

Chapter One

Introduction, Rationale, and Objectives

1.1 Introduction:

one of the most popular drinks worldwide is coffee, and its name is derived from the name of province Keffe which is located in Ethiopia (Shateri and Djafarian, 2016). Also, is a popular beverage that is heavily in the Sudan (Gaffer, 2013). Coffee is the most traded commodity second after oil, it has thousands of chemical compounds which can name chlorogenic acid, caffeine, potassium, niacin, magnesium and tocopherols (Shateri and Djafarian, 2016). Coffee can be prepared in two main ways: filtered and un filtered (Gaffer, 2013). cafestol is a lipid soluble diterpene present in coffee beans. unfiltered coffee brews contain 3-6 mg of this diterpene per cup. Filtered coffee does not contain cafestol because the diterpene is retained by a paper filter (Roos *et al.*, 2001). Caffeine is the most widely consumed psychoactive substance (Khojah, 2016) enhances dopamine signaling in the brain also stimulation of the central nervous system and cardiac muscle, increased urinary output and relaxation of smooth muscles (Volkow *et al.*, 2015).

Lipids may be defined as compounds which are relatively insoluble in water, but freely soluble in non polar organic solvents like benzene, chloroform, ether, hot alcohol, acetone, etc (vasudevan, sreekumari, and vaidyanathan, 2011). Lipids are either compounds that yield fatty acids when hydrolyzed or complex alcohols that combine with fatty acids to form esters, are carried in the blood stream by complexes known as lipoproteins, the four main types of lipoproteins are chylomicrons, VLDL, LDL and HDL (Arnesson and brickell, 2007). the major lipids present in plasma are fatty acid, triglycerides, cholesterol and phospholipids; other lipid soluble substance present in much smaller amount but of considerable physiological importance include steroid hormones and fat soluble vitamins (marshall, bangert and lapsley, 2012).

The effect of coffee consumption on serum lipid levels varies depending on the method of preparation and the type of coffee. Certain coffee preparation methods may result in increased serum total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) levels, whereas other

methods do not (Karabudak, andturkozu and koksai, 2015).for example, jee et al said: consumption of unfiltered coffee increase serum levels of total and LDL cholesterol(Jee *et al.* , 2001). While , Kondo et al said: The relationship between coffee consumption and incidence of CVD has been studied extensively, coffee intake might have an antiatherogenic property(Kondo *et al.*,2010).regular coffee ingestion may favorably affect cardiovascular risk status by modestly reducing LDL oxidation susceptibility and decreasing LDL-cholesterol levels(Yuhawa *et al.* , 2004).

1.2 Rationale:

Coffee is a widely consumed and socially accepted drink all over the world, especially in the Sudan.

Coffee contain more over chemical substance that can affect in our body.

For example caffeine in coffee has been shown to have various pharmacological and cellular responses in a wide spectrum of biological system include: stimulation of the central nervous system and cardiac muscles, increase urinary output and relaxation of smooth muscles.

Also cafestol and kahweol are active chemicals in coffee mechanistically responsible for blood lipids esterification.

For all this it is important to know if coffee good or bad habits.

1.3 Objectives :

1.3.1 General objective:

To assess serum lipids profile among coffee consumption people.

1.3.2 Specific objectives:

- 1- To measure and compare the serum total cholesterol, high density lipoprotein cholesterol, and low density lipoprotein cholesterol mean levels with normal range.
- 2- To correlate between serum total cholesterol, high density lipoprotein cholesterol, low density lipoprotein cholesterol mean levels and duration.
- 3- To compare between number of coffee cups per day and serum total cholesterol, high density lipoprotein cholesterol, and low density lipoprotein cholesterol mean levels in coffee drinker.

Chapter Two

literature review:

2.1 Coffee:

2.1.1 History of coffee:

From its legendary origins to modern times, coffee has been praised and valued for its taste and, more importantly, its effects on arousal. As a result, this simple fruit of the coffee plant became the basis for an industry that has grown over the centuries to multibillion dollar proportions. World coffee exports average six million tons annually or over 100 million bags of coffee beans (International Coffee Organization {ICO}, 2006).

Although humans have been drinking coffee for centuries, it is not clear just where coffee originated or who first discovered it. However, the predominant legend has it that an observant goatherd named Kaldi discovered coffee in the Ethiopian highlands. Various dates for this legend include 900 B.C, 800 B.C, 300 A.D, 600 to 800 A.D, and 800 A.D. Regardless of the actual date, it is said that Kaldi noticed that his goats did not sleep at night after eating berries from what would later be known as a coffee tree. When Kaldi reported his observation to the local monastery, the abbot became the first person to brew a batch of coffee and note its alerting effect when he drank it. Word of the arousing effects and pleasant taste of this new beverage soon spread beyond the monastery, initially east to the Arabian Peninsula and eventually throughout the world (International Coffee Organization {ICO}, 2006).

The first known reference to coffee in Arabic writings came from an Islamic physician, Abu Bakr Muhammad ibn Zakariya El Razi (known as “Rhazes”), who wrote a now lost medical textbook circa 900 A.D. Rhazes made the first reference to what can be reliably identified as coffee, and archaeologists have found iron roasting pans dating to 1000 A.D. The oldest extant accounts of coffee roasting date to the writings of the famous Islamic physician Ibn Sina, They cultivated coffee and, by the 15th century, were growing it in quantity in the Yemeni district, from which it spread, by the next century, to Syria, Egypt, Persia, and Turkey. Today, coffee plants are cultivated in more than 50 countries, resulting in

a wide variety of coffees, each with its own combination of flavor, body, and aroma. Depending on the type of coffee bean, where it is grown, the conditions under which it thrives, and how it is harvested and processed, coffee can have a wide variety of flavors and textures (International Coffee Organization {ICO}, 2006).

2.1.2 Nomenclature:

Linnaeus was first to describe and classify the coffee plant in his book *Species Plantarum*, published in 1753. The plants that yield coffee beans as their fruit are members of the genus *Coffea*, which is in the Rubiaceae family, a part of the subkingdom Angiospermae (Table 1-1). Other members of that family include the gardenia and plants from which quinine is derived. Coffee plants are quite varied. They can range in size from small shrubs to 30-foot trees, and their leaves vary in color from purple to yellow. Because of this variability, botanists have failed to agree on a precise classification system, but there appear to be at least 25 perhaps as many as 100 species. However, the two varieties of coffee that its drinkers are most familiar with are Arabica and Canephora (commonly called Robusta).

Table(1-1) Botanical Classification of Coffee:

Kingdom	Vegetable
Subkingdom	Angiospermae
Class	Dicotyledoneae
Subclass	Sympetalae
Order	Rubiales
Family	Rubiaceae
Genus	<i>Coffea</i>
Subgenus	<i>Eucoffea</i>
Species	<i>C.arabica</i> , <i>C.canephora</i>

2.1.3 The Composition of Coffee:

The substance present in coffee that are described as physiologically active can be subdivided as follows:

- (a) Substances naturally present in the green beans, including : caffeine; chlorogenic acid; the glycosides and lipid; and those

transformed or formed at roasting such as trigonellin and volatile constituents, which create the unique flavor.

(b) Exogenous substances, the ubiquitous polycyclic aromatic hydrocarbons, the pesticides and the mycotoxins which may sometimes contaminate coffee (Stickgold and Walker, 2009).

2.1.3.1 .Caffeine:

Caffeine has the chemical formula $C_8H_{10}N_4O_2$ and can be chemically named 1,3,7- trimethylxanthine, 1H-Purine-2,6-dione, 3,7-dihydro-1,3,7-trimethyl, methyltheobromine, 1,3,7-Trimethyl-2,6-dioxopurine, and 7-Methyltheophylline (Institute of Medicine (U.S.) Committee on Military Nutrition Research, 2001).

Caffeine is part of the pure alkaloid group, which are sometimes called methylated xanthines; its isomers are theophylline, theobromine, and paraxanthine. It cannot melt and is moderately soluble in water similar to the average human body temperature, but is fully soluble in hot water (Weinberg and Bealer, 2001).

Found naturally in over 60 species, caffeine has evolved as a protective mechanism to kill bacteria, fungi, weeds, and insects in plants (Chambers, 2009). Humans have continued to discover these different caffeinated plants across different continents and time periods, as early as 700,000 B.C. At room temperature, the compound caffeine is odorless, white, bitter, and in a powder or crystal structure (Wilson and Temple, 2004).

Coffee is the top source of caffeine, Arabica is the most widely grown species, is used for brewed coffee, and has 0.58-1.7% caffeine by weight in its dry green bean form and 1% caffeine by volume, brewed. Robusta is mainly used for instant coffee mixes and has 1.16-3.27% caffeine by weight in its bean form and 2% caffeine by volume, brewed; instant coffee is often a mixture of both Arabica and Robusta. Arabica has half of the caffeine content as well, due to the bean's high lipid content. Roasting coffee can alter its caffeine content, as it burns away caffeine and water. A dark roast will have more caffeine by bean weight, but a light roast will have more caffeine by coffee volume (Stickgold and Walker, 2009).

Depending on the quantity of coffee, the bean type, and the strength, a cup of coffee can contain 40-400 mg caffeine, with the average six ounce

cup having 60-180 mg. It should be noted that decaffeinated coffee also has a caffeine content of 5-25 mg per serving, depending on preparation (Alpert, 2012).

The Absorption and Metabolism of Caffeine

Caffeine is metabolized quickly because of its water solubility, and is absorbed in the stomach and intestines. Alterations in pH or food present in the gastrointestinal tract affect the rate of absorption. Once in the liver, 98% of the ingested amount is then metabolized by cytochrome p450 monooxygenase into many metabolites through several steps; less than two percent of the end product trimethyl uric acids, like 1-methyluric acid, are then excreted in the urine. The untouched remaining 2% is then distributed via the blood as a demethylated compound, like paraxanthine, theophylline, or theobromine. These compounds continue to break down further into one of 25 different metabolites for hepatobiliary elimination by enzymes CYP1A2, CYP2E1, xanthine oxidase, and N-acetyltransferase (Chambers, 2009).

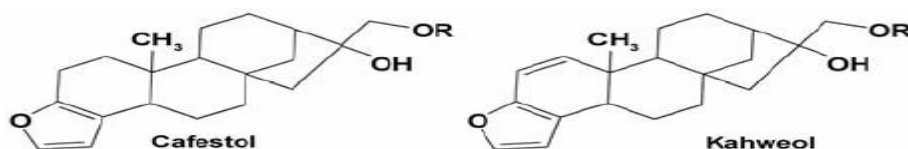
Caffeine, mostly as paraxanthine, is transported amongst all body tissues and secretions equally, from breastmilk to saliva and semen. Caffeine also passes through the blood-brain barrier in the central nervous system via simple diffusion and carrier-mediated transport. It is then that the metabolic rate, serotonin secretion, heart rate, norepinephrine/epinephrine secretion, gastric secretion, smooth muscle relaxation, glomerular filtration, skin temperature, muscle contractility, and urinary output all increase (Stickgold and Walker, 2009).

Peak caffeine concentrations occur from 15-120 minutes after ingestion. The concentrations of caffeine in the body rely on a multitude of factors. Metabolism is slowed, meaning that it stays in the body longer, by alcohol, Asian ethnicity, oral contraceptives, liver damage, the luteal phase of the menstrual cycle, high altitudes, pregnancy, and by being male or a newborn. Cigarettes, Caucasian ethnicity, and female or child status speed metabolism of the drug. Additionally, theobromine is an inhibitor of itself and caffeine, so habitual caffeine consumers may find an extended effect (Weinberg and Bealer, 2001).

Since caffeine metabolizes quickly, it does not accumulate within the body. As the theobromine and caffeine forms do not bind to plasma

albumin, they do not additionally break down; this extended period gives caffeine its half-life of 2-4 hours and up to 9.5 hours, with variable times between age, gender, and substance use (Institute of Medicine (U.S.) Committee on Military Nutrition Research, 2001).

2.1.3.2 Cafestol and Kahweol:



Cafestol is a lipid soluble diterpene present in coffee beans. Unfiltered coffee brews contain 3-6 mg of this diterpene per cup. Filtered coffee does not contain cafestol because the diterpene is retained by a paper filter (Roos et al, 2001).

Two compounds that have caught recently more attention, due to their physiological effects, are cafestol and kahweol, two pentacyclic furan diterpenes exclusively found in coffee. Their structure is nearly identical differing only in the degree of saturation of one conjugated bond to the furan ring. Beside these two main diterpenes, other ones have been identified, namely 16-O-methylcafestol, which is only found in the robusta coffee variety and is defined as a specific marker according to the German industrial standard DIN 10779 (Roos et al, 2001).

Only a small fraction of cafestol and kahweol, about 0.7–3.5% represent in coffee beans as free diterpene alcohols, while the majority occurs esterified with fatty acids in the form of diterpene esters.

Kurzrock and Speer (2001) identified up to 14 different fatty acid esters linked to cafestol and kahweol, being the most common palmitic acid (36–49%), followed by linoleic acid (22–30 %) and several others only present in minor amounts, like oleic, stearic and eicosanoic acid (Kurzrock and Speer, 2001).

2.1.3.3 Chlorogenic acids:

are a family of esters formed between certain trans-cinnamic acids (phenolic acids that usually are caffeic acid, ferulic acid and p-coumaric acids) and quinic acid, These secondary metabolites are present in coffee beans and contribute to the astringency and bitterness of a coffee

beverage, and have potential as an antioxidant for human health (Oestreich- Janzen, 2010).

Chlorogenic acids are one of the most abundant polyphenols present in plant and plant-based foods and coffee has been reported to be one of the richest sources of chlorogenic acids in the human diet compared to other beverages, A cup of Arabica coffee brew (200 mL) contains 70-200 mg of chlorogenic acid, while in Robusta it may reach 70-350 mg . Due to thermal instability, further processing, particularly roasting of green coffee beans, has been reported to progressively degrade chlorogenic acids up to 93% for dark roasting (Farah, 2012).

Chlorogenic acids and quinic acid may form chlorogenic lactones during coffee roasting which contribute to increased bitterness of the coffee brew (Ginz and Engelhardt, 2001).

2.1.3.4 Polysaccharides:

are the major component of coffee beans for instance 44-47%, in the form of arabinogalactans, mannans and cellulose. Polysaccharides play an important role in retaining volatiles and therefore flavour, and also contribute to the perceived viscosity of the coffee brew. Other carbohydrate compounds such as glucose and fructose are mainly found in immature beans while higher amounts of sucrose accumulate in mature beans and contribute to perceived coffee sweetness (Wasserman *et al.*, 2012).

2.1.3.5 The lipid fraction of coffee:

also known as the coffee oil consists of triglycerides (75%), free and esterified diterpene alcohols (19%), free and esterified sterols (5%), and a small quantity of other lipid types such as tocopherols. The diterpenes kahweol and cafestol in coffee are often mentioned as having a negative effect on health in relation to cholesterol (Kölling-Speer, 2001).

Roasting of coffee beans results in partial migration of coffee oil to the bean's surface (Savonitti, 2005). While some changes in coffee lipid profile occurs during roasting, sterols and most triglycerides remain unchanged (Maier, 2005). These lipid fractions of the beans are extracted into the coffee brew and provide the crema emulsion of espresso coffee that carries flavour volatiles and fat-soluble vitamins, and contributes to

perceived texture and mouthfeel of the coffee brew (Oestreich-Janzen, 2010).

2.1.3.6 Protein:

content of Arabica is slightly lower than Robusta, even though total amino acid composition is similar. The amino acids content of green bean has an important contribution to flavor development during roasting through Maillard reactions (Liu and Kitts, 2011). Maillard or caramelization reactions occur due to a reaction between the amine group of amino acids or nitrogen-containing compounds and the carboxyl group of reducing sugars, hydroxy-acids and phenols to yield amino aldoses and amino ketones by condensation, The resulting product is the brownish colour melanoidins and other components such as several nitrogen and/or sulfur containing heterocyclic compounds which are thought to be important flavor compounds in coffee (Buffo and Cardelli-Freire, 2004).

2.1.3.7 minerals :

Potassium is the major mineral present in roasted coffee, however, manganese, iron, and copper are also present in smaller amounts and act as important catalysts of certain biochemical reactions which facilitate the production and release of flavour components in coffee bean during processing (Oestreich-Janzen, 2010).

2.2 Coffee Consumption:

Coffee is made from the beans of ripe berries of a tropical evergreen shrub. Coffee trees belong to the genus *Coffea*, a member of the Rubiaceae family which includes more than 500 genera and 6000 species of tropical trees and shrubs. There are more than 80 species within *Coffea*, but the commercial production of coffee relies on two species, Arabica coffee (*Coffea arabica*) and Robusta coffee (*Coffea canephora*). However, it is the Arabica that predominates, representing more than 70% of the world coffee production.

There are three major steps in preparing green beans for consumption. Firstly, they must be precisely roasted to highlight any outstanding characteristics of the coffee. Secondly, beans are ground according to brewing requirements. Lastly, the freshly roasted and ground coffee must

be brewed at the right temperature for the correct amount of time to bring out its best quality and flavor. Coffees of various origins are sometimes blended in different proportions so as to make a cup with varying acidity and different taste characteristics. Hundreds of coffee varieties grow in different regions in the world produces beans with distinctive characteristics. The essential aim of the blending is to balance the flavors needed to create a superior coffee. Many blends contain up to 7 different types of beans. The variety of recipes for roasting, brewing, and serving coffee reflects the diversity of consumer tastes and cultural preferences, Common spices used in coffee include cardamom, cloves, ginger, anise, cinnamon, nutmeg, and saffron; but the spice of choice is often cardamom (Almaghrabi, 2007).

2.2.1 Effect Coffee consumption on human body

2.2.1.1 Cholesterol and coffee

are statistically correlated, with boiled coffee increasing blood lipid concentration, Filtered coffee does not have this effect, as this relationship is due to the small lipid content in coffee beans. Other methods of preparation impacted by this finding include Turkish, French-press, and espresso style coffee (Alpert, 2012).

2.2.1.2 Coffee and liver:

caffeine has been reported to have effects on metabolism as a whole, including an increase in the metabolic rates of lipolysis, glycogenolysis, and gluconeogenesis (Smit *et al.*, 2004) Increases in these metabolic processes lead to increased energy charge in the form of ATP production through glucose oxidation(Kennedy and Scholey, 2004).

The researchers themselves concluded that this increase was due to an increase in both carbohydrate and lipid oxidation and might suggest an increase in the Cori cycle's transformation of lactate to glucose in the liver and glucose to lactate in anaerobic muscle tissues, These effects mean that caffeine, overall, increases resting metabolic rate (Glade, 2010).

2.2.1.3 Coffee and brain:

caffeine reportedly increases catecholamine release and triggers CNS activation through increased cAMP concentration in postsynaptic cells . Concentration enhancement of this common second messenger “may increase the strength of transmitted signals” by increasing the strength of EPSPs (excitatory post-synaptic potentials) facilitated by cAMP cascades (Glade, 2010).

2.3 Lipids:

2.3.1 Definitions:

Lipids may be defined as compounds which are relatively insoluble in water, but freely soluble in nonpolar organic solvents like benzene, chloroform, ether, hot alcohol, acetone, etc.(Vasudevan, Sreekumari and Vaidyanthan, 2011).

In addition, lipids and lipoproteins are intimately involved in the development of atherosclerosis, a pathogenic process that is the underlying cause of the common cardiovascular disorders of:

(1) myocardial infarction, (2) cerebrovascular disease, and (3) peripheral vascular disease (Burtis *et al.*, 2008).

2.3.2 Functions of Lipids:

1. Storage form of energy (triglycerides)
2. Structural components of bio membranes (phospholipids & cholesterol)
3. Metabolic regulators (steroid hormones and prostaglandins)
4. Act as surfactants, detergents and emulsify in agents (amphipathic lipids)
5. Act as electric insulators in neurons
6. Provide insulation against changes in external temperature (subcutaneous fat)
7. Give shape and contour to the body
8. Protect internal organs by providing a cushioning effect (pads of fat)
9. Help in absorption of fat soluble vitamins (A, D, E and K)

10. Improve taste and palatability of food (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.3.3 Classification of lipids:

Based on the chemical nature, lipids are classified as:

1. Simple lipids: They are esters of fatty acids with glycerol or other higher alcohols .
2. Compound lipids: They are fatty acids esterified with alcohol; but in addition they contain other groups. Depending on these extra groups, they are sub classified in: A. Phospholipids, containing phosphoric acid. B. Non-phosphorylated lipids.
3. Derived lipids: They are compounds which are derived from lipids or precursors of lipids, e.g. fatty acids, steroids. For details of cholesterol and steroids.
4. Lipids complexed to other compounds (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.3.4 Cholesterol:

Cholesterol is found almost exclusively in animals and is a key membrane component of all cells. It is a steroid alcohol with 27 carbon atoms that are arranged in a tetra cyclical sterane ring system, with a C-H side chain. Cholesterol is primarily composed of C-H bonds, and hence it is fairly water insoluble. It does, however, contain a polar hydroxyl (OH) group on its A-ring Thus, it is both a polar and non polar molecule (amphipathic) (Burtis *et al.*, 2008).

Cholesterol Absorption

Before being absorbed, cholesterol is first solubilized through a process called emulsification. Emulsification occurs by the formation of mixed micelles that contain un esterified cholesterol, fatty acids, mono glycerides, phospholipids, and conjugated bile acids. Bile acids, by acting as detergents, are the most critical factor in micelle formation. Increased amounts of fat in the diet results in the increase of mixed micelles, which in turn allows for more cholesterol absorption (Burtis *et al.*, 2008).

Most cholesterol absorption occurs in the middle jejunum and terminal ileum parts of the small intestine and is mediated by the enterocyte surface protein, NPC1L1. This protein is the target for the drug ezetimibe that blocks cholesterol absorption. Typically, between 30% to 60% of dietary cholesterol is absorbed per day, which represents as much as 1 g/day when one is on a high fat diet (Burtis *et al.*, 2008).

Once cholesterol enters the intestinal mucosal cell, it is packaged with triglycerides, phospholipids, and a large protein called apolipoprotein (apo) B-48 into large lipoprotein particles called chylomicrons. Chylomicrons are secreted into the lymph and eventually enter the circulation where they deliver the absorbed dietary lipid to the liver and peripheral tissues (Burtis *et al.*, 2008).

Cholesterol synthesis

Cholesterol also is endogenously synthesized with almost 90% of its synthesis occurring in the liver and intestine. Most peripheral cells instead depend on the exogenous delivery of cholesterol by lipoproteins. Cholesterol biosynthesis occurs in three stages:

In the first stage ;acetyl-CoA, a key metabolic intermediate derived from carbohydrates, amino acids, and fatty acids, forms the six-carbon thioester HMG- CoA.

In the second stage, HMG-CoA is reduced to mevalonate, and then is decarboxylated to a series of five carbon isoprene units. These isoprene units are then condensed to form first a 10-carbon (geranyl pyrophosphate) and then a 15 carbon intermediate (farnesyl pyrophosphate). Two of these CI, molecules then combine to produce the final product of the second stage, squalene, a 30 carbon acyclic hydrocarbon. The second stage is important because it contains the step involving the microsomal enzyme HMG-CoA reductase, which is the rate limiting enzyme in cholesterol biosynthesis and is inhibited by the statin type drugs. The enzyme that forms farnesyl pyrophosphate, geranyl transferase, is an important second site of regulation because inhibition here permits the formation of physiologically important intermediate isoprenoids in the absence of cholesterol synthesis.

The third stage occurs in the endoplasmic reticulum, with many of the intermediate products being bound to a specific carrier protein. Squalene is initially oxidized and then undergoes cyclization to form the 4-ring,30-carbon intermediate, lanosterol. In a series of oxidation decarboxylation reactions, a number of side chains are removed from the tetra cyclical sterane ring structure to form the 27-carbon molecule of cholesterol (Burtis *et al.*, 2008).

Cholesterol Esterification

Cholesterol is esterified to a fatty acid to form a cholesteryl ester by two different enzymes. In the cell, excess cholesterol is esterified by acyl cholesterol acyl transferase (ACAT), which helps reduce the cytotoxicity of excess free cholesterol. Once esterified, cholesteryl esters are stored in intracellular lipid drops. The esterification of cholesterol by ACAT involves the energy dependent activation of a fatty acid with thio coenzyme A (CoASH) to form an acyl-CoA, which in turn reacts with the hydroxyl group on cholesterol to form an ester.

Cholesteryl esters also are formed in the circulation by the action of lecithin cholesterol acyl transferase (LCAT) on cholesterol in lipoproteins, particularly on high density lipoproteins (HDL). The LCAT reaction does not require CoASH. It results from fatty acid transfer from the second carbon position of lecithin (phosphatidylcholine) to cholesterol . Cholesteryl esters account for about 70% of the total cholesterol in plasma, and LCAT is responsible for the formation of most of the cholesteryl esters in plasma. LCAT is secreted by the liver into the circulation and is activated by apolipoprotein A-I, the main protein on HDL. Once cholesterol is esterified, it loses its free hydroxyl group and becomes much more hydrophobic and goes from the surface of lipoprotein particles to the hydrophobic core (Burtis *et al.*, 2008).

Cholesterol Catabolism

Except for specialized endocrine cells that use cholesterol for the synthesis of steroid hormones, most peripheral cells have limited ability to further catabolize cholesterol. Cholesteryl esters are hydrolyzed to free cholesterol by various lipases in all cells, but thereafter, cholesterol has to be returned to the liver to undergo any further catabolism. Approximately one third of the daily production of cholesterol, or about 400mg/day, is

converted in the liver into bile acids . About 90% of the bile acids are reabsorbed in the lower third of the ileum and are eventually returned to the liver by the enter hepatic circulation. Bile acids that enter the large intestine are partially de conjugated by bacterial enzymes to secondary bile acids. Cholic acid is converted, for example, to deoxycholic acid, and chenodeoxycholic acid is converted to lithocholic acid. Not all cholesterol delivered to the liver is converted to bile salts. Much of it is resecreted into the circulation on lipoproteins and the remainder is directly excreted into the bile unchanged, where it is solubilized into mixed micelles by bile acids and phospholipids. When the amount of cholesterol in bile exceeds the capacity of these solubilizing agents, it is possible for cholesterol to precipitate and form cholesterol gallstones (Burtis *et al.*, 2008).

2.3.5 Acylglycerol (Glycerol Esters):

Glycerol is a three-carbon alcohol that contains a hydroxyl group on each of its carbon atoms. Chemically, it is possible to esterify each hydroxyl group with a fatty acid .The two terminal carbon atoms in the glycerol molecule are chemically equivalent and designated a and a'. The center carbon is labeled P. A common alternative labeling system uses the numeral 1 for the α carbon, 2 for the β carbon, and 3 for the α carbon. The class of acyl glycerol is determined by the number of fatty acyl groups present:

- (1)one fatty acid, mono acylglycerols (monoglycerides);
- (2)two fatty acids, diacylglycerols (diglycerides);and
- (3) three fatty acids, triacylglycerols (triglycerides).

2.3.6 Lipoproteins:

2.3.6.1 Chylomicrons:

Synthesis of chylomicrons

Chylomicrons are formed in the intestinal mucosal cells, and secreted into the lacteals of lymphatic system . They are rich in triglyceride. When the chylomicrons are synthesized by the intestinal mucosa, they contain only

apo-B-48 and apo-A but apo-C and apo-E are added from HDL in blood during transport (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Metabolism of Chylomicrons

Main sites of metabolism of chylomicrons are adipose tissue and skeletal muscle. The half-life of chylomicrons in blood is about 1 hour. The enzyme lipoprotein lipase (LpL) is located at the endothelial layer of capillaries of adipose tissue, muscles and heart; but not in liver. Apo C-II present in the chylomicrons activates the LpL. The LpL hydrolyses triglycerides present in chylomicrons into fatty acids and glycerol. Muscle or adipose tissue cells take up the liberated fatty acids. Following injection of heparin, the LpL is released from the tissues and lipemia is thus cleared. This is called post heparin lipolytic activity. Lack of C-II leads to decreased activity of LpL and consequent accumulation of chylomicrons and VLDL in blood. Insulin increases LpL activity (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Liver Takes up Chylomicron Remnants

As the TAG content is progressively decreased, the chylomicrons shrink in size. These remnants containing apo-B-48 and apo-E are taken up by hepatic cells by receptor mediated endocytosis. Apo-E binds the hepatic receptors (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Function of Chylomicrons

Chylomicrons are the transport form of dietary triglycerides from intestines to the adipose tissue for storage; and to muscle or heart for their energy needs (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.3.6.2 Very low density lipoproteins:

Synthesis of VLDL

They are synthesized in the liver from glycerol and fatty acids and incorporated into VLDL along with hepatic cholesterol, apo-B-100, C-II and E. Apo-B-100 is the major lipoprotein present in VLDL when it is secreted. Apo-E and C-II are obtained from HDL in plasma (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Metabolism of VLDL

The half life of VLDL in serum is only 1 to 3 hours. When they reach the peripheral tissues, apo-C-II activates LpL which liberates fatty acids that are taken up by adipose tissue and muscle. The remnant is now designated as IDL (intermediate density lipoprotein) and contains less of TAG and more of cholesterol. The major fraction of IDL further loses triglyceride, so as to be converted to LDL (low density lipoprotein). This conversion of VLDL to IDL and then to LDL is referred to as lipoprotein cascade pathway. A fraction of IDL is taken up by the hepatic receptors (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Function of VLDL

VLDL carries triglycerides (endogenous triglycerides) from liver to peripheral tissues for energy needs (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.3.6.3 Low density lipoprotein (LDL):

LDL transports cholesterol from liver to peripheral tissues. The only apoprotein present in LDL is apo B100. Most of the LDL particles are derived from VLDL, but a small part is directly released from liver. The half-life of LDL in blood is about 2 days (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Metabolism of LDL and LDL Receptors

LDL is taken up by peripheral tissues by receptor mediated endocytosis. LDL receptors are present on all cells but most abundant in hepatic cells. LDL receptors are located in specialized regions called clathrincoated pits. Binding of LDL to the receptor is by apo-B-100 and uptake of cholesterol from LDL is a highly regulated process. When the apo-B-100 binds to the apo-B-100 receptor, the receptor-LDL complex is internalized by endocytosis.

The endosome vesicle thus formed fuses with lysosomes. The receptor is recycled and returns to the cell surface. The LDL particle, along with apoproteins and cholesterol ester are hydrolysed by lysosomal hydrolases, forming amino acids and free cholesterol. The free receptors can now return to the membrane surface to bind further LDL molecules (Fig. 12.13). Approximately 70% of LDL is degraded in the liver, and the rest in extra-hepatic tissues. For their work on LDL receptors, Michael Brown

and Joseph Goldstein were awarded Nobel prize in 1985 (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Function of LDL

About 75% of the plasma cholesterol is incorporated into the LDL particles. LDL transports cholesterol from liver to the peripheral tissues. The cholesterol thus liberated in the cell has three major fates:

- i. It is used for the synthesis of other steroids like steroid hormones.
- ii. Cholesterol may be incorporated into the membranes.

Cholesterol may be esterified to a MUFA by acyl cholesterol acyl transferase (ACAT) for storage. The cellular content of cholesterol regulates further endogenous synthesis of cholesterol by regulating HMG CoA reductase (Vasudevan, Sreekumari and Vaidyanthan, 2011).

LDL and Clinical Applications

LDL concentration in blood has positive correlation with incidence of cardiovascular diseases. A fraction of cholesterol is taken up by macrophages, this is not a regulated pathway. Increased levels of LDL or modification of LDL by glycation (as seen in diabetes mellitus) or oxidation increases the fraction of cholesterol taken up by macrophages. LDL infiltrates through arterial walls, and is taken up by macrophages or scavenger cells. This is the starting event of atherosclerosis leading to myocardial infarction .

When these cells become engorged with cholesterol, foam cells are formed, that get deposited in the sub endothelial space triggering formation of atheromatous plaque. Pro coagulant changes are induced in the endothelium resulting in increased chances of thrombosis and coronary artery disease.

Since LDL-cholesterol is thus deposited in tissues, the LDL (low density lipoprotein) variety is called “bad cholesterol” and LDL as “Lethally Dangerous Lipoprotein” in common parlance (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.3.6.4 High density lipoproteins(HDL):

High density lipoproteins transport cholesterol from peripheral tissues to the liver. The major apoproteins in HDL are Apo-A1, with some Apo-A2, Apo-C and Apo-E. HDL serves as a plasma reservoir of Apo-C and Apo-E which can be transferred to VLDL and chylomicrons and back (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Metabolism of HDL

The intestinal cells synthesis components of HDL and release into blood. The nascent HDL in plasma is discoid in shape . The free cholesterol derived from peripheral tissue cells are taken up by the HDL. The apo-A-1 of HDL activates LCAT (lecithin cholesterol acyl transferase) present in the plasma. The LCAT then binds to the HDL disk. The cholesterol from the cell is transferred to HDL by a cholesterol efflux regulator protein which is an ABC protein.

Lecithin is a component of phospholipid bilayer of the HDL disk.. The second carbon of lecithin contains one molecule of polyunsaturated fatty acid (PUFA). It is transferred to the third hydroxyl group of cholesterol to form cholesterol ester. The esterified cholesterol which is more hydrophobic, moves into the interior of the HDL disk.

This reaction continues; till HDL becomes spherical with a lot of cholesterol esters are formed. This HDL particle designated as HDL-3.

Mature HDL spheres are taken up by liver cells by apo-A-1 mediated receptor mechanism. HDL is taken up by hepatic scavenger receptor B1. Hepatic lipase hydrolyses HDL phospholipid and TAG, and cholesterol esters are released into liver cells. The cholesterol that reaches the liver is used for synthesis of bile acids or excreted as such in bile. The scavenger receptor B1 (SR-B1) is identified as an HDL receptor with dual role in HDL metabolism. In liver and steroidogenic tissues, it delivers cholesteryl ester to tissues whereas in the tissues it is involved in reverse cholesterol transfer.

When the HDL-3 remains in circulation, the cholesterol ester from HDL is transferred to VLDL, IDL and LDL by a Cholesterol Ester Transfer Protein (CETP). This will help to relieve product inhibition of LCAT so that more cholesterol can be taken up. Triacylglycerol from VLDL, IDL and LDL is transferred to HDL in exchange for the cholesterol ester. The

HDL particles that are rich in triacylglycerol and spherical are called HDL-2. These particles are first acted upon by hepatic triglyceride lipase (HTGL) before being taken up by scavenger B1 receptors in liver.

The efflux of cholesterol from peripheral cells to HDL is mediated by the ABC transporter protein. The reverse cholesterol transport to liver through HDL needs the activity of LCAT, CETP and Apo-D (Vasudevan, Sreekumari and Vaidyanthan, 2011).

Functions of HDL

- i. HDL is the main transport form of cholesterol from peripheral tissue to liver, which is later excreted through bile. This is called reverse cholesterol transport by HDL.
- ii. The only excretory route of cholesterol from the body is the bile.
- iii. Excretion of cholesterol needs prior esterification with PUFA. Thus PUFA will help in lowering of cholesterol in the body, and so PUFA is anti-atherogenic.

Clinical Significance of HDL

The level of HDL in serum is inversely related to the incidence of myocardial infarction. As it is “anti-atherogenic” or “protective” in nature, HDL is known as “good cholesterol” in common. It is convenient to remember that "H" in HDL stands for "Healthy". HDL level below 35 mg/dl increases the risk, while level above 60 mg/dl protects the person from coronary artery diseases (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.4 Plasma lipids:

Total plasma lipid is 400-600 mg/dl. Normal values of lipid fractions are shown in . Out of this, 40% is cholesterol; 30% is phospholipids; 20% is triglycerides. Since lipids are insoluble in water, they need the help of carriers in plasma. Therefore, they are complexed with proteins to form lipoproteins. The protein part of lipoprotein is called apolipoprotein. The lipoproteins are usually abbreviated as Lp (Vasudevan, Sreekumari and Vaidyanthan, 2011).

2.5 The relationship between coffee consumption and serum lipids:

The effect of coffee consumption on serum lipid levels varies depending on the method of preparation and the type of coffee. Certain coffee preparation methods may result in increased serum total cholesterol (TC) and lowdensity lipoprotein cholesterol (LDL-C) levels, whereas other do not (Karabudak, Turkozu, and Koksak, 2015).

Like Yukawa *et al*: coffee ingestion (8 g of Arabic coffee) resulted in a significant decrease in serum levels of cholesterol, LDL cholesterol, and MDA, and a significant decrease in susceptibility of LDL to oxidation, indicating that coffee consumption might protect against atherosclerosis due to lowering serum lipid levels and improving LDL susceptibility (Yukawa *et al*, 2003). For example, previous studies have indicated that increased consumption of unfiltered coffee results in a dose dependent increase in serum levels of TC, LDL-C and triglycerides (TG) (Cai *et al*, 2012).

In addition to the quantity of coffee consumed, coffee consumption habits may affect the serum lipid levels. Coffee consumption combined with smoking is known to affect serum lipid levels. A previous study revealed that smoking while consuming coffee caused atherogenic risk to increase synergistically, with increased serum LDL-C levels and reduced high density lipoprotein cholesterol (HDL-C) levels (Yuan, Sun, and Butler, 2011). However, additional studies in inhabitants, is only 0.4-0.7 kg per capita. By contrast, coffee have not identified an increase in the risk of coronary heart disease as a result of this combination (Iopez-Garcia *et al.*, 2006; Rebllo, and van Dam, 2013).

Of recent, some researchers have reported that cafestol and kahweol are the active chemicals in coffee mechanistically responsible for the increase in serum cholesterol level after coffee consumption (Adebayo *et al.*, 2007). In humans, short term consumption of diterpens (cafestol and kahweol) is associated with increased serum activity levels of the lipids transfer protein cholesteryl ester transfer protein (CETP) and phospholipid transfer protein (PLTP) and decreased activity levels of lecithin cholesterol acyltransferase (LCAT). CETP catalyses the transfer of cholesteryl esters synthesized by LCAT from HDL to the apolipoprotein B containing lipoproteins LDL and VLDL. PLTP can

affect the net mass transfer of phospholipids between lipoproteins and it also converts small sized HDL (HDL₃) into both larger (HDL₂ sized) and smaller (pre β migrating) HDL particles. consumption of cafestol and kahweol cause a long term increase in CETP as well as PLTP activity; the increase in CETP activity may contribute to the rise in LDL cholesterol (Roos *et al.*, 2000). Cafestol increase plasma triacylglycerol by increasing the production rate of VLDL₁, apo B, probably via increased assembly of VLDL₁ in liver (Roos *et al.*, 2001).

Chapter Three:

Material and methods:

3.1 Study area:

This study was carried out in Khartoum state.

3.2 Study design:

This was descriptive cross sectional study.

3.3 Study duration:

This study was carried out from March to June. 2018.

3.4 Sample size:

Seventy subjected was involved in this study, 30 were males and 40 were females.

3.5 Inclusion criteria:

Study included healthy people with normal body mass index (from 18.5 to 25.0 kg/m²) who are drinking coffee daily.

3.6 Exclusion criteria:

Study excluded any people had chronic diseases like; hypertension, diabetes mellitus and, renal failure, or smoker, pregnant women, or any diseases that affect blood lipids.

3.7 Data collection method, technique, and analyzed plan:

3.7.1 Data collection:

The data collected by using questionnaire.

3.7.2 technique:

3.7.2.1 Sampling technique:

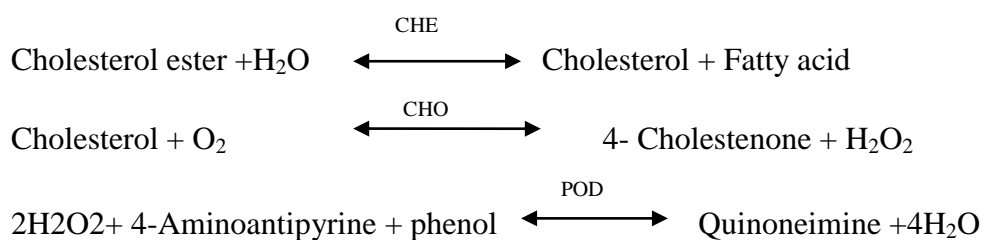
Samples were taken in lithium heparin vacotainers by using 5ml syringes; to take 3 ml of venous blood, centrifuged at 2000 rpm for 5 minutes to obtain plasma and preserved at 2-8°C for transportation and investigations by using full chemistry analyzer.

3.7.2.2 Estimation technique:

Total cholesterol:

Total cholesterol had been measured by using cholesterol oxidase peroxidase method.

The reaction principle:

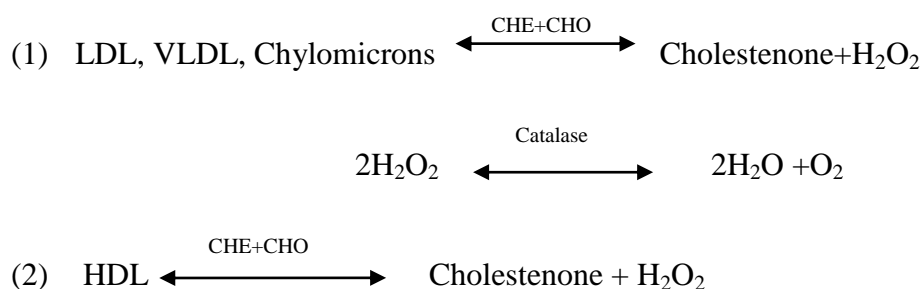


By the catalysis of CHE and CHO, Cholesterol ester is catalyzed to yield H_2O_2 , which oxidase 4-Aminoantipyrine with phenol to form a colored dye of Quinoneimine. The absorbency increase is directly proportional to the concentration of cholesterol (appendix II)

High density lipoprotein cholesterol

High density lipoprotein cholesterol (HDL-C) had been measured by using direct method.

The reaction principle:

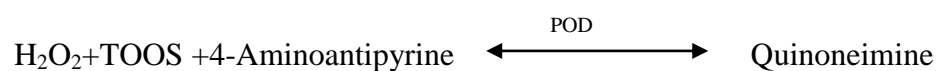
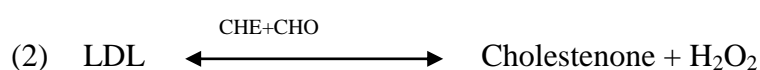
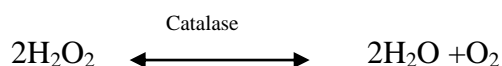
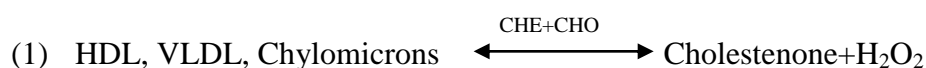


$\text{H}_2\text{O}_2 + \text{HDAOS} + \text{4-Aminoantipyrine} \xrightleftharpoons{\text{POD}} \text{Quinoneimine}$
The system monitors the change in absorbance at 600 nm. This change in absorbance is directly proportional to the concentration of cholesterol in the sample and is used by the system to calculate and express the HDL-c concentration (appendix III).

Low density lipoprotein cholesterol

High density lipoprotein cholesterol (LDL-C) had been measured by using direct method.

The reaction principle



The system monitors the change in absorbance at 600 nm. This change in absorbance is directly proportional to the concentration of cholesterol in the sample and is used by the system to calculate and express the LDL-c concentration (appendix IV).

3.7.3 Quality control:

A daily maintenance was firstly done then a set of two levels of human assayed control made by Mindary had been analyzed with each batch of samples. In addition, when the control do not recover within the acceptable tolerances range, corrective actions such as a new calibration and specific maintenance or troubleshooting procedures were done.

3.7.4 Ethical consideration:

This study ethically provided by Sudan University of Science and technology ethical clearance committee, and then informed consent was taken from all participants.

3.7.5 Statistical analysis:

Descriptive statistics for quantities and qualitative parameters, student t test, independent t test, analysis of variance (ANOVA) and mean differences were done by using SPSS program (statistical packaged for social science) version 21 where the value of $p < 0.05$ was considered as significant or association.

Chapter four

Results

This study include 70 subjects healthy coffee drinkers.

4.1 Distribution of study population according to gender:

Figure (4-1) show the distribution of coffee drinkers according to gender, out of 70 coffee drinkers 43% were male and 57% were female.

4.2 Mean of study variables among the study population

Table (4-1) show mean of study variable, the range of age from 14 to 65 years with mean 30.76 ± 10.20 , the range of BMI from 18.3 to 25.0 kg/m^2 with mean 22.55 ± 2.34 , and range of duration from 1 to 29 years with mean 7.70 ± 6.14 .

4.3 Comparison of study parameters with normal value:

Table (4-2) shows total cholesterol levels in coffee drinkers was insignificantly different when compared to normal value with p value(0.067), also LDL-c levels in coffee drinkers was insignificantly different when compared to normal value with p value(0.715), while HDL-c significantly decrease in coffee drinkers when compared to normal value with p value (0.000).

4.4 Effect of number of coffee consumption cups per day on levels of TC , LDL, and HDL :

Table (4-3) shows total cholesterol levels in coffee drinkers there drinks 1-2 cups per day was insignificantly different when compared to drinks 3 or more cups per day with p value(0.394), while LDL-c levels in coffee drinkers there drinks (1-2)cups per day was significantly decrease when compared to drinks 3 or more cups with p value (0.022), also HDL-c significantly increase in coffee drinkers there drinks 1-2 cups per day compared to drinks 3 or more cups per day with p value (0.003).

4.5 Effect of gender on levels of TC , LDL, and HDL :

Table (4-4) shows total cholesterol levels in male drank coffee was insignificantly different when compared to female drank coffee with p value(0.838), also LDL-c levels in male drank coffee was insignificantly

different when compared to female drank coffee with p value(0.699), while HDL-c significantly decrease in male drank coffee compared to female drank coffee with p value (0.032).

4.6 Frequency of TC, LDL, and HDL:

In table(4-5) The frequency analysis showed that, 59(84%) had optimal TC, 9(13%) had border line, and 2(3%) had high TC.

Frequency analysis to HDL-c showed that, 4(6%) had no risk, 29(41%) had moderate risk, and 37(53%) had high risk .

Frequency analysis to LDL-c showed that,40(57%) had optimal, 19(27%) had near optimal, 8(11%) had border line high, 2(3%) had high, 1(2%) had very high levels of LDL-c.

4.7 Correlation between TC level and duration:

in figure (4-2) showed TC level was positively correlated with duration (R=0.264, P=0.027). this indicates an increasing in duration will lead to increase in TC levels.

4.8 Correlation between HDL level and duration :

in figure (4-3) showed HDL-c level was negatively correlated with BMI (R= -0.355, P=0.033). this indicates an increasing in duration will lead to decrease in HDL-c levels.

4.9 Correlation between LDL level and duration:

in figure (4-4) showed LDL-c level was positively correlated with BMI (R=0.307, P=0.010). this indicates an increasing in duration will lead to increase in LDL-c levels.

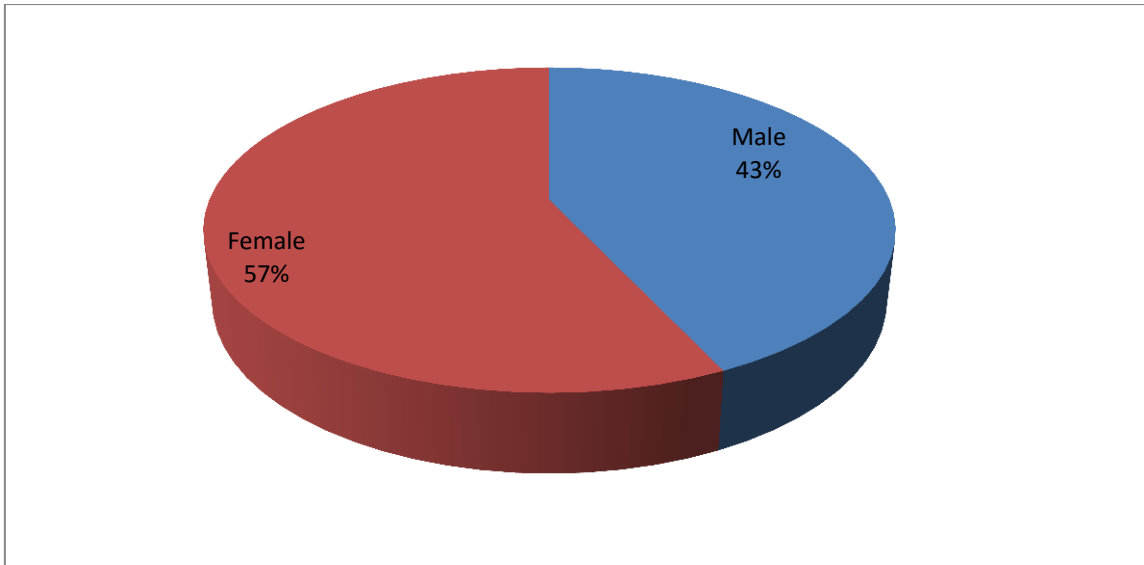


Figure (4-1) Distribution of study population according to gender

Table (4-1) Mean of study variables (age, BMI, and duration) among the study population:

Parameters	Minimum	Maximum	Mean±SD
Age	14	65	30.76±10.50
BMI	18.3	25.0	22.55±2.34
Duration (Years)	1	29	7.70±6.14

Table (4-2) Comparison of study parameters with normal value:

Parameters	Mean±SD	R.V	P-value
TC (mg/dl)	160.10±35.12	169.5 (140-199)	0.067
HDL (mg/dl)	38.94±10.7	>55	0.000
LDL (mg/dl)	98.65±30.56	<100	0.715

Table (4-3) Mean of study parameters according to number of coffee cup:

Parameters	1-2 Cups (Mean±SD)	≥3 Cups(Mean±SD)	<i>P-value</i>
TC (mg/dl)	149.54±27.79	158.82±36.58	0.394
HDL (mg/dl)	46.64±14.49	37.18±8.91	0.003
LDL (mg/dl)	84.77±20.94	100.59±31.78	0.022

Table (4-4) Mean of study parameters according to gender:

Parameters	Male (Mean±SD) NO 30	Female (Mean±SD) NO 40	<i>P-value</i>
TC (mg/dl)	156.10±40.72	157.85±30.79	0.838
HDL (mg/dl)	35.90±8.74	41.21±11.57	0.032
LDL (mg/dl)	99.30±34.88	96.42±27.29	0.699

Table (4-5) Percentage of study parameters :

Parameters	Frequency	Percentage (%)
TC		
Optimal	59	84.0
Border line	9	13.0
High	2	3.0
HDL		
No Risk	4	6.0
Moderate Risk	29	41.0
High Risk	37	53.0
LDL		
Optimal	40	57.0
Near Optimal	19	27.0
Border line high	8	11.0
High	2	3.0
Very high	1	2.0
Total	70	100.0

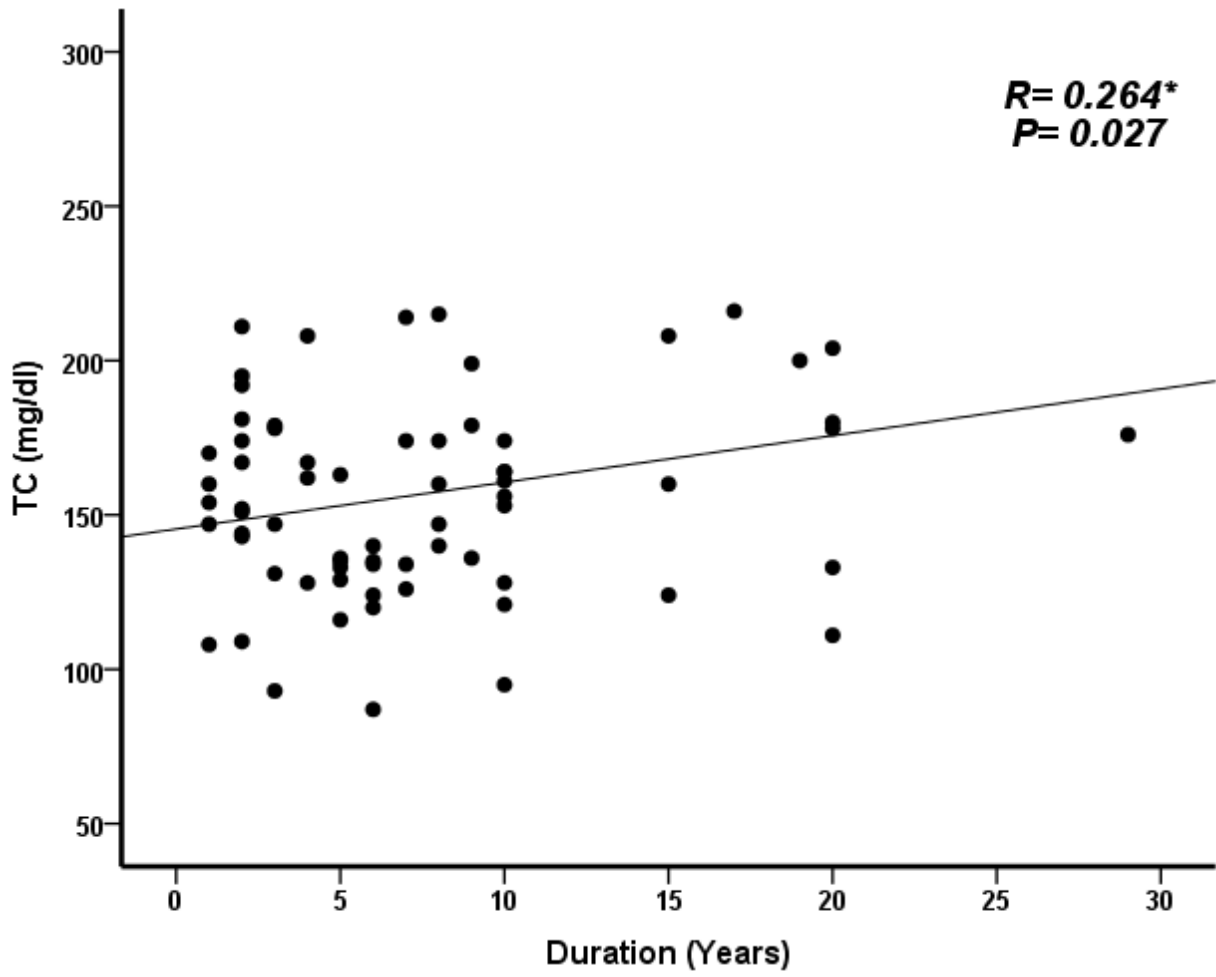


Figure (4-2) Correlation between TC level and duration (R= 0.264, P= 0.027)

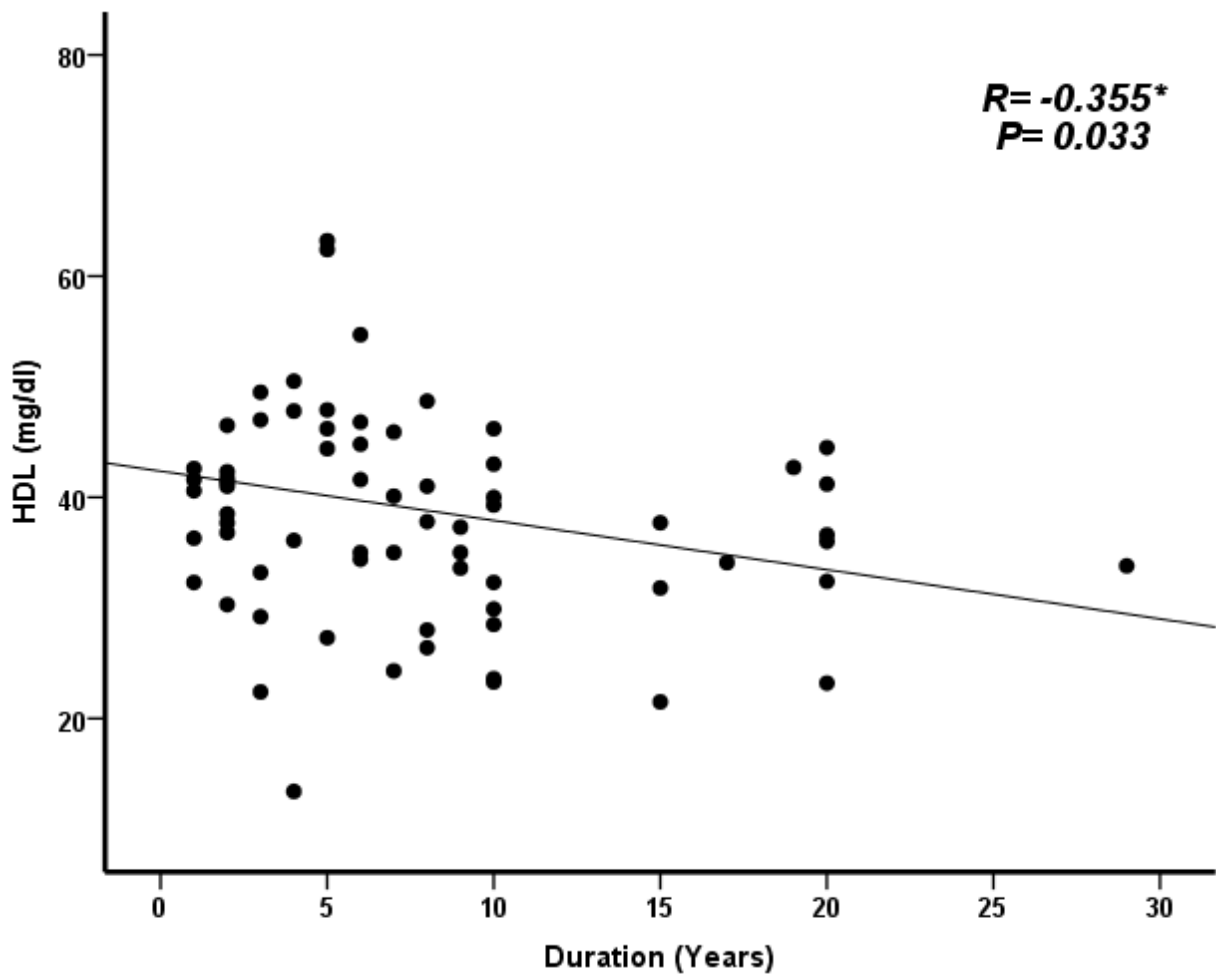


Figure (4-3) Correlation between HDL level and duration($R = -0.355$, $P = 0.033$)

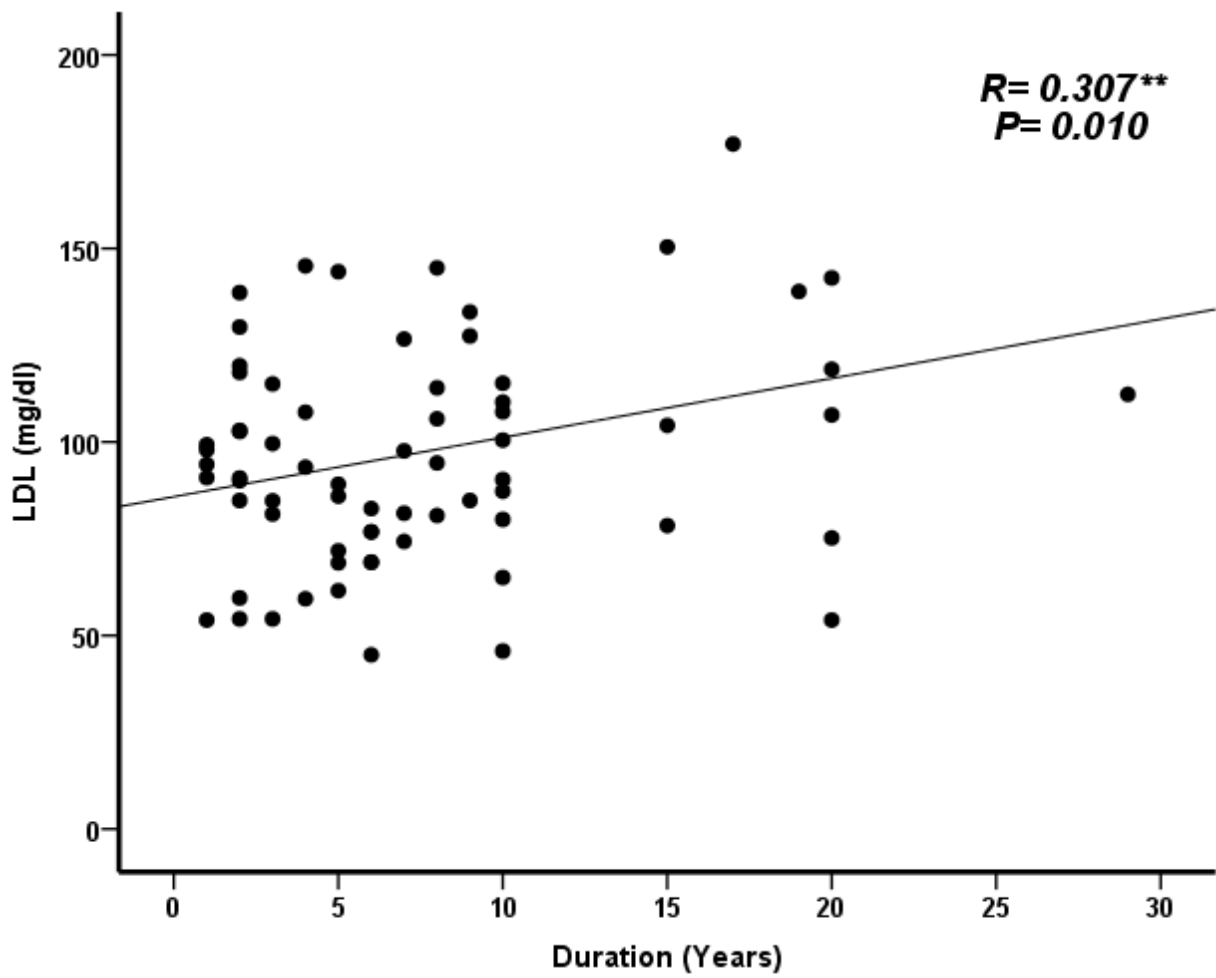


Figure (4-4) Correlation between LDL level and duration($R=0.307$, $P= 0.010$)

Chapter Five

Discussion, conclusion, and recommendation

5.1 Discussion:

This study was carried out to assess the effects of coffee consumption on the serum total cholesterol, high density lipoprotein, and low density lipoprotein mean levels and The results showed insignificantly different in total cholesterol levels and LDL-C in coffee drinkers when compared to normal value p value (0.067, 0.715) respectively. While HDL-C significantly decrease in coffee drinkers when compared to normal value with p value (0.000). The present finding corroborated several reports likewise demonstrated by Mcanlis *et al*; coffee did not affect serum lipid levels and susceptibility of LDL-C to oxidation in human (Mcanlis *et al*, 1998). Karabudak *et al* reported that no significant association between the consumption of Turkish or instant coffee and serum lipid levels (Karabudak *et al.*, 2015). Likewise Gaffer *et al* : no significant difference between means of plasma levels of total cholesterol in filtered coffee drinker group compared to control group (Gaffer *et al.*, 2013). A Randomized controlled meta analysis trial by Ricketts *et al* indicated that diterpens such as cafestol, found in unfiltered coffee, increased the levels of serum cholesterol (Ricketts *et al.*, 2007). A reason of difference in results may be due to in different genotypes and genetic environments may be involved in the variations observed in the effects of coffee on serum lipid levels in different populations, as found in the current study.

Notably, the present study identified no statistically significant differences in the serum TC levels when comparing subjects who consumed 1-2 cups with consumed 3 or more cups per day. While serum LDL-C showed significance decrease in subjects who consumed 1-2 cups when compared to who consumed 3 or more cups per day. Also serum HDL-C showed significance increase in subjects who consumed 1-2 cups when compare to who consumed 3 or more cups per day. This was in line of Urgert *et al*, long term consumption of unfiltered coffee potentially raises serum LDL-C in humans (Urgert *et al*, 1997).

According to gender, serum TC and LDL-C showed no statistically significant differences in male group drank coffee when compared with female group that drank coffee. While serum HDL-C showed

significantly decrease in male drank coffee when compared with female drank coffee.

According to The frequency TC, LDL-c and HDL-c with normal value showed that coffee is atherogenic.

According to duration of coffee consumption , increasing in duration will lead to increase in TC, and LDL-C levels and decrease in HDL-C levels. Salonen *et al*, indicated that high levels of coffee consumption were associated with a reduction in the serum levels of HDL-C in smokers, but increased levels of HDL-C were observed in non-smokers (Salonen *et al.*, 1987).

Finally, the different genotypes and genetic environmental may be involved in the variations observed in the effects of coffee on serum lipid levels in different population, as found in the current study.

5.2 Conclusion :

This study was concluded that the serum total cholesterol, and low density lipoprotein cholesterol levels in coffee consumption peoples were within international value, while serum high density lipoprotein cholesterol levels in coffee consumption peoples were significant low when compared with international value. There is significant correlation between serum total cholesterol, low density lipoprotein cholesterol, and high density lipoprotein cholesterol levels in coffee consumption peoples with duration.

5.3 Recommendation:

Recommended that:

- Coffee have benefit if taken in save quantity, so not drink large amount.
- All coffee drinker should be encouraged to employ lifestyle practices that reduce the risk of cardiovascular disease, like excesses, stop smoking, and drink coffee moderately.
- Taken fasting sample to see effect on triacylglycerol .
- Furthermore assessment include liver function test, uric acid and glucose.
- Take large number of study population and control group.

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