear to prevent oxidative stress in neurons and prevent apoptosis and neurological damage.

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Antioxidants are also being investigated as possible treatments for neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis, and as a way to prevent noise-induced hearing loss[23].

ii. Disease Prevention

Antioxidants can cancel out the cell-damaging effects of free radicals. And there is evidence that some types of vegetables, and fruits in general, protect against a number of cancers. These observations suggested the idea that antioxidants might help prevent these conditions[24].

Many nutraceutical and health food companies sell formulations of antioxidants as dietary supplements and these are widely used in industrialized countries. These supplements may include specific antioxidant chemicals, like resveratrol (from grape seeds or knotweed roots), combinations of antioxidants, like the "ACES" products that contain beta carotene (provitamin **A**), vitamin **C**, vitamin **E** and Selenium, or herbs that contain antioxidants - such as green tea and jiaogulan. Although some levels of antioxidant vitamins and minerals in the diet are required for good health[25, 26, 27].

iii. Physical Exercise

During exercise, oxygen consumption can increase by a factor of more than 10. This leads to a large increase in the production of oxidants and results in damage that contributes to muscular fatigue during and after exercise[28,29,30].

The inflammatory response that occurs after strenuous exercise is also associated with oxidative stress, especially in the 24 hours after an exercise session. The immune system response to the damage done by exercise peaks 2 to 7 days after exercise, which is the period during which most of the adaptation that leads to greater fitness, occurs. During this process, free radicals are produced by neutrophils to remove damaged tissue. As a result, excessive antioxidant levels may inhibit recovery and adaptation mechanisms. Antioxidant supplements may also prevent any of the health gains that normally come from exercise, such as increased insulin sensitivity[31, 32, 33].

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1.1.13 Adverse Effects of Antioxidants

Relatively strong reducing acids can have antinutrient effects by binding to dietary minerals such as iron and zinc in the gastrointestinal tract and preventing them from being absorbed. Notable examples are oxalic acid, tannins and phytic acid, which are high in plant-based diets. Calcium and iron deficiencies are not uncommon in diets in developing countries where less meat is eaten and there is high consumption of phytic acid from beans and unleavened whole grain bread[34, 35, 36].

Foods	**Reducing acid present**
Cocoa and chocolate, spinach, turnip and rhubarb.	Oxalic acid
Whole grains, maize, legumes.	Phytic acid
Tea, beans, cabbage.	Tannins

Nonpolar antioxidants such as eugenol a major component of oil of cloves have toxicity limits that can be exceeded with the misuse of undiluted essential oils. Toxicity associated with high doses of water-soluble antioxidants such as ascorbic acid is less of a concern, as these compounds can be excreted rapidly in urine. More seriously, very high doses of some antioxidants may have harmful long-term effects[37, 38].

This was thought to occur since the environment of cancer cells causes high levels of oxidative stress, making these cells more susceptible to the further oxidative stress induced by treatments. As a result, by reducing the redox stress in cancer cells, antioxidant supplements could decrease the effectiveness of radiotherapy and chemotherapy. On the other hand, other reviews have suggested that antioxidants could reduce side effects or increase survival times[39, 40].

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1.2 Justification of the Study

Cardiovascular disease is the most important adult health problem in wealthy countries, but now a days this C.V.D are also problems in our countries, where biological factors such as obesity, hypertension, dyslipidemia, diabetes, inappropriate diet, cigarette smoking, and sedentary life-style have contributed to its dissemination[1].

Research concerning nutritional regimens has shown that persons who consume large amounts of fruit and vegetables have lower incidences of cardiovascular diseases, stroke, and tumors, although the precise mechanisms for this protective effect are not known. Possible explanations include (a) increased consumption of dietary fiber, (b) reduced consumption of dietary cholesterol and other lipids, and (c) increased intake of the antioxidant vitamins (Beta-carotene, C, and E).On the basis of epidemiologic observations and laboratory studies, beta carotene (vitamin A), vitamin-C, vitamin-E has attracted wide interest as agents that may prevent cardiovascular disease[41].

Oxidation of low-density lipoprotein (LDL or "bad") cholesterol is important in the development of fatty buildups in the arteries. This process, called atherosclerosis, can lead to heart attacks and strokes. Until recently, it was thought that LDL cholesterol lipoprotein oxidation and its biological effects could be prevented by using antioxidant supplements[42, 43].

At this time, the scientific evidence supports a diet high in food sources of antioxidants and other heart-protecting nutrients, such as fruits, vegetables, whole grains and nuts instead of antioxidant supplements to reduce risk of CVD.

The purpose of this study was to conduct a systematic review of the scientific literature to identify and assess the evidence for the efficacy of three antioxidants, vitamin E, vitamin C, and Beta-carotene for the prevention and treatment of cardiovascular disease (CVD) or modification of known risk factors for CVD.

Although the antioxidant defense system includes both endogenously and exogenously (diet) derived compounds, dietary antioxidants including vitamin C (ascorbic acid), vitamin E (α-tocopherol), and ß-carotene (provitamin A) have received the greatest attention with regard to coronary heart disease prevention.

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1.3 Objective:

1.3.1 General Objective:

The study will reveal the effects of Antioxidants (Beta-carotene, Vit- C, and vit-E) activity on cardiovascular disease patient (Myocardial Infraction, Hypertension, and Ischemic Heart Disease)

1.3.2 Specific Objectives:

i. *To determine the Socio-demographic Status of cardiovascular Disease Patients (MI, Hypertension and Ischemic Heart Disease).*
ii. *To observe the Sign & Symptoms of cardiovascular disease patients (Myocardial Infraction, Hypertension, Ischemic Heart Disease).*
iii. *To measure the antioxidants activity level in blood by estimating the Hb level, serum folic acid, serum lipid profile before intervention in C.V.D patient.*
iv. *To supplement Beta-carotene, Vit-E, Vit-C (Tablet) among the cardiovascular disease patients to assess the effects of Antioxidants activity.*
v. *To asses the Biochemical parameters in blood such as: Hb level, serum folic acid, serum lipid profile of Myocardial infraction, Hypertension, and Ischemic Heart Disease patients after the intervention program.*
vi. *To describe the effects of antioxidants activity on Myocardial Infraction, Hypertension, Ischemic Heart Disease patients.*
vii. *To evaluate the effectiveness of antioxidants in different clinical and biochemical parameters of hypertention, MI and IHD patients.*
viii. *To observe the association among different variables of the C.V.D patients attending in the study.*
ix. *To find out the co-relation among different variables of the C.V.D patients attending in the study.*

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1.4 Operational Definition:

i. Antioxidants: Antioxidants are natural substances that exist as vitamins, minerals and other compounds in foods. They are believed to help prevent disease by fighting free radicals, substances that harm the body when left unchecked.

ii. Free radicals: Free radicals are atoms or groups of atoms that have at least one unpaired electron, which makes them highly reactive. Free radicals promote beneficial oxidation that produces energy and kills bacterial invaders. In excess, however, they produce harmful oxidation that can damage cell membranes and cell contents.

iii. Atherosclerosis: Atherosclerosis, a disease of the large arteries, is the primary cause of heart disease and stroke.

iv. Cardiovascular diseases: Cardiovascular pertains to cardio (heart) and vascular (blood vessels). Thus, cardiovascular disease (CVD) refers to dysfunctional conditions of the heart (heart disease) and vessels (particularly the arteries) that supply oxygen to organs and tissues throughout the body, including the brain, heart, and other vital organs.

v. Antioxidant foods: Antioxidants comes from our diet appear to be of great importance in controlling damage by free radicals that is called. Antioxidant foods.such as: vitamin & minerals food-vit-a, vit-e, vita-c, selenium etc.

vi. Antioxidant dietary supplements: Antioxidant dietary supplements means antioxidants are come from diatery source, like vitamin & menarals food.

vii. Oxidative stress: If there are too many free radicals produced and too few antioxidants, a condition of "oxidative stress" develops which may cause chronic damage

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CHAPTER TWO

LITERATURE REVIEW

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LITERATURE REVIEW

Coronary artery disease (CAD) is the major cause of death in developed countries[1]. It may present as a typical `heart attack', as sudden death, or it may be detected at a later date and be described as a silent infarct. Good coronary care units, the use of thrombolytic and anti-arrhythmic drugs, and accurate methods for assessing cardiac function and coronary artery pathology have all successfully reduced in-hospital mortality. However, about 75% of those who have a myocardial infarction (MI) die outside hospital, and sudden death is the first and only manifestation in about 20% of all those who present with CAD. There are five conditions that predispose to premature death from CAD. These are: hypertension; hypercholesterolaemia; the post-menopausal state; a thrombotic tendency; and the risk of developing ventricular arrhythmias. Patients with any of these conditions may be offered appropriate pharmacological treatment. In addition, those known to have CAD because they have had a MI or suffer from angina or strongly suspected of having CAD, because they have cerebrovascular or peripheral vascular disease, should also be offered secondary preventive treatment with, for example, lipid-lowering therapy and aspirin. [43, 44]

All the above patient groups and the general population can be offered lifestyle advice, which may be considered under five headings: (i) smoking cessation; (ii) weight control; (iii) taking reasonable exercise; (iv) eating a proper diet; and (v) taking antioxidant therapy. The purpose of this review is to consider the role of antioxidants as a means of reducing the risk of premature death from CAD.So, it has been proposed that carotenoids (vit-A) and retinoids (vit-E) and vit-C are agents that may prevent those disorders. [45]

According to the Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle (G.S.O., G.E.G., M.D.T., S.B.); the Departments of Environmental Health and Medicine, University of Washington, they conducted a multicenter, randomized, double-blind controlled primary prevention trial the Beta-Carotene ,Vitamin-E,and Vit-C Efficacy Trial involving a total of 18,314 smokers, former smokers, and workers exposed to asbestos. The effects of a combination of 30 mg of beta carotene per day and 25,000 IU of retinol (vitamin A) in the form of retinyl palmitate per day, 50mg vit-C on the primary

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end point and 50mg vitamin-E, the incidence of Cardiovascular Diseases, were compared with those of placeb. In this study a total of 388 new cases of lung cancer were diagnosed during the 73,135 person-years of follow-up. The active-treatment group had a relative risk of lung cancer of 1.28 (95 percent confidence interval, 1.04 to 1.57; P = 0.02), as compared with the placebo group. There were statistically significant differences in the risks of other types of cardiovascular diseases. In the active-treatment group, the relative risk of death from any cause was, 1.46 (95 percent confidence interval, 1.07 to 2.00); and of death from cardiovascular disease, 1.26 (95 percent confidence interval, 0.99 to 1.61). On the basis of these findings, the randomized trial was stopped 21 months earlier than planned; follow-up will continue for another 5 years. After an average of four years of supplementation, the combination of beta carotene vit-E, vit-C had benefit and may have had an adverse effect on the no incidence of lung cancer, cardiovascular disease and no risk of death from lung cancer, cardiovascular disease. [46, 47, 48]

According to S.L. Nuttall, M.J. Kendall and U. Martin the Clinical Pharmacology Section, Department of Medicine, University of Birmingham, Birmingham, UK, they conducted the potential role of antioxidants in the prevention and treatment of disease, particularly coronary artery, cerebrovascular and peripheral vascular disease, needs to be determined. This review indicates how agents may achieve their beneficial effects and also presents some early evidence that combinations of antioxidants, available in the form of pharmaceutical preparations, may produce measurable changes in serum, and presumably tissue, antioxidant defences. The next stage will be to define a desirable level of antioxidant activity and demonstrate that achieving such a level can produce meaningful clinical effects. [49, 50]

According to a Statement for Healthcare Professionals from the American Heart Association Diane L. Tribble, PhD for the Nutrition Committee, they conducted Dietary recommendations aimed at reducing the risk of coronary heart disease have focused largely on the intake of nutrients that affect established risk factors, including plasma lipid and lipoprotein levels, blood pressure, and body weight. Recent developments in our understanding of the atherosclerotic process and factors that trigger ischemic events have led to the consideration of dietary constituents that may alter risk through other mechanisms. Prominent among these are antioxidants, which are proposed to inhibit multiple proatherogenic and prothrombotic oxidative events in the artery wall.

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This report provides a brief overview of evidence concerning a role for dietary antioxidants in disease prevention, with emphasis on studies in human populations, and describes a number of issues that should be resolved before it would be prudent to make recommendations regarding the prophylactic use of antioxidant supplements.[51, 52, 53, 54, 55]

Stampfer et al, they conducted two particularly illustrative prospective cohort studies for antioxidants were published as companion papers in 1993. The first, by Stampfer et al, involved analyses of data from >85 000 Nurses' Health Study participants who were followed up for periods of ≤8 years. Risk of major coronary disease was lowest in women within the highest compared with those within the lowest quintile of reported vitamin E intake after adjustment for age and smoking status (relative risk, 0.66; 95% CI, 0.50 to 0.87). Lower risk was associated with levels of vitamin E intake that were achievable only by supplementation. Subsequent analyses revealed a 43% lower risk for vitamin E supplement users versus nonusers and an inverse relationship between risk and duration of supplement use. The second study, by Rimm et al, described a similar benefit for vitamin E based on data from >39 000 male participants of the Health Professionals Follow-up Study (HPFS) who were followed up for 4 years. [56, 57, 58, 59, 60]

Rimm et al also observed a lower risk of major coronary events in men reporting high versus those reporting low intakes of ß-carotene, but in subgroup analyses, this relationship was only significant in current and former smokers. These findings are consistent with several other studies that indicated an inverse association between dietary intake of ß-carotene or provitamin A carotenoids and risk of cardiovascular disease, particularly among smokers.[61, 62, 63, 64.]

Enstrom et al,[65] they conducted analyses revealed a relationship between vitamin C intake and heart diseases risk, in contrast to the results based on data from >11 000 US adults examined in the first National Health and Nutrition Examination Survey (NHANES I). Individuals reporting high intakes of vitamin C exhibited significantly lower risk of death from all causes, particularly from coronary heart disease, over a 10-year follow-up period. Among men, multivariate-adjusted relative risk was 0.75 (95% CI, 0.53 to 0.97) in individuals within the highest versus those within the lowest vitamin C intake group (50 mg/d dietary vitamin C plus regular supplements containing vitamin C versus <50 mg/d dietary vitamin C).However, Result were not adjusted for the intake of other antioxidants.

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The Indian Experiment of Infarct Survival Study [66] tested the therapeutic efficacy of antioxidants in reducing post-MI complications, many of which are proposed to result from oxidative reperfusion injury. Infarct size (as assessed from plasma levels of cardiac enzymes and ECG changes) and angina and total cardiac events (within the study period) were significantly reduced in individuals receiving antioxidants in the post-MI period.

The Multivitamins and Probucol (MVP) Study, they conducted another potential therapeutic role for antioxidants are in the reduction of restenosis after angioplasty. This role has been addressed in several recent trials. The Multivitamins and Probucol (MVP) Study tested the effects of a combination of vitamin C (1000 mg/d), vitamin E (1400 IU/d), and ß-carotene (100 mg/d); probucol (a lipid-lowering drug with antioxidant effects; 1000 mg/d); the dietary antioxidants plus probucol (in the same amounts); or placebo alone on the rate and severity of restenosis. The Probucol Angioplasty Restenosis Trial (PART) compared probucol (1000 mg/d) with placebo. In both studies, treatments were initiated 1 month before and maintained for 6 months after elective angioplasty. Relative to placebo, probucol significantly reduced restenosis. [67, 68, 69, 70]

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RESEARCH METHODOLOGY

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RESEARCH METHODOLOGY

3.1 Study Design:

Study Design is intervention (Follow up) Study.The study design has been described by the following flow chart:

Subjects		Before		Supplementation For '1' Month	After	
		Clinical Sign & Symptoms	Biochemical Test		Clinical Sign & Symptoms	Biochemical Test
Subjects Categories	Sample Size (N=150)	1. Chest Pain 2. Palpitation 3. Breathing Problem 4. Edema 5. Blood Pressure	1. Hb Level 2. Serum Homocystein Level (Folic Acid Level) 3. Serum Lipid Profile	Supplementation (Vitamin-A-ß Carotene, E, C) with Usual Diet & Medication. **Dose's:** 1)ß-carotene=6 mg 2)Vitamin C=200 mg 3)Vitamin E=50 mg	1. Chest Pain 2. Palpitation 3. Breathing Problem 4. Edema 5. Blood Pressure	1. Hb Level 2. Serum Homocystein Level (Folic Acid Level) 3. Serum Lipid Profile
• Myocardial infraction	n=50					
• Ischemic Heart Disease (IHD)	n=50					
• Hypertension	n=50					

3.2 Study Population& Area

The study Population was cardiovascular diseases patients the Department of cardiology Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka.

3.3 Study Period

The study period of the research work was from July 2010 to March 2011.

3.4 Sample size

The total numbers of subjects were 150 and calculated according to the standard method. Sample size was calculated according to following formula:

$$n = \frac{z^2 \times pqN}{e^2 (N-1) + Z^2 pq} = 124.32 \approx 125$$

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Where,

N= Total screened cardiovascular disease patients =200

n= Small sample size

z= standard normal value of 1.96, that is 95% confidence level

p= proportion of target population, which is 30.7%

q= assumed proportion, that is 1-p

e= precision of estimate as 0.05

However for convenience we have taken 150 samples

3.5 Development of the questionnaire (Key Variables)

A standard questionnaire was developed to obtain the relevant information's regarding:

Socio-economic status:

- Age
- Sex
- BMI
- Education
- Occupation
- Income
- Family type
- Family size

Clinical sing & symptoms:

- Chest pain
- Palpitation
- Breathing problem
- Edema
- Blood Pressure (BP)

Biochemical Investigation :

- Hb%
- Serum folic acid
- Serum lipid profile

Of the participants as per objective of the study.

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3.6 Data collection Tools

The following tools were used for the study:

i. Questionnaire.
ii. Blood collection tools.
iii. Laboratory facilities for assessment of biochemical parameters (Lab facilities of BSMMU, Dhaka were used).

3.7 Inclusion and Exclusion Criteria

Inclusion criteria:

i. Patients who are suffering form hypertention.
ii. Patients who are suffering form Myocardial Infraction.
iii. Patients who are suffering form Ischemic Heart Disease (IHD)
iv. Disease patient who have come for Medical Checkup for Cardiovascular patients.

Exclusion criteria:

i. Unable to provide complete data
ii. Ethical Barriers

3.8 Sampling Techniques

In this study purposively (Indoor & Outdoor patients).150 subjects were taken.It was found that 50 were Hypertensive, 50 were MI, and 50 are Ischemic Heart Disease (IHD) patients who were interviewed. Questionnaire was structured and close-ended technique used to subjects and collect potential information from the health card.

3.9 Data Analysis

All the statistical analysis and all other data processing were done by using SPSS 12.0 windows program. Comparative statistics was mainly used. Data were analyzed in terms of frequency distribution, percentage means and standard deviation.Appropriate statistical tests such as: T-test, chi-square test, Anova test, and co-relation analysis were applied and justified at 5% level of significance. Information has been presented in tabular & graphical form.

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3.10 Quality control and quality assurance

Quality control and quality assurance is very important in any study. Validity and reliability of the study has depended on it. Therefore, the following measures had been considered for this purpose:

i. To control the measurement variation of the instrument, pre test of the questionnaire has been done and modification had incorporated in the final draft.
ii. Intra-interviwer variation has removed by collecting the information herself by investigator.
iii. Interviewed questionnaire had checked daily in order to ensure its completeness, correctness and internal consistency.
iv. To minimize the biological variation, inclusion and exclusion criteria had been considered while selection of the sample.
v. Use of the statistical modeling to remove confounding bias from the findings

3.11 Ethical Consideration

Following the WHO/National Research of ethical consideration is:

i. Sensitive question tell participant
ii. Informed decision making concept
iii. You are not paying any incentive
iv. Brief, eassy language about of your research
v. I will be transform my question in bangle, because in Illiterate people

3.12 Limitations of the study

i. Study finding will be only applicable having similar situation.
ii. Finding could be association with information and selection biases but using inclusion, exclusion and stratified method of analysis biases are minimize.
iii. Selected purposively respondent

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RESULTS

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RESULTS

This Chapter showed the results of this study. All the results were presented in the tabular & graphical froms. The study was done on 150 patients of cardiovascular diseases.They were divided into three groups of which 50 were MI, 50 were Hypertention & 50 were IHD.

Distribution of the participants by Socio Demographic Informations:

Figure: 1 Age of the Respondent

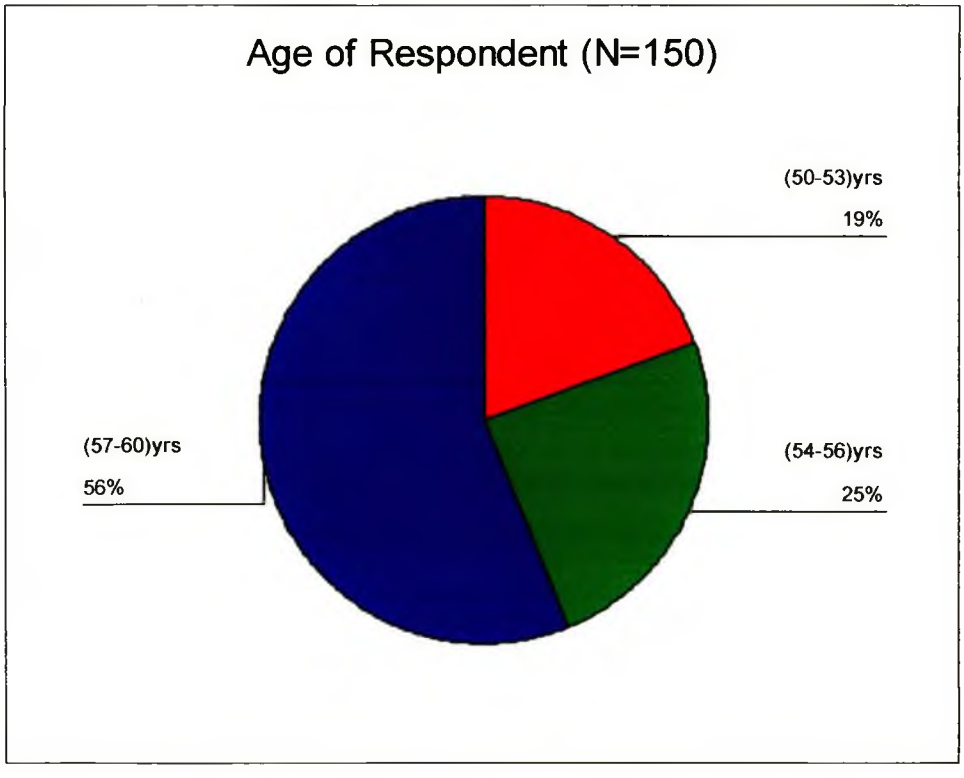

Figure-1 describes the age groups of the respondent.The largest group belonged to age (57-60) years, and the smallest age group belonged to age (50-53) years, and the rest of them were (54-56) years age group.

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Figure: 2 Sex of the Respondent

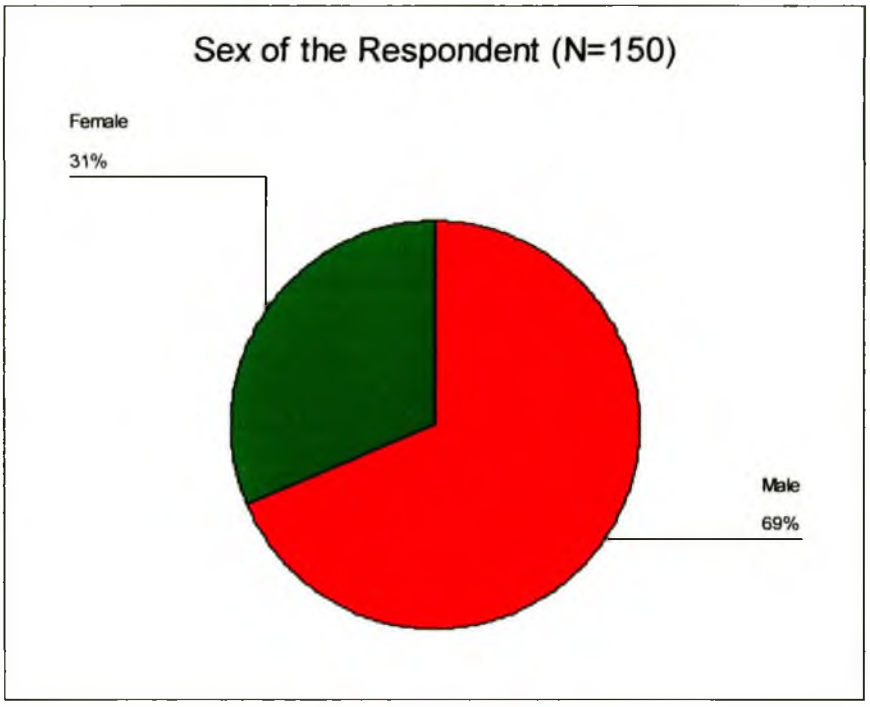
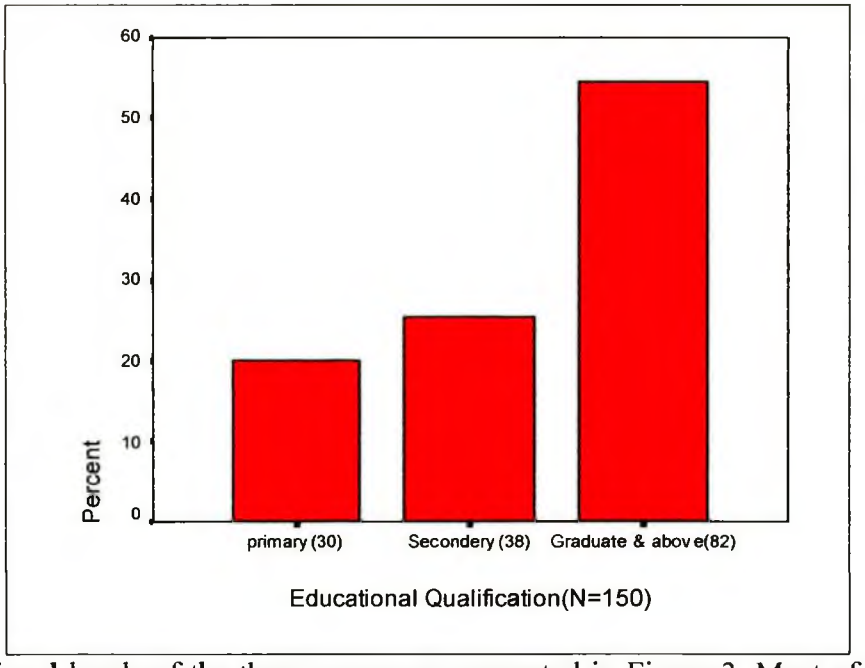


Figure 2 shows that the largest group 69 % were male and rest of them were female.

Figure: 3 Educational level of Respondent



The educational levels of the three groups are presented in Figure-3 .Most of them were in the group of graduate & above (54.7 %), seondery educational level were (25.3 %), and primary level were (20.0%).

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Figure: 4 Occupation of the Respondent

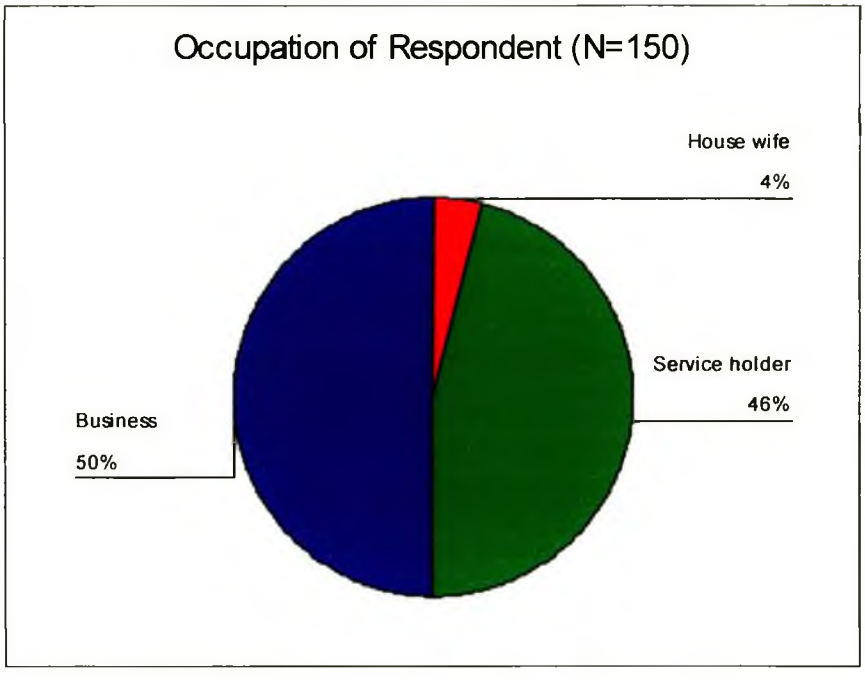

Figure 4 shows the occupation of respondent. 50.0 % business man were the largest group, followed by 46.0 % were service holder and 4.0 % were house wife.

Figure: 5 Income of the Respondent

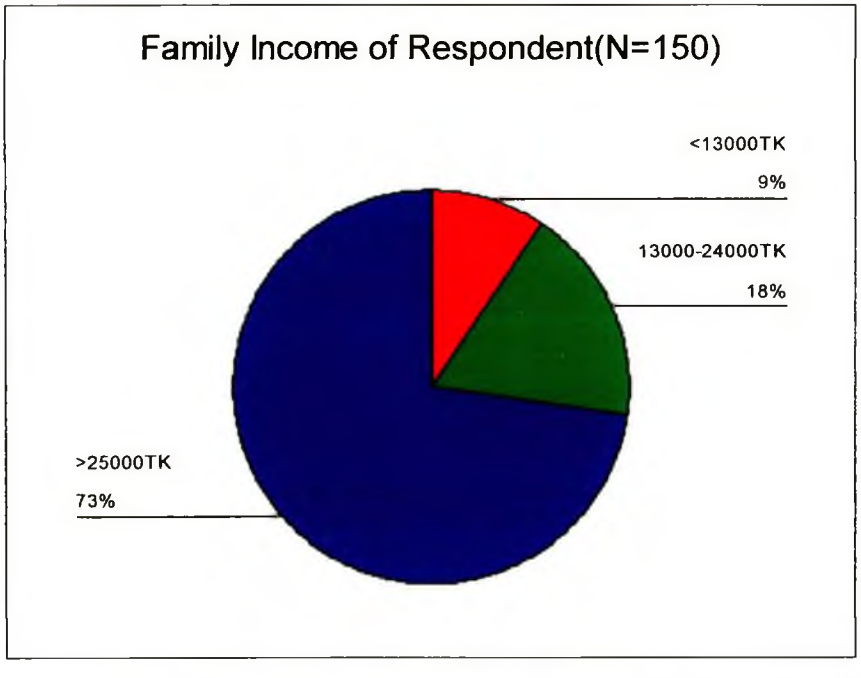

Figure 5 describes the largest group 72.7 % were family income >25000TK, 18.0 % of the participants family income between 13000-24000TK, rest of the participants family income below 13000TK

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Table 1 Distribution of sing & symptoms of participants

Sing& symptoms	Level	Frequency (n = 150)	Percentage (%)
Chest pain	Yes	126	84.0
	No	24	16.0
Palpitation	Yes	125	83.3
	No	25	16.7
Breathing Problem	Yes	112	74.7
	No	38	25.3
Edema	Yes	58	38.7
	No	92	61.3

Table 1 shows the before antioxidant treatment, the subjects were suffered from chest pain, palpitatipn, breathing problem and edema in percentage 84.0 %, 83.3 %,74.7 % and 38.7 % respectively.

Table 2 Distribution of BP of participants

BP of participants	Level	Frequency (n=150)	Percentage (%)
BP systolic	Normal(80-140mmHg)	47	31.3
	Abnormal	103	68.7
BP diastolic	Normal(60-90mmHg)	16	10.7
	Abnormal	134	89.3

Table 2 shows the sing & symptoms regarding Blood Pressure.The normal systolic pressure was 31.3 % where as the normal dystolic pressure was 10.7 % before the antioxidant treatment.

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Table 3 Distribution of Biochemical information of the participants

Biochemical Variables	Level	Frequency (n=150)	Percentage (%)
Serum Homocystein level (Folic acid level ng/ml)	Normal(2.7-16.1ng/ml)	70	46.7
	Abnormal	80	53.3
Serum Cholesterol Level(mg/dl)	Normal(120-200mg/dl)	8	5.3
Serum HDL-Cholesterol (mg/dl)	Abnormal	142	94.7
Serum HDL-Cholesterol (mg/dl)	Normal(>40mg/dl)	44	29.3
	Abnormal	106	70.7
Serum LDL-Cholesterol (mg/dl)	Normal(<130mg/dl)	13	8.7
	Abnormal	137	91.3
Serum Triglycerides (mg/dl)	Normal(<150mg/dl)	4	2.7
	Abnormal	146	97.3
Hemoglobin (g/dl)	Normal(11.5-16g/dl)	4	2.7
	Abnormal	146	97.3

Table 3 shows the biochemical information on participants. The normal level of serum homocystein, serum cholesterol, HDL-Cholesterol, Serum LDL-Cholesterol, Serum Triglyceride and serum heamoglobin level were 46.7%, 5.3%, 29.3%, 8.7%, 2.7%,2.7% respectively before the antioxidant intervention.

Table 4 Distribution of sing & symptoms to measure the effect of antioxidants on participants

Sing& symptoms		N=150		
		Before	After	*P* *value
		Frequency (%)	Frequency (%)	
Chest pain	Yes	126 (84.0)	12 (8.0)	0.000
	No	24 (16.0)	138 (92.0)	
Palpitation	Yes	125 (83.3)	23 (15.3)	0.000
	No	25 (16.7)	127 (84.7)	
Breathing Problem	Yes	112 (74.7)	19 (12.7)	0.000
	No	38 (25.3)	131 (87.3)	
Edema	Yes	58 (38.7)	31 (20.7)	0.000
	No	92(61.3)	119(79.3)	

* chi –square test

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Table 4 shows the effects antioxidant on sing & symptoms of participants after antioxidant intervention.Before antioxidants treatment chest pain was 84.0 % and after antioxidant treatment chest pain was 8.0 %, before antioxidants treatment palpitation was 83.3 % and after antioxidant treatment palpitation was 15.3 %, before breathing problem was 74.7 % and after breathing problem was 12.7 % and before edema was 38.7 % and after edema was 20.7 % and all the difference was significant (p=.000).

Table 5 Distribution of BP to measure the effect of antioxidants on participants

BP on participants	Level	N=150		
		Before	After	
		Frequency (%)	Frequency (%)	*P** value
BP systolic	Normal (80-140mmHg)	47(31.3)	143(95.3)	0.000
	Abnormal	103(68.7)	7(4.7)	
BP diastolic	Normal (60-90mmHg)	16(10.7)	142(94.7)	0.000
	Abnormal	134(89.3)	8(5.3)	

*chi –square test

This table reflected the effects of antioxidants on the Blood Pressure.Antioxidants treatment on both BP systolic and BP dystolic normal participant significantly (p=0.000) increased. On the other hand antioxidants treatment on both BP systolic and BP dystolic in abnormal participants significantly (p=0.000) decreased.

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Table 6 Distribution of Biochemical information to measure the effect of antioxidants on participants

Biochemical Variables	Level	N=150		
		Before	After	
		Frequency (%)	Frequency (%)	*P** value
Serum Homocystein level (Folic acid level ng/ml)	Normal(2.7-16.1 ng/ml)	70(46.7)	143(95.3)	0.000
	Abnormal	80(53.3)	7(4.7)	
Serum Cholesterol Level(mg/dl)	Normal(120-200 mg/dl)	8(5.3)	128(85.3)	0.000
	Abnormal	142(94.7)	22(14.7)	
Serum HDL-Cholesterol (mg/dl)	Normal(>40 mg/dl)	44(29.3)	137(91.3)	0.000
	Abnormal	106(70.7)	13(8.7)	
Serum LDL-Cholesterol (mg/dl)	Normal(<130mg/dl)	13(8.7)	143(95.3)	0.000
	Abnormal	137(91.3)	7(4.7)	
Serum Triglycerides (mg/dl)	Normal(<150 mg/dl)	4(2.7)	134(89.3)	0.000
	Abnormal	146(97.3)	16(10.7)	
Hemoglobin (g/dl)	Normal(11.5-16g/dl)	4(2.7)	146(97.3)	0.000
	Abnormal	146(97.3)	4(2.7)	

*chi –square test

Above the table shows the effects of antioxidants on biochemical measurements.Before antioxidants treatment normal homocystein level were 46.7%, normal serum cholesterol 5.3 %, normal Serum HDL-Cholesterol 29.3 % , normal Serum LDL-Cholesterol 8.7 % and normal Serum Triglyceride 2.7 %,which were significantly(p=0.000) increased after antioxidant treatment.

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Table 7 Achievement of antioxidants effects on the participant by biochemical test

Variables	N=150						*P*-value*
	Before		After		Difference of 95% confidence interval		
	Mean	Std. Deviation	Mean	Std. Deviation	Lower	Upper	
BP systolic	146.9	39.8	99.7	25.4	40.6	53.8	0.000
BP diastolic	120.3	22.8	90.2	6.4	25.8	34.2	0.000
Hemoglobin (g/dl)	12.8	1.25	13.4	1.18	.884	.696	0.000
Serum folate(ng/dl)	6.5	5.5	7.10	4.7	.801	.337	0.000
Serum cholesterol (mg/dl)	296.5	98.8	189.4	67.3	94.6	119.6	0.000
Serum HDL-chol. (mg/dl)	37.3	12.3	53.3	12.2	18.6	13.4	0.000
Serum LDL-chol. (mg/dl)	146.9	22.3	114.8	15.1	28.2	36.1	0.000
Serum TG μ (mg/dl)	291.8	128.5	150.6	61	120.6	161.9	0.000

*T-test (Paired)

Table 7 shows achievement of the antioxidant on the biochemical analysis of participants.Before antioxidants intervention the mean&std (146±39) of BP systolic and after antioxidants intervention the mean&std (99±25) of BP systolic were significantly(p=0.000) reduced. Whereas before antioxidants intervention the mean&std (120±22) of BP diastolic and after antioxidants intervention the mean&std (90±6) of BP diastolic were significantly (p=0.000) reduced. In the same way, before intervention program serum heamoglobin mean&std (12±1), serum folate mean&std (6±5) and serum HDL mean&std (37±12) were significantly (p=0.000) increased after intervention program.whereas, before intervention program serum cholesterol mean&std (37±12), serum LDL mean&std (146±22) and serum TG mean&std (291±128) were significantly (p=0.000) reduced after intervention program.

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Table 8 Antioxidant effects on sign & symptoms of Hypertensive, Mycardial Infraction, and Ischemic Heart diseasePatients.

Diagnosis									
Sing& symptoms of participants	**Level**	**Hypertention (n=50)**		**MI (n=50)**		**IHD (n=50)**		**Total (N=150)**	
		Before (%)	After (%)	Before (%)	After (%)	Before (%)	After (%)	Before (%)	After (%)
Chest pain	Yes	82.0	6.0	76.0	8.0	94.0	10.0	84.0	8.0
	No	18.0	94.0	24.0	92.0	6.0	90.0	16.0	92.0
Palpitation	Yes	80.0	12.0	82.0	24.0	88.0	10.0	83.3	15.3
	No	20.0	88.0	18.0	76.0	12.0	90.0	16.7	84.7
Breathing Problem	Yes	68.0	6.0	78.0	24.0	78.0	8.0	74.7	12.7
	No	32.0	94.0	22.0	76.0	22.0	92.0	25.3	87.3
Edema	Yes	26.0	8.0	36.0	28.0	54.0	26.0	38.7	20.7
	No	74.0	92.0	64.0	72.0	46.0	74.0	61.3	79.3

Crosstabs

The table 8 describes the antioxidants effects on sing & symptoms in hypertensive, MI, and IHD patients.It showed that antioxidant lowering effect was much higher of chest pain on IHD(10.0%) followed by MI (8.0%) and hypertention (6.0%).In relation to palpitation, showed that antioxidant effects was less on IHD (10.0%), followed by hyperention (12.0%), and MI (24.0%) patients.In the same way we can describe the antioxidant effects on breathing problem and edema in hypertensive, MI, and IHD patients.

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Table 9 Antioxidant effects on BP of Hypertensive, Mycardial Infraction, and Ischemic Heart diseases Patients.

Diagnosis									
BP of participants	**Level**	**Hypertention (n=50)**		**MI (n=50)**		**IHD (n=50)**		**Total (N=150)**	
		Before (%)	**After (%)**	**Before (%)**	**After (%)**	**Before (%)**	**After (%)**	**Before (%)**	**After (%)**
BP systolic	Normal(80-140mmHg)	-	100.0	40.0	92.0	54.0	94.0	31.0	95.3
	Abnormal	100.0	-	60.0	8.0	46.0	6.0	68.7	4.7
BP diastolic	Normal(60-90mmHg)	-	100.0	12.0	90.0	20.0	94.0	10.7	94.7
	Abnormal	100.0	-	88.0	10.0	80.0	6.0	89.3	5.3

Crosstabs

The table 9 describes the antioxidants effects on BP in hypertensive, MI, and IHD patients.It showed that antioxidant lowering effect was much higher of BP systolic on hypertensive (100.0%) patients, followed by IHD (94.0%) and MI (94.0%) patients.In relation to BP diastolic, showed that the antioxidant effects BP diastolic was become normal level on hypertention (100.0%), followed by IHD (94.0%), and MI (90.0%) patients.

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Table 10 Antioxidant effects on Biochemical Investigations of Hypertensive, Mycardial Infraction, and Ischemic Heart diseases Patients.

Diagnosis									
Biochemical Variables	**Level**	**Hypertention (n=50)**		**MI (n=50)**		**IHD (n=50)**		**Total (N=150)**	
		Before (%)	After (%)	Before (%)	After (%)	Before (%)	After (%)	Before (%)	After (%)
Serum Homocystein level (ng/ml)	Normal(2.7-16.1ng/ml)	72.0	100.0	26.0	92.0	42.0	94.0	46.7	95.3
	Abnormal	28.0	-	74.0	8.0	58.0	6.0	53.3	4.7
Serum Cholesterol Level(mg/dl)	Normal(120-200mg/dl)	4.0	88.0	8.0	80.0	4.0	88.0	5.3	85.3
	Abnormal	96.0	12.0	92.0	20.0	96.0	12.0	94.7	14.7
Serum HDL-Cholesterol (mg/dl)	Normal(>40 mg/dl)	14.0	98.0	30.0	94.0	44.0	82.0	29.3	91.3
	Abnormal	86.0	2.0	70.0	6.0	56.0	18.0	70.7	8.7
Serum LDL-Cholesterol (mg/dl)	Normal(<130mg/dl)	4.0	98.0	16.0	96.0	6.0	92.0	8.7	95.3
	Abnormal	96.0	2.0	84.0	4.0	94.0	8.0	91.3	4.7
Serum Triglycerides (mg/dl)	Normal(<150mg/dl)	-	86.0	4.0	88.0	4.0	94.0	2.7	89.3
	Abnormal	100.0	14.0	96.0	12.0	96.0	6.0	97.3	10.7
Hemoglobin (g/dl)	Normal(11.5-16g/dl)	59.0	92.0	69.0	95.0	57.0	91.0	40.0	89.0
	Abnormal	41.0	8.0	31.0	5.0	43.0	9.0	60.0	11.0

Crosstabs

The table 10 describes the antioxidants effects on biochemical investigation in hypertensive, MI, and IHD patients.It showed that antioxidant lowering effect was much higher of serum homocystein on hypertention (100.0%) followed by IHD (94.0%) and MI (92.0%).In terms of serum cholesterol, showed that antioxidant effects was less on MI (80.0%), followed by hyperention (88.0%), and MI (88.0%) patients.and It showed that antioxidant lowering effect was much higher of serum HDL-cholesterol on hypertention (98.0%) followed by MI (94.0%) and IHD (82.0%). In relation to serum cholesterol,-LDL showed that antioxidant effects was less on IHD (92.0%), followed by MI (96.0%), and hypertention (98.0%) patients In the same way we can describe the antioxidant effects of serum TG and hemoglobin level in hypertensive, MI, and IHD patients.

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Table 11 Achievement of antioxidants effects on the participant by biochemical test

Biochemical Variables	Diagnosis								
	Hypertention(n=50)			MI(n=50)			IHD(n=50)		
	Before Mean± Std	After Mean± Std	P-value	Before Mean± Std	After Mean± Std	P-value	Before Mean±Std	After Mean± Std	P-value
BP systolic	183±11	91±5	0.000	130±38	95±26	.000	127±35	112±31	0.000
BP diastolic	144±19	85±6	0.000	107±11	91±5	.000	108±13	93±4	0.000
Hemoglobin (g/dl)	12±1	13±1	0.000	13±1	14±1	.000	12±1	13±1	0.000
Serum homocystein (ng/dl)	8±5	8±4	0.001	5±5	6±4	.114	6±5	7±4	0.001
Serum cholesterol (mg/dl)	287±58	182±32	0.000	286±111	198±87	.000	315±115	188±70	0.000
Serum HDL-cholesterol (mg/dl)	34±5	55±2	0.000	36±11	51±10	.000	40±16	53±13	0.000
Serum LDL-cholesterol (mg/dl)	149±15	119±11	0.000	144±26	116±17	.000	147±24	107±13	0.000
Serum TG (mg/dl)	303±115	147±50	0.000	287±146	163±79	.000	285±122	141±45	0.000

*T-test (Paired)

Table 11 shows the achievement of antioxidants effects on hypertension, MI and IHD by biochemical test.It showed that mean±std of BP systolic were significantly (p=.000) reduced on hypertention after intervention program followed by MI and IHD patients.In terms of BPdiastolic mean±std were significantly (P=.000) reduced on hypertention after intervention program followed by MI and IHD patients .In relation to hemoglobin, serum homocystein, serum HDL mean±std were significantly (P=.000) increased on MI after intervention program followed by hypertention and IHD patients, whereas serum cholesterol, serumLDL, and serum TG, mean±std were significantly (P=.000) reduced on after intervention program IHD followed by MI and Hypertention patients.

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Table 12 Achievement of antioxidants effects on the participant by Clinical sign & symptoms

Clinical Sing& symptoms	Diagnosis								
	Hypertention			MI			IHD		
	Before Mean± Std	After Mean± Std	P-value	Before Mean ±Std	After Mean± Std	P-value	Before Mean± Std	After Mean± Std	P-value
Chest pain	1±.38	1±.24	.000	1±.43	1±.27	.000	1±.24	1±.30	.000
Palpitation	1±.40	1±.32	.000	1±.38	1±.43	.000	1±.32	1±.30	.000
Breathing Problem	1±.47	1±.24	.000	1±.41	1±.43	.000	1±.41	1±.27	.000
Edema	1±.44	1±.27	.002	1±.48	1±.45	.209	1±.50	1±.44	.000

*T-test (Paired)

Table 12 shows the achievement of antioxidants effects on hypertension, MI and IHD by clinical sign & symptoms.It showed that before intervention program mean±std of chest pain, palpitation, breathing problem and edema were significantly (p=.000) reduced on hypertentive patients after intervention program.In terms of MI mean±std of chest pain, palpitation, breathing problem and edema were significantly (P=.000) reduced.In relation to IHD mean±std of chest pain, palpitation, breathing problem and edema were significantly (p=.000) reduce.

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Table 13 Distribution of Biochemical Investigation and BP to measure the effect of antioxidants by Sex

Biochemical Variables	Level	Sex (N=150)					
		Before			After		
		Male (%)	Female (%)	*p*-value**	Male (%)	Female (%)	*p*-value**
Serum chol (total)	Normal (120-200) mg/dl	4.9(5)	6.4(3)	.699	83.5(86)	89.4(42)	.346
	Abnormal	95.1(98)	93.6(44)		16.5(17)	10.6(5)	
Serum HDL - chol	Normal(>40) mg/dl	35.0(36)	17.0(8)	.025	92.2(95)	89.4(42)	.562
	Abnormal	65.0(67)	83.0(39)		7.8(8)	10.6(5)	
Serum LDL-chol	Normal(<130) mg/dl	9.7(10)	6.4(3)	.502	94.2(97)	97.6(46)	.319
	Abnormal	90.3(93)	93.6(44)		5.8(6)	2.1(1)	
Serum TG	Normal(<150) mg/dl	1.9(2)	4.3(2)	.415	91.3(94)	85.1(40)	.257
	Abnormal	98.1(101)	95.7(45)		8.7(9)	14.9(7)	
BP systolic	Normal (80-140) mmHg	40.8(42)	10.6(5)	.000	93.2(96)	100.0(47)	.067
	Abnormal	59.2(61)	89.4(42)		6.8(7)		
BP diastolic	Normal (60-90) mmHg	15.5(16)	_	.004	92.2(95)	100.0(47)	.050
	Abnormal	84.5(87)	100.0(47)		7.8(8)		
Serum Homocystein	Normal (2.7-16.1) ng/ml	30.1(31)	83.0(39)	.000	94.2(97)	97.9(46)	.319
	Abnormal	69.9(72)	17.0(8)		5.8(6)	2.1(1)	
Heamoglobin	Normal (11.5-16)g/dl	28.1(29)	83.0(39)	.000	94.2(97)	97.9(46)	.000
	Abnormal	71.8(74)	17.0(8)		5.8(6)	2.1(1)	

**chi –square test (Pearson)

Table 13 shows the relationship between biochemical parameters and BP by sex. Frequency of after normal serum cholesterol of male-female were higher than the before normal serum cholesterol (P=0.346).Percentage of the after normal serum HDL of male-female were higher than the before normal serum HDL (P=0.562). Frequency of after normal serum LDL of male-female were higher than the before normal serum LDL (P=0.319).Percentage of the after normal serum TG of male-female were higher than the before normal serum TG (P=0.257). Frequency of after normal BP systolic of male-female were higher than the before normal BP systolic (P=0.067).Percentage of the after

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normal BP diastolic of male-female were higher than the before normal BP diastolic (P=0.050). Frequency of after normal serum homocystein of male-female were higher than the before normal serum homocystein (P=0.319). Frequency of after normal heamoglobin level of male-female were higher than the before normal heamoglobin level (P=0.000).

Table 14 Distribution of Biochemical Investigation and BP to measure the effect of antioxidants by Education

Biochemical Variables	Level	Education (N=150)							
		Before				After			
		Primary	Secondary	Graduate and above	*p*-value**	Primary	Secondary	Graduate and above	*p*-value**
		(%)	(%)	(%)		(%)	(%)	(%)	
Serum chol (total)	Normal (120-200) mg/dl	6.7(2)	2.6(1)	6.1(5)	.687	90.0(27)	84.2(32)	84.1(69)	.722
	Abnormal	93.3(28)	97.4(37)	93.9(77)		10.0(3)	15.8(6)	15.9(13)	
Serum HDL-chol	Normal (>40)mg/dl	53.3(16)	31.6(12)	19.5(16)	.002	76.7(23)	94.7(36)	95.1(78)	.006
	Abnormal	46.7(14)	68.4(26)	80.5(66)		23.3(7)	5.3(2)	4.9(4)	
Serum LDL-chol	Normal (<130)mg/dl	6.7(2)	2.6(1)	12.2(10)	.203	90.0(27)	100.0(38)	95.1(78)	.151
	Abnormal	93.3(28)	97.4(37)	87.8(72)		10.0(3)		4.9(4)	
Serum TG	Normal (<150)mg/dl	–	5.3(2)	2.4(2)	.401	93.3(28)	84.2(32)	90.2(74)	.444
	Abnormal	100(30)	94.7(36)	97.6(80)		6.7(2)	15.8(6)	9.8(8)	
BP systolic	Normal(80-140mmHg)	66.7(20)	10.5(4)	28.0(23)	.000	100.0(30)	92.1(35)	95.1(78)	.306
	Abnormal	33.3(10)	89.5(34)	72.0(59)			7.9(3)	4.9(4)	
BP diastolic	Normal(60-90mmHg)	13.3(4)	10.5(4)	9.8(8)	.862	96.7(29)	97.4(37)	92.7(76)	.490
	Abnormal	86.7(26)	89.5(34)	90.2(74)		3.3(1)	2.6(1)	7.3(6)	
Serum Homocystein	Normal(2.7-16.1 ng/ml)	50.0(15)	52.6(20)	42.7(35)	.549	93.3(28)	97.4(37)	95.1(78)	.729
	Abnormal	50.0(15)	47.4(18)	57.3(47)		6.7(2)	2.6(1)	4.9(4)	
Heamoglobin	Normal(11.5-16 g/dl)	40.0(12)	52.6(20)	42.7(35)	.549	93.3(28)	97.4(37)	92.6(76)	.725
	Abnormal	60.0(18)	47.4(18)	57.3(47)		6.7(2)	2.6(1)	7.4(6)	

**chi-square test (pearson)

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Table 14 shows the relationship between biochemical investigation and BP by education. Frequency of after normal serum cholesterol of participant education were higher than the before normal serum cholesterol (P=0.722).Percentage of the after normal serum HDL of different education were higher than the before normal serum HDL (P=0.006). Frequency of after normal serum LDL of participant education were higher than the before normal serum LDL (P=0.151).Percentage of the after normal serum TG of participant education were higher than the before normal serum TG (P=0.444). Frequency of after normal BP systolic of participant education were higher than the before normal BP systolic (P=0.306).Percentage of the after normal BP diastolic of different education were higher than the before normal BP diastolic (P=0.490). Frequency of after normal serum homocystein of different education were higher than the before normal serum homocystein (P=0.729), whereas frequency of after normal serum heamoglobin level of different education were higher than the before normal serum heamoglobin level (P=0.725).

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Table 15 Distribution of Biochemical Investigation and BP to measure the effect of antioxidants by Occupation

Biochemical Variables	Level	Occupation (N=150)							
		Before				After			
		House wife	Service holder	Business	*p*-value**	Housewife	Service holder	Business	*p*-value**
		(%)	(%)	(%)		(%)	(%)	(%)	
Serum chol (total)	Normal (120-200)mg/dl	33.3(2)	5.8(4)	2.7(2)	.068	66.7(4)	85.5(59)	86.7(65)	.036
	Abnormal	66.7(4)	94.2(65)	97.3(73)		33.3(2)	14.5(10)	13.3(10)	
Serum HDL-chol	Normal (>40)mg/dl	-	33.3(23)	28.0(21)	.486	93.3(5)	85.5(59)	97.3(73)	.118
	Abnormal	100.0(6)	66.4(46)	72.0(54)		16.7(1)	14.5(10)	2.7(2)	
Serum LDL-chol	Normal (<130)mg/dl	16.7(1)	11.6(8)	5.3(4)	.331	100.0(6)	94.2(65)	96.0(72)	.404
	Abnormal	83.3(5)	88.4(61)	94.7(71)		-	5.8(4)	4.0(3)	
Serum TG	Normal (<150)mg/dl	-	4.3(30	1.3(1)	.525	83.3(5)	94.2(65)	85.3(64)	.047
	Abnormal	100.0(6)	95.7(66)	98.7(74)		16.7(1)	5.8(4)	14.7(11)	
BP systolic	Normal(80-140 mmHg)	66.7(4)	39.1(27)	21.3(16)	.012	100.0(6)	95.7(66)	94.7(71)	.825
	Abnormal	33.3(2)	60.9(42)	78.7(59)		-	4.3(3)	5.3(4)	
BP diastolic	Normal(60-90 mmHg)	-	5.8(4)	16.0(12)	.097	100.0(6)	91.3(63)	97.3(73)	.230
	Abnormal	100.0(6)	94.2(65)	84.0(63)		-	8.7(6)	2.7(2)	
Serum Homocystein	Normal(2.7-16.1 ng/ml)	-	47.8(33)	49.3(37)	.064	83.3(5)	94.2(65)	97.3(73)	.245
	Abnormal	100.0(6)	52.2(36)	50.7(38)		16.7(1)	5.8(4)	2.7(2)	
Heamoglobin	Normal(11.5-116 g/dl)	16.6(1)	5.8(4)	2.7(2)	.062	66.7(4)	85.5(59)	86.7(65)	.248
	Abnormal	83.3(5)	94.2(65)	97.3(73)		33.3(2)	14.5(10)	13.3(10)	

**chi-square test (Pearson)

Table 15 shows the relationship between biochemical investigation and BP by occupation. Frequency of after normal serum cholesterol of different occupation were higher than the before normal serum cholesterol (P=0.036).Percentage of the after normal serum HDL of different occupation were higher than the before normal serum HDL (P=0.118). Frequency of after normal serum LDL of different occupation were higher than the before normal serum LDL (P=0.404).Percentage of the after normal serum TG of

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different occupation were higher than the before normal serum TG (P=0.047). Frequency of after normal BP systolic of participant ocupation were higher than the before normal BP systolic (P=0.825).Percentage of the after normal BP diastolic of participant ocupation were higher than the before normal BP diastolic (P=0.230). Frequency of after normal serum homocystein of participant ocupation were higher than the before normal serum homocystein (P=0.245), while frequency of after normal serum heamoglobin (%) of participant ocupation were higher than the before normal serum heamoglobin (%) (P=0.248).

Table 16 Distribution of clinical sign & symptoms to measure the effect of antioxidants by Sex

Clinical Variables		Sex (N=150)					
		Before			After		
		Male (%)	Female (%)	*p*-value**	Male (%)	Female (%)	*p*-value**
Chest pain	Yes	82.5(85)	87.2(41)	.465	99.7(10)	4.3(2)	.253
	No	17.5(18)	12.8(6)		90.3(93)	95.7(45)	
Palpitation	Yes	85.4(88)	78.7(37)	.306	16.5(17)	12.8(6)	.558
	No	14.6(15)	21.3(10)		83.5(86)	87.2(41)	
Breathing problem	Yes	78.6(81)	66.0(31)	.098	16.5(17)	4.3(2)	.036
	No	21.4(22)	34.0(16)		83.5(86)	95.7(45)	
Edema	Yes	44.7(46)	25.5(12)	.026	27.2(28)	6.4(3)	.004
	No	55.3(57)	74.5(35)		72.8(75)	93.6(44)	

****chi –square test (Pearson)**

Table 16 shows the relationship between clinical sing-symptoms by sex. Frequency of before chest pain of male-female were higher than the after chest pain (P=0.465, 0.253).Percentage of the before palpitation of male-female were higher than the after palpitation (0.306, 0.558). Frequency of before breathing problem of male-female were higher than the after breathing problem (P=0.098, 0.036). Frequency of before edema of male-female were higher than the after edema (P=0.026, 0.004).

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Table 17 Distribution of clinical sign & symptoms to measure the effect of antioxidants by Education

Clinical Variables		Education (N=150)							
		Before				After			
		Primary	Secondary	Graduate and above	*p*-value**	Primary	Secondary	Graduate and above	*p*-value**
		(%)	(%)	(%)		(%)	(%)	(%)	
Chest pain	Yes	93.3(28)	89.5(34)	78.0(64)	.084	6.7(2)	10.5(4)	7.3(6)	.797
	No	6.7(2)	10.5(4)	22.0(18)		93.3(28)	89.5(34)	92.7(76)	
Palpitation	Yes	86.7(26)	81.6(31)	82.9(68)	.846	13.3(4)	5.3(2)	20.7(17)	.086
	No	13.3(4)	18.4(7)	17.1(140		86.7(26)	94.7(36)	79.3(65)	
Breathing problem	Yes	83.3(25)	73.7(28)	72.0(59)	.465	3.3(1)	10.5(4)	17.1(14)	.138
	No	16.7(5)	26.3(10)	28.0(23)		96.7(29)	89.5(34)	82.9(68)	
Edema	Yes	53.3(16)	44.7(17)	30.5(25)	.060	23.3(7)	21.1(8)	19.5(16)	.905
	No	46.7(14)	55.3(21)	69.5(570		76.7(23)	78.9(30)	80.5(66)	

****chi –square test (Pearson)**

Table 17 shows the relationship between clinical sing& symptoms by education. Frequency of before chest pain education of primary, secondary, Graduate & above were higher than the after chest pain (P=0.084, 0.797).Percentage of the before palpitation education of primary, secondary, Graduate & above were higher than the after palpitation (0.846, 0.086). Frequency of before breathing problem education of primary, secondary, Graduate & above of male-female were higher than the after breathing problem (P=0.465, 0.138). Frequency of before edema education of primary, secondary, Graduate & above were higher than the after edema (P=0.060, 0.905).

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Table 18 Distribution of clinical sign & symptoms to measure the effect of antioxidants by Occupation

Clinical Variables		Occupation (N=150)							
		Before				After			
		Housewife	Service holder	Business	*p*-value**	Housewife	Service holder	Business	*p*-value**
		(%)	(%)	(%)		(%)	(%)	(%)	
Chestpain	Yes	50.0(3)	85.5(59)	85.3(64)	.068	33.3(2)	4.3(3)	9.3(7)	.036
	No	50.0(3)	14.5(10)	14.7(11)		66.7(4)	95.7(66)	90.7(68)	
Palpitation	Yes	100.0(6)	84.1(58)	81.3(61)	.486	16.7(1)	21.7(15)	9.3(70	.118
	No	-	15.9(11)	18.7(14)		83.3(5)	78.3(54)	90.7(68)	
Breathing problem	Yes	100.0(6)	72.5(50)	74.7(56)	.331	-	15.9(11)	10.7(8)	.404
	No	-	27.5(19)	25.3(19)		100.0(6)	84.1(58)	89.3(67)	
Edema	Yes	16.7(1)	39.1(27)	40.0(30)	.525	-	29.0(20)	14.7(11)	.047
	No	83.3(5)	60.9(42)	60.0(45)		100.0(6)	71.0(49)	85.3(64)	

**chi –square test (Pearson)

Table 18 shows the relationship between clinical sing& symptoms by occupation. Frequency of before chest pain, occupation of housewife, service holder, business were higher than the after chest pain (P=0.063, 0.036).Percentage of the before palpitation occupation of housewife, service holder, business were higher than the after palpitation (0.486, 0.118). Frequency of before breathing problem occupation of housewife, service holder, business were higher than the after breathing problem (P=0.331, 0.404). Frequency of before edema occupation of housewife, service holder, business were higher than the after edema (P=0.525, 0.047).

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Table 19 Relationship/ Association between respondent education and clinical sign & symptoms

Clinical Variables	Education(N=150)			*P* value**
	Primary	Secondery	Graduate & above	
	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)	
Chest pain (before)	1.07(.97-1.16)	1.11(1.00-1.21)	1.22(1.13-1.31)	.085
Chest pain (after)	1.93(1.84-2.03)	1.89(1.79-2)	1.93(1.87-1.98)	.800
Palpitation (before)	1.13(1-1.26)	1.18(1.06-1.31)	1.17(1.09-1.25)	.849
Palpitation (after)	1.87(1.74-2)	1.95(1.87-2.02)	1.79(1.70-1.88)	.087
Brething problem(before)	1.17(1.03-1.31)	1.26(1.12-1.41)	1.28(1.18-1.38)	.471
Brething problem(after)	1.97(1.90-2.03)	1.89(1.79-2)	1.83(1.75-1.91)	.140
Edema(before)	1.47(1.28-1.66)	1.55(1.39-1.72)	1.70(1.59-1.80)	.060
Edema(after)	1.77(1.61-1.93)	1.79(1.65-1.93)	1.80(1.72-1.89)	.906
BP diastolic (before)	111.2(105-116)	125(118-133)	121(115-126)	.000
BP diastolic (after)	94(93.10-94.90)	88(85.76-90.5)	89.8(91.1-101)	.123
BP systolic (before)	117(104.-130)	172(162-181)	146(137-154)	.029
BP systolic (after)	101(90-111)	106(97-115)	96(91.14-101)	.001

** Anova

Table 19 gives the mean of before –after **chest pain** by the primary, secondary, graduate&above education of participant.Mean of chest pain by the education of participant P-value were (P=.085, P=.800).Mean of chest pain (before-after) by the primary education were (1.07-1.93),whereas Mean of chest pain (before-after) by the secondary education were (1.1-1.89) and Mean of chest pain (before-after) by the graduate education were (1.22-1.93).

Table 19 shows the mean of before –after **palpitation** by the primary, secondary, graduate&above education of participant.Mean of palpitation by the education of participant P-value were (P=.849, P=.087).Mean of palpitation (before-after) by the primary education were (1.13-1.87), whereas Mean palpitation of (before-after) by the

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secondary education were (1.18-1.95) and Mean of palpitation (before-after) by the graduate education were (1.17-1.79).

Table 19 describes the mean of before –after **breathing problem** by the primary, secondary, graduate&above education of participant.Mean of breathing problem by the education of participant P-value were (P=.471, P=.140).Mean breathing problem of (before-after) by the primary education were (1.17-1.97),whereas Mean of breathing problem (before-after) by the secondary education were (1.26-1.89) and Mean of breathing problem (before-after) by the graduate education were (1.28-1.83).

Table 19 shows the mean of before –after **edema** by the primary, secondary, graduate&above education of participant.Mean of edema by the education of participant P-value were (P=.060, P=.906).Mean of edema (before-after) by the primary education were (1.47-1.77), whereas Mean of edema (before-after) by the secondary education were (1.55-1.79) and Mean of edema (before-after) by the graduate education were (1.70-1.80).

Table 19 gives the mean of before –after **BP-diastolic** by the primary, secondary, graduate&above education of participant.Mean of BP-diastolic by the education of participant P-value were (P=.000, P=.123).Mean BP-diastolic of (before-after) by the primary education were(111-94), whereas Mean of BP-diastolic (before-after) by the secondary education were (125-88) and Mean of BP-diastolic (before-after) by the graduate education were (121-89).

Table 19 shows the mean of before –after **BP-systolic** by the primary, secondary, graduate&above education of participant.Mean of BP-systolic by the education of participant P-value were (P=.029, P=..001).Mean BP-systolic of (before-after) by the primary education were(117-101), whereas Mean of BP-systolic (before-after) by the secondary education were (172-106) and Mean of BP-systolic (before-after) by the graduate education were (146-96).s

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Table 20 Relationship/ Association between respondent Occupation and Clinical sign & Symptoms

Clinical Variables	Occupation (N=150)			P value**
	House wife	Service holder	Business	
	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)	
Chest pain (before)	1.50(.93-2.07)	1.14(1.06-1.23)	1.15(1.06-1.23)	.068
Chest pain (after)	1.67(1.12-2.21)	1.96(1.91-2.01)	1.91(1.84-1.97)	.492
Palpitation (before)	1(1-1)	1.16(1.07-1.25)	1.19(1.10-1.28)	.335
Palpitation (after)	1.83(1.40-2.21)	1.78(1.68-1.88)	1.91(1.84-1.97)	.531
Brething problem(before)	1(1-1)	1.28(1.17-1.38)	1.25(1.15-1.35)	.035
Brething problem(after)	2(2-2)	1.84(1.75-1.93)	1.89(1.82-1.96)	.120
Edema(before)	1.83(1.40-2.26)	1.61(1.49-1.73)	1.60(1.49-1.71)	.410
Edema(after)	2(2-2)	1.71(1.60-1.82)	1.85(1.77-1.94)	.047
BP diastolic (before)	115(100-129.47)	117(112.50-122)	123(117.63-129)	.001
BP diastolic (after)	84.17(79-89)	91.97(90-93)	89.15(87-90)	.230
BP systolic (before)	117.50(71-163)	137(127-147)	158(149-166.45)	.017
BP systolic (after)	96.67(76-117)	93.62(88-99)	105(99.39-111.81)	.002

** Anova

Table 20 describes the mean of before –after **chest pain** by the house wife, service holder, business occupation of participant.Mean of chest pain by the occupation of participant P-value were (P=.068, P=.492).Mean of chest pain (before-after) by the house wife occupation were (1.50-1.67),whereas Mean of chest pain (before-after) by the serviceholder were (1.14-1.96) and Mean of chest pain (before-after) by the business were (1.15-1.91).

Table 20 gives the mean of before –after **palpitation** by the house wife, service holder, business occupation of participant.Mean of palpitation by the occupation of participant P-value were (P=.335, P=.531).Mean of palpitation (before-after) by the house wife occupation were (1-1.83), whereas Mean palpitation of (before-after) by the service

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holder occupation were (1.16-1.78) and Mean of palpitation (before-after) by the business occupation were (1.19-1.91).

Table 20 describes the mean of before –after **breathing problem** by the house wife, service holder, business occupation of participant.Mean of breathing problem by the occupation of participant P-value were (P=.035, P=.120).Mean breathing problem of (before-after) by the house wife occupation were (1-2),whereas Mean of breathing problem (before-after) by the service holder occupation were (1.28-1.84) and Mean of breathing problem (before-after) by the business occupation were (1.25-1.89).

Table 20 gives the mean of before –after **edema** by the house wife, service holder, business occupation of participant.Mean of edema by the occupation of participant P-value were (P=.410, P=.047).Mean of edema (before-after) by the house wife occupation were (1.83-2), whereas Mean of edema (before-after) by the service holder were (1.61-1.71) and Mean of edema (before-after) by the business occupation were (1.60-1.85).

Table 20 shows the mean of before –after **BP-diastolic** by the house wife, service holder, business occupation of participant.Mean of BP-diastolic by the occupation of participant P-value were (P=.001, P=.230).Mean BP-diastolic of (before-after) by the house wife occupation were(115-84), whereas Mean of BP-diastolic (before-after) by the service holder ocupation were (117-91) and Mean of BP-diastolic (before-after) by the business occupation were (89-158).

Table 20 gives the mean of before –after **BP-systolic** by the house wife, service holder, business occupation of participant.Mean of BP-systolic by the occupation of participant P-value were (P=.017, P=.002).Mean BP-systolic of (before-after) by the house wife occupation were(117-96), whereas Mean of BP-systolic (before-after) by the service holder occupation were (137-93) and Mean of BP-systolic (before-after) by the business occupation were (158-105).

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Table 21 Relationship /Association between respondent Education and Biochemical Investigation

Biochemical Variables	Education (N=150)			P-value**
	Primary	Secondery	Graduate & above	
	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)	
Hemoglobin (before)	12.7(12.2-13.2)	13.2(12.9-13.5)	12.7(12.4-12.9)	.072
Hemoglobin (after)	13.6(13.1-14.1)	14.1(13.8-14.4)	13.4(13.1-13.6)	.005
Serum Homocystein (before)	6.5(4.4-8.6)	5.81(4.29-7.32)	6.86(5.55-8.17)	.631
Serum Homocystein (after)	7.15(5.31-8.98)	6.55(5.18-7.92)	7.34(6.23-8.45)	.703
Serum cholesterol (before)	303.5(272-336)	295(274.4-317)	294(269.-320)	.911
Serum cholesterol (after)	178.2(152-204)	190(173-205.2)	193.4(177-210)	.575
Serum HDL (before)	43.6(37.6-49.7)	37.7(34.1-41.3)	34.7(32.4-37)	.003
Serum HDL (after)	51.5(45.9-57)	56.9(52.9-60.9)	52.3(49.9-54.8)	.111
Serum LDL (before)	147.9(138-157)	148(141-155)	145(140-150)	.738
Serum LDL (after)	107(101-112)	116(112-120)	116(113-120)	.007
Serum TG (before)	255(213-297)	290(253-327)	305(275-336)	.184
Serum TG (after)	140(124-156)	148(130-166)	155(140-160)	.514

** Anova

Table 21 gives the mean of before –after **hemoglobin** by the primary, secondary, graduate&above education of participant.Mean of hemoglobin by the education of participant P-value were (P=.072, P=.005).Mean of hemoglobin (before-after) by the primary education were (12.7-13.6), whereas Mean of hemoglobin (before-after) by the secondary education were (13.2-14.1) and Mean of hemoglobin (before-after) by the graduate education were (12.7-13.4).

Table 21 shows the mean of before –after **serum homocystein** by the primary, secondary, graduate&above education of participant.Mean of serum homocystein by the education of participant P-value were (P=.631, P=.703).Mean of serum homocystein (before-after) by the primary education were (6.5-7.15),whereas Mean serum

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homocystein of (before-after) by the secondary education were (5.81-6.55) and Mean of serum homocystein (before-after) by the graduate education were (6.86-7.34).

Table 21 gives the mean of before –after **serum cholesterol** by the primary, secondary, graduate&above education of participant.Mean of serum cholesterol by the education of participant P-value were (P=.911, P=.575).Mean serum cholesterol of (before-after) by the primary education were (303-178),whereas Mean of serum cholesterol (before-after) by the secondary education were (295-190) and Mean of serum cholesterol (before-after) by the graduate education were (294-193).

Table 21 describes the mean of before –after **serum HDL** by the primary, secondary, graduate & above education of participant.Mean of serum HDL by the education of participant P-value were (P=.003, P=.111).Mean of serum HDL (before-after) by the primary education were (43.6-51.67), whereas Mean of serum HDL (before-after) by the secondary education were (37.7-56.9) and Mean of serum HDL (before-after) by the graduate education were (34.7-52.3).

Table 21 shows the mean of before –after **serum LDL** by the primary, secondary, graduate & above education of participant.Mean of serum LDL by the education of participant P-value were (P=.738, P=.007).Mean serum LDL of (before-after) by the primary education were(147-107), whereas Mean of serum LDL (before-after) by the secondary education were (148-116) and Mean of serum LDL (before-after) by the graduate education were (145-116).

Table 21 gives the mean of before –after **serum TG** by the primary, secondary, graduate&above education of participant.Mean of serum TG by the education of participant P-value were (P=.184, P=.514).Mean serum TG of (before-after) by the primary education were(255-140), whereas Mean of serum TG (before-after) by the secondary education were (290-148) and Mean of serum TG (before-after) by the graduate education were (305-155).

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Table 22 Relationship/Association between respondent occupation and biochemical Investigation

Biochemical Variables	Occupation (N=150)			P value**
	House wife	Service holder	Business	
	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)	
Hemoglobin (before)	12.6(12.1-13.2)	12.6(12.2-12.9)	13.1(12.8-13.3)	.107
Hemoglobin (after)	13.9(13.5-14.3)	13.3(13.1-13.6)	13.9(13.6-14.1)	.013
Serum Homocystein (before)	7.05(84-14)	6.63(5.2-7.9)	6.40(5.1-7.6)	.945
Serum Homocystein (after)	8.02(1.3-14.6)	7.18(6-8.3)	6.95(5.8-8.1)	.858
Serum cholesterol(before)	219(161-277)	282(261-302)	316(290-341)	.017
Serum cholesterol (after)	189(148-230)	185(167-203)	193(179-207)	.772
Serum HDL (before)	33.3(27.9-38.7)	39(35.8-42.2)	36(33.3-38.7)	.251
Serum HDL (after)	42.8(37.3-48.2)	50.7(47.8-53.6)	56.6(53.8-59.3)	.001
Serum LDL (before)	133(96-170)	147(141-153)	147(143-151)	.332
Serum LDL (after)	107(84-130)	114(111-117)	115(112-119)	.443
Serum TG (before)	273(175-371)	302(267-336)	283(257-310)	.658
Serum TG (after)	172(92-252)	145(131-158)	153(139-168)	.472

** Anova

Table 22 gives the mean of before –after **hemoglobin** by the house wife, service holder, business occupation of participant.Mean of hemoglobin by the occupation of participant P-value were (P=.107, P=.013).Mean of hemoglobin (before-after) by the house wife occupation were (12.6-13.9), whereas Mean of hemoglobin (before-after) by the serviceholder were (12.6-13.3) and Mean of hemoglobin (before-after) by the business were (13.1-13.9).

Table 22 shows the mean of before –after **serum homocystein** palpitation by the house wife, service holder, business occupation of participant.Mean of serum homocystein by the occupation of participant P-value were (P=.945, P=.858).Mean of serum homocystein (before-after) by the house wife occupation were (7.05-8.02), whereas Mean serum homocystein of (before-after) by the service holder occupation were (6.63-7.18) and Mean of serum homocystein before-after) by the business occupation were (6.40-6.95).

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Table 22 gives the mean of before –after **Serum cholesterol** by the house wife, service holder, business occupation of participant.Mean of Serum cholesterol by the occupation of participant P-value were (P=.017, P=.772).Mean of Serum cholesterol (before-after) by the house wife occupation were (219-189),whereas Mean of Serum cholesterol (before-after) by the service holder occupation were (1282-185) and Mean of Serum cholesterol (before-after) by the business occupation were (316-193).

Table 22 describes the mean of before –after **Serum HDL** by the house wife, service holder, business occupation of participant.Mean of Serum HDL by the occupation of participant P-value were (P=.251, P=.001).Mean of Serum HDL (before-after) by the house wife occupation were (33.3-42.8), whereas Mean of Serum HDL (before-after) by the service holder were (39-50) and Mean of Serum HDL (before-after) by the business occupation were (36-56).

Table 22 shows the mean of before –after **Serum LDL** by the house wife, service holder, business occupation of participant.Mean of Serum LDL by the occupation of participant P-value were (P=.332 P=.443).Mean of Serum LDL (before-after) by the house wife occupation were(133-107), whereas Mean of Serum LDL (before-after) by the service holder ocupation were (147-114) and Mean of Serum LDL (before-after) by the business occupation were (147-115).

Table 22 gives the mean of before –after **serum TG** by the house wife, service holder, business occupation of participant.Mean of serum TG by the occupation of participant P-value were (P=.658, P=.472).Mean of serum TG (before-after) by the house wife occupation were(273-172), whereas Mean of serum TG (before-after) by the service holder occupation were (302-145) and Mean of serum TG (before-after) by the business occupation were (283-153).

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Table 23 Relationship/ Association between respondent Income and clinical sing & symptoms

Clinical Variables	Income (N=150)			P-value**
	<13000	13000-24000	>25000	
	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)	
Chest pain (before)	1(1-1)	1.19(1.03-1.34)	1.17(1.10-1.25)	.231
Chest pain (after)	1.86(1.65-2.07)	1.89(1.76-2.02)	1.94(1.89-1.98)	.484
Palpitation (before)	1(1-1)	1.11(.98-1.24)	1.20(1.13-1.28)	.114
Palpitation (after)	1.93(1.77-2.08)	1.78(1.61-1.95)	1.85(1.79-1.92)	.423
Brething problem(before)	1.14(.93-1.35)	1.30(1.11-1.48)	1.26(1.17-1.34)	.561
Brething problem(after)	1.86(1.65-2.07)	1.81(1.66-1.97)	1.89(1.83-1.95)	.571
Edema(before)	1.29(1.02-1.56)	1.56(1.36-1.76)	1.67(1.58-1.76)	.016
Edema(after)	1.79(1.54-2.03)	1.67(1.48-1.86)	1.83(1.75-1.90)	.191
BP diastolic (before)	112(107-116)	106(100-112)	124(120-129)	.000
BP diastolic (after)	92(90-94)	91(88-94)	89(88-90)	.178
BP systolic (before)	129(117-140)	135(118-151)	152(144-159)	.028
BP systolic (after)	107(92-123)	100(88-113)	98(93-102)	.411

** Anova

Table 23 shows the mean of before –after **chest pain** by the income of participant.Mean of chest pain by the income of participant P-value were (P=.231, P=.484).Mean of chest pain (before-after) by the <13000 income were (1-1.86), whereas Mean of chest pain (before-after) by the 13000-24000 income were (1.19-1.789) and Mean of chest pain (before-after) by the >25000 income were (1.17-1.94).

Table 23 gives the mean of before –after **palpitation** by the income of participant.Mean of palpitation by the income of participant P-value were (P=.114, P=.423).Mean of palpitation (before-after) by the <13000 income were (1-1.93), whereas Mean palpitation of (before-after) by the 13000-24000 income were (1.11-1.78) and Mean of palpitation (before-after) by the >25000 were (1.20-1.85).

Table 23 describes the mean of before –after **breathing problem** by the income of participant.Mean of breathing problem by the income of participant P-value were

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(P=.561, P=.571).Mean breathing problem of (before-after) by the<13000 income were (1.14-1.86),whereas Mean of breathing problem (before-after) by the 13000-24000 income were (1.30-1.81) and Mean of breathing problem (before-after) by the >25000 were (1.26-1.89).

Table 23 gives the mean of before –after **edema** by the income of participant.Mean of edema by the income of participant P-value were (P=.016, P=.191).Mean of edema (before-after) by the <13000 were (1.29-1.79), whereas Mean of edema (before-after) by the 13000-24000 were (1.56-1.67) and Mean of edema (before-after) by the >25000 were (1.67-1.83).

Table 23 shows the mean of before –after **BP-diastolic** by the income of participant.Mean of BP-diastolic by the income of participant P-value were (P=.000, P=.178).Mean BP-diastolic of (before-after) by the <13000 income were(112-92), whereas Mean of BP-diastolic (before-after) by the 13000-24000 income were (106-91) and Mean of BP-diastolic (before-after) by the >25000 were (124-89).

Table 23 describes the mean of before –after **BP-systolic** by the income of participant.Mean of BP-systolic by the income of participant P-value were (P=.028, P=.411).Mean BP-systolic of (before-after) by the<13000 were(129-107), whereas Mean of BP-systolic (before-after) by the 13000-24000 income were (135-100) and Mean of BP-systolic (before-after) by the >25000 were (152-98).

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Table 24 Relationship/Association between respondent Income and Biochemical Investigation

Biochemical Variables	Income (N=150)			P value**
	<13000	13000-24000	>25000	
	Mean (95%CI)	Mean (95%CI)	Mean (95%CI)	
Hemoglobin (before)	13.1(12.8-13.4)	13.1(12.7-13.5)	12.7(12.4-13)	.224
Hemoglobin (after)	14.2(13.9-14.5)	13.8(13.4-14.2)	13.5(13.2-13.7)	.046
Serum Homocystein (before)	2.31(2-2.55)	5.44(3.2-7.6)	7.34(6.2-8.4)	.003
Serum Homocystein (after)	3.73(3.1-4.2)	5.96(4-7.8)	7.82(6.8-8.7)	.004
Serum cholesterol(before)	343(284-401)	295(240-349)	290(274-307)	.179
Serum cholesterol (after)	207(156-257)	193(167-218)	186(173-198)	.527
Serum HDL (before)	37(25.8-48.1)	37(31.7-42.3)	37.4(35.3-39.5)	.987
Serum HDL (after)	54.2(47.8-60.7)	49.7(46.6-52.9)	54.1(51.6-56.6)	.247
Serum LDL (before)	154(141-166)	144(140-148)	146(142-151)	.419
Serum LDL (after)	102(97-107)	118(115-121)	115(112-118)	.002
Serum TG (before)	312(231-394)	319(278-359)	282(257-307)	.338
Serum TG (after)	132(124-139)	142(126-157)	155(142-167)	.309

** Anova

Table 24 gives the mean of before –after **hemoglobin** by the income of participant.Mean of hemoglobin by the income of participant P-value were (P=.224, P=.046).Mean of hemoglobin (before-after) by the income were (13.1-14.2), whereas Mean of hemoglobin (before-after) by the income were (13.1-13.8) and Mean of hemoglobin (before-after) by the income were (12.7-13.5).

Table 24 shows the mean of before –after **serum homocystein** by the income of participant.Mean of serum homocystein by the income of participant P-value were (P=.003, P=.004).Mean of serum homocystein (before-after) by the<13000 income were (2.31-3.73), whereas Mean serum homocystein of (before-after) by the 13000-25000 income were (5.44-5.96) and Mean of serum homocystein before-after) by the >25000 income were (7.34-7.82).

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Table 24 gives the mean of before –after **Serum cholesterol** by the income of participant.Mean of Serum cholesterol by the income of participant P-value were (P=.179, P=.527).Mean of Serum cholesterol (before-after) by the <13000 income were (343-207),whereas Mean of Serum cholesterol (before-after) by the 13000-25000 income were (295-193) and Mean of Serum cholesterol (before-after) by the >25000 income were (290-186).

Table 24 describes the mean of before –after **Serum HDL** by the income of participant.Mean of Serum HDL by the income of participant P-value were (P=.987, P=.247).Mean of Serum HDL (before-after) by the<13000 income were (37-54.2), whereas Mean of Serum HDL (before-after) by the 13000-25000 income were (37-49.7) and Mean of Serum HDL (before-after) by the >25000 income were (37.4-54.1).

Table 24 gives the mean of before –after **Serum LDL** by the income of participant.Mean of Serum LDL by the income of participant P-value were (P=.419 P=.002).Mean of Serum LDL (before-after) by the<13000 income were (154-102), whereas Mean of Serum LDL (before-after) by the13000-25000 income were (144-118) and Mean of Serum LDL (before-after) by the >25000 income were (146-115).

Table 24 shows the mean of before –after **serum TG** by the income of participant.Mean of serum TG by the income of participant P-value were (P=.338, P=.309).Mean of serum TG (before-after) by the<13000 income were(312-132), whereas Mean of serum TG (before-after) by the 13000-25000 income were (319-142) and Mean of serum TG (before-after) by the >25000 income were (282-155).

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Table 25 Correlation among different variables of the participants

Variables	Age	BMI	BP systolic before	BP systolic after	BP diastolic before	BP diastolic after	Hb before	Hb after
Age	1	-.018	.041	-.011	.011	-.021	.257**	.269**
BMI		1	-.232**	.105	.091	-.360**	.094	.144
BP systolic (before)			1	.283**	.392**	-.318**	.175**	.102
BP systolic (after)				1	-.208*	.206*	-.194*	-.249**
BP diastolic (before)					1	-.363**	.249**	.334**
BP diastolic (after)						1	.013	.024
Hb (before)							1	.887**
Hb (after)								1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 25 shows correlation among different variables of the participants.Significant correlations of BP systolic before between BP systolic after (r=0.283), BP diastolic before(r=-.208) and BP diastolic after(r=-.363), Hb before(r=0.013) between Hb after(r=0.887) and BMI (r=-.018) were observed.

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Table 26 Correlation among different variables of the participants

Variables	Folic acid (before)	Folic acid (after)	Serum cholesterol (before)	Serum cholesterol (after)	Serum HDL (before)	Serum HDL (after)	Serum LDL (before)	Serum LDL (after)	Serum TG (before)	Serum TG (after)
Folic acid (before)	1	.972**	-.027	-.120	-.076	-.130	-.020	.142	.219**	.132
Folic acid (after)		1	-.039	-.135	-.084	-.141	.000	.141	.170*	.104
Serum Cholesterol (before)			1	.623**	.033	.077	.071	.070	.176*	.026
Serum Cholesterol (after)				1	.050	.034	.040	-.030	.097	-.022
Serum HDL (before)					1	.164*	.067	-.025	-.168*	-.089
Serum HDL (after)						1	.090	.086	-.056	-.072
Serum LDL (before)							1	.178*	.073	.252*
Serum LDL (after)								1	.126	.048
Serum TG (before)									1	.246*
Serum TG (after)										1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

Table 26 shows correlation among different variables of the participants.Significant correlations of folate before between folate after (r=.972), cholesterol before and cholesterol after(r=.623), serum HDL after and serum HDL after (r=.164), serum LDL after and serum LDL after (r=.178), serum TG after and TG after (r=.246) were observed.

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CHAPTER VII

DISCUSSION

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DISCUSSION

The study was carriedout the effects of antioxidants on cardiovascular diseases.In this study investigations were made about their socio-economic status, clinical sign & symptoms and biochemical parameters.Specialy emphases were given the effects of antioxidants on cardiovascular diseases (Hypertention, MI, IHD patients).

The present study comprises 150 cardiovascular diseases patients, of whom the largest group 56.0% belongs to age57-60 years(Figure-1).Within three groups; the largest group 68.7 % were male, rest of them 31.3 % were female (Figure-2).In relation to education most of them were graduate & above 54.7 % (Figure-3),the largest group 50.0 % were business (Figure-4). The largest group 72.7 % were family income >25000TK (Figure-5). Rimm EB et al this study has been shown the socio-demography conditions which were similar to us. [71]

In this study before antioxidants treatment chest pain was 84.0 % after antioxidant treatment chest pain was 8.0 %, before antioxidants treatment palpitation was 83.3 % and after antioxidant treatment palpitation was 15.3 %, before breathing problem was 74.7 % and after breathing problem was 12.7 % and before edema was 38.7 % and after edema was 20.7 % and all the difference was significant (p=.000)(Table-4) whereas,antioxidants treatment on both BP systolic and BP dystolic normal participant significantly (p=0.000) increased. In the other hands antioxidants treatment on both BP systolic and BP dystolic abnormal participant significantly (p=0.000) decreased (Table-5).Meanwhile before antioxidants treatment normal homocystein level were 46.7%, normal serum cholesterol 5.3 %, normal Serum HDL-Cholesterol 29.3 % , normal Serum LDL-Cholesterol 8.7 % and normal Serum Triglyceride 2.7 %,which were significantly(p=0.000) increased after antioxidant treatment(Table-6),the less similarity were show by Enstrom JE, Kanim LE, Klein MA of their study. [72]

The present study described, before antioxidants intervention the mean&std (146±39) of BP systolic and after antioxidants intervention the mean&std (99±25) of BP systolic were significantly (p=0.000) reduced. Whereas before antioxidants intervention the mean&std (120±22) of BP diastolic and after antioxidants intervention the mean&std (90±6) of BP

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diastolic were significantly (p=0.000) reduced. In the same way, before intervention program serum heamoglobin mean&std (12±1), serum folate mean&std (6±5) and serum HDL mean&std (37±12) were significantly (p=0.000) increased after intervention program.whereas, before intervention program serum cholesterol mean&std (37±12), serum LDL mean&std (146±22) and serum TG mean&std (291±128) were significantly (p=0.000) reduced after intervention program (Table-7) and also the study showed that antioxidant lowering effect was much higher of chest pain on hypertention (6.0%) followed by MI (8.0%) and IHD (10.0%).In relation to palpitation, showed that antioxidant effects was less on IHD (10.0%), followed by hyperention (12.0%), and MI (24.0%) patients.In the same way we candescribe antioxidant effects in hypertensive, MI, and IHD patients(Table-8).Whereas the present study describes the antioxidants effects on BP in hypertensive, MI, and IHD patients.It showed that antioxidant lowering effect was much higher in BP systolic on hypertensive (100.0%) patients, followed by IHD (94.0%) and MI (92.0%) patientsIn relation to BP diastolic, showed that from antioxidant effects BP was become normal level on hypertention (100.0%), followed by IHD (94.0%), and MI (90.0%) patients(Table-9).This study correlates with the study of Kritchevsky SB, Tell GS, Shimakawa T, Dennis B, Li R, Kohlmeier L, Steere E, Heiss G,where they found more or less same to our study. [73]

The presents study described the antioxidants effects on biochemical investigation in hypertensive, MI, and IHD patients. It showed that antioxidant lowering effect was much higher of serum homocystein on hypertention (100.0%) followed by IHD (94.0%) and MI (92.0%).In terms of serum cholesterol, showed that antioxidant effects was less on MI (80.0%), followed by hyperention (88.0%), and MI (88.0%) patients.and It showed that antioxidant lowering effect was much higher of serum HDL-cholesterol on hypertention (98.0%) followed by MI (94.0%) and IHD (82.0%). In relation to serum cholesterol,-LDL showed that antioxidant effects was less on IHD (92.0%), followed by MI (96.0%), and hypertention (98.0%) patients In the same way we can describe the antioxidant effects of serum TG and hemoglobin level in hypertensive, MI, and IHD patients (Table-10), which were almost similar to that of a study done by Stephens NG at el. [74]

The study shows the achievement of antioxidants effects on hypertension, MI and IHD by biochemical test.It showed that mean±std of BP systolic were significantly (p=.000)

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reduced on hypertention after intervention program followed by MI and IHD patients.In terms of BPdiastolic mean±std were significantly (P=.000) reduced on hypertention after intervention program followed by MI and IHD patients .In relation to hemoglobin, serum homocystein, serum HDL mean±std were significantly (P=.000) increased on MI after intervention program followed by hypertention and IHD patients, whereas serum cholesterol, serumLDL, and serum TG, mean±std were significantly (P=.000) reduced after intervention program on IHD followed by MI and Hypertention patients(Table-11). In this study shows the achievement of antioxidants effects on hypertension, MI and IHD by clinical sign & symptoms.It showed that mean±std of chest pain, palpitation, breathing problem and edema were significant (p=.000) on hypertention.In terms of MI mean±std of chest pain, palpitation, breathing problem and edema were significant (P=.000)..In relation to IHD mean±std of chest pain, palpitation, breathing problem and edema were significant (p=.000) (Table-12), the study were similar shown by Rapola JM, Virtamo J, Ripatti S, Huttunen JK, Albanes D, Taylor PR, Heinonen OP. [75]

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CHAPTER SIX

CONCLUSION & RECOMMENDATIONS

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6.1 Conclusion

The study was carried out among 150 cardiovascular diseases patients of which 50 were myocardial infraction (MI), 50 were Hypertensive, 50 were Ischemic Heart Disease (IHD), to observe the effect of antioxidants.

i. The study reveled that vit-A, (ß –carotein), vit-E, and vit-C have lowering effect on clinical sing and symptoms and high blood pressure of MI, IHD, and Hypertensive patients.

ii. The antioxidants have also lowering effects on Serum lipid profile (Serum cholesterol, Serum HDL, Serum LDL, and Serum TG) suffering from various cardiovascular diseases, such as; MI, IHD, and hypertension.

iii. On the other hand Hb level and Folic acid levels were increased after antioxidant intervention in C.V.D patients.

iv. Antioxidants activity lowering effects on sign and symptoms were much higher in hypertensive patients followed by MI and IHD, where as antioxidant effects on BP (systolic and diastolic) were much higher in IHD patients followed by hypertensive and MI patients.

v. The antioxidant had higher percentage of increasing effect on serum folate in hypertensive patients and also hemoglobin levels were increased in MI patients. Serum cholesterol, serum LDL were decreased in hypertensive patients whereas TG level were decreased in IHD patients.

vi. There were significant relationship among socio-demography (sex, education, occupation, and income), clinical sign & symptoms (chest pain, palpitation, edema, breathing problem, high BP) and biochemical investigations (Hemoglobin level, serum folic acid, and serum lipid profile) were found.

vii. Positive Significant correlation among different variables (clinical and biochemical investigation) of the participants were observed.

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6.2 Recommendations

i. This study was carried out in a small scale sample that's why it is recommended that similar study can be carried out in a large scale data to have effective result.

ii. Antioxidants based food intake to be ensured.

iii. On the basis of the analysis and interpretation of the findings it is necessary to take antioxidants as supplementations to reduce dysfunctional conditions of the heart (heart diseases) and vessels that supply oxygen to organs and tissues throughout the body and also have effects on clinical sign & symptoms and biochemical parameters.

iv. More antioxidants supplementations are needed for increasing serum folic acid level, serum heamoglobin level and serum HDL cholesterol levels in human beings to prevent cardiovascular diseases.

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APPENDIX

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APPENDIX

PRE & POST DATA COLLECTION SHEET

Dear respondent

I am a student of **MPhil in INFS, Dhaka University.** As a part of my academic requirement I am conducting a research work on **"Effect of antioxidants on cardiovascular disease".** I would sincerely appreciate if you would give me a few minutes and help me in doing the research. All the information's provided by you will be kept **strictly confidential** and used only for research purpose.

Thank you.

Case no……. Date……

SECTION 1: SOCIO-DEMOGRAPHIC INFORMATION:

1. Respondent name: …… Mobile: ……
2. Address……
3. Age: …… .Years
4. Sex: □Male □Female (√)
5. Height: …….. cm
6. Weight: ……..kg
7. BMI: ……
8. Educational qualification (√):
   □ Primary □ secondary □ Graduate & above
9. Occupation (√):
   □House wife □Service holder □Business
   □Others (please specify): ……
10. Number of family member (√):
    □ Small Family (<4 members) □ Medium Family (5-7 members) □Large Family (>8 members)
11. Family type: □ Nuclear Family □ Joint family
12. Family income (√):
    □<13000 TK □ 13000-24000 TK □ >25000 TK

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13. Diagnosis: ☐ Hypertension ☐ MI ☐ UA (IHD)

14. Blood Pressure (BP): Systolic: Before.....mmHg

After.....mmHg

Diastolic: Before.....mmHg

After.....mmHg

SECTION 2: SIGN & SYMPTOMATIC INFORMATION ON CARDIOVASCULAR DISEASES:

Before	After
1. Chest pain? (√): ☐Yes ☐No 2. Palpitation? (√): ☐Yes ☐No 3. Breathing problem? (√): ☐ Yes ☐ No 4. Edema? (√): ☐ Yes ☐ No.	1. Chest pain? (√): ☐Yes ☐No 2. Palpitation? (√): ☐Yes ☐No 3. Breathing problem? (√): ☐ Yes ☐ No 4. Edema? (√): ☐ Yes ☐ No.

SECTION 3: BIOCHEMICAL INVESTIGATION ON CARDIOVASCULAR DISEASES:

Biochemical Test	Before Result	After Result
Hg level (g/dl)		
Serum Folic acid level (ng/ml)		
Serum Cholesterol (Total) (mg/dl)		
Serum HDL- cholesterol (mg/dl)		
Serum LDL- holesterol (mg/dl)		
Serum riglycerides (mg/dl)		