DISSERTATION ON

EFFECT OF HDL ON PHYSICAL PERFORMANCE AND COGNITION IN ELDERLY

submitted in partial fulfillment of the regulations

for the award of the degree of

M.D., IN GERIATRIC MEDICINE
BRANCH – XVI

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI

DEPARTMENT OF GERIATRIC MEDICINE
MADRAS MEDICAL COLLEGE
CHENNAI – 600003

MAY 2019

CERTIFICATE

This is to certify that the dissertation titled **"EFFECT OF HDL ON PHYSICAL PERFORMANCE AND COGNITION IN ELDERLY"** is the bonafide work done by **Dr. OVIYA ELANGO**, Post Graduate Student, Department of Geriatric Medicine, Madras Medical College, Chennai – 600003, in partial fulfilment of the University rules and regulations for the award of **MD DEGREE** in **GERIATRIC MEDICINE BRANCH – XVI**, under our guidance and supervision, for the examination to be held on **May 2019**.

Prof. Dr.JAYANTHI, M.D., FRCP,
M.D.(Geriatrics)
Dean
Madras medical college,
Rajiv Gandhi Govt. General Hospital
Chennai – 600003

Prof. Dr.G.S.SHANTHI
Professor and Head
Professor and Head
Madras medical college,
Rajiv Gandhi Govt. General Hospital
Chennai – 600003

,

DECLARATION

I solemnly declare that this dissertation titled **" EFFECT OF HDL ON PHYSICAL PERFORMANCE AND COGNITION IN ELDERLY"** was done by me at Madras Medical College, Chennai – 600003, during the period August 2017 to July 2018 under the guidance and supervision of **Prof. Dr.G.S.SHANTHI, M.D. (Geriatrics),** to be submitted to the The Tamilnadu Dr.M.G.R. Medical University, towards the partial fulfilment of requirements for the award of **MD DEGREE IN GERIATRIC MEDICINE BRANCH – XVI**.

PLACE: CHENNAI ***DR.OVIYA ELANGO***

DATE :

MD GERIATRIC MEDICINE,
Post Graduate Student,
Department of Geriatric Medicine,
Madras Medical College,
Rajiv Gandhi Govt. General Hospital
Chennai – 600003.

ACKNOWLEDGEMENT

I thank the Dean of Madras Medical College, for permitting me to conduct the study and use the hospital resources for the study.

I express my heartfelt gratitude to **Prof. Dr.G.S.SHANTHI, M.D.** (Geriatrics), Professor and Head, Department of Geriatric Medicine, for guidance and suggestions throughout the study period.

I also extend my sincere thanks to **Prof. Dr.S.DEEPA, M.D.,** Associate Professor, Department of Geriatric Medicine for guiding me during the study.

I am extremely thankful to **Dr.K.UMA KALYANI, M.D., D.Diab.,** Assistant Professor, **Dr.M.SENTHIL KUMAR, M.D.,** Assistant Professor, **Dr.D.THANGAM, M.D.,** Assistant Professor and **Dr.C.PRIYA MALINI, M.D., D.Diab.,** Assistant Professor, Department of Geriatric Medicine, for their valuable insights.

I thank the entire team of physiotherapists who has helped me in data collection.

I also extend my thanks to all the postgraduate students, my colleagues and paramedical staff for their cooperation and support.

LIST OF ABBREVIATIONS

HDL	High density lipoprotein
LDL	Low density lipoprotein
VLDL	Very low density lipoprotein
APO	Apolipoprotein
LPL	Lipoprotein lipase
CAD	Coronary artery disease
CKD	Chronic kidney disease
CVA	Cerebrovascular accident
SHTN	Systemic hypertension
T2DM	Type 2 diabetes mellitus
COPD	Chronic obstructive pulmonary disease
BMI	Body mass index
MMSE	Mini mental status examination
SSPB	Short physical performance battery

TABLE OF CONTENTS

INTRODUCTION

There is a significant person-to person variation in the degree of decline in physical performance and cognition in the elderly. A major factor accounting for this variability is the presence or absence of multiple chronic diseases like stroke, coronary artery disease, peripheral vascular diseases.

Numerous studies have demonstrated the importance of dyslipidemia as a risk factor for such vascular events. However, most of these studies have considered only total serum cholesterol concentration. More recently, some authors have shown that other lipid variables like HDL-cholesterol, may play a role in predicting the risks of vascular disease.

One of the most important mechanism by which HDL-cholesterol plays its atheroprotective function is the reverse cholesterol transport. Furthermore, HDL-cholesterol exerts direct nitric oxide-mediated vasodilatory effects. The capacity of HDL-cholesterol to enhance the activity of nitric oxide in the small arteries may have a role in the reduction of arterial inflammation at the place of atheroma formation.

Being a negative predictor of vascular events, the effect of HDL-cholesterol on physical performance and cognition in elderly has been studied in Europe and Central Asia – and a positive correlation found.

The studies done in this subject were mainly from Korea and China (Central Asia). We could not find any Indian studies. Hence the purpose of this study is to assess the effect of HDL cholesterol on physical performance and cognition in the Indian population .If a positive correlation is established, improved HDL-cholesterol levels by pharmacological intervention could get better physical performance and cognition in the aged.

AIMS AND OBJECTVES

AIM

- To study the effect of HDL on physical performance and cognition in elderly.

- To analyze the association of HDL with co-morbidities.

OBJECTIVES

Persons attending geriatric OPD at Rajiv Gandhi Govt. General Hospital, are categorized into three tertiles based on HDL levels and 50 patients from each group are selected by simple random sampling The physical performance and cognition of patients in each group is assessed through SPPB and MMSE score and the effect of HDL cholesterol on physical performance and cognition in elderly, studied.

REVIEW OF LITERATURE

Lipoproteins are “large macromolecular complexes composed of lipids and proteins (apolipoproteins) that play an essential role in

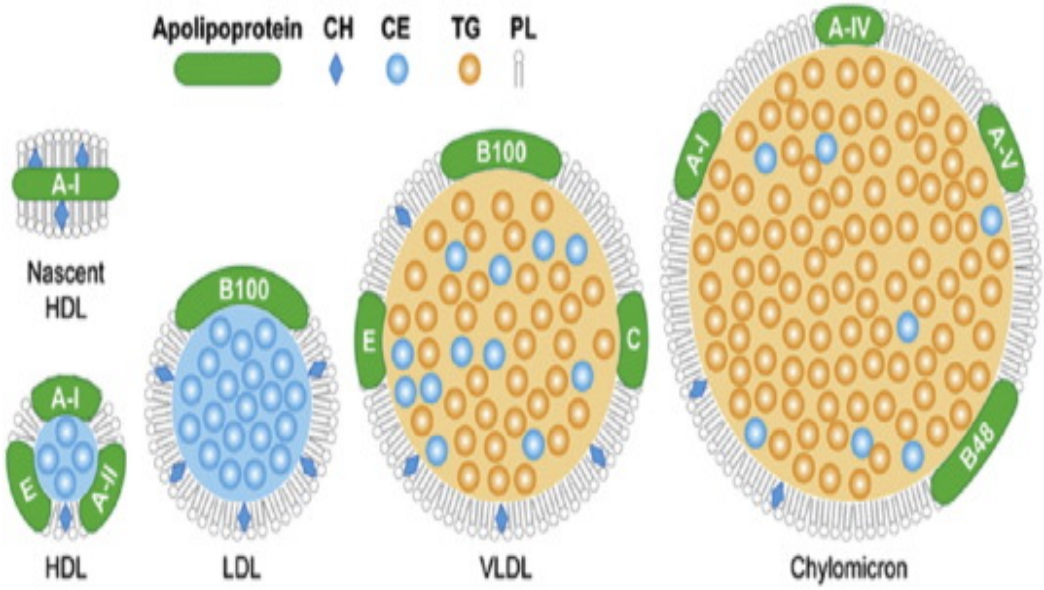
- Absorption of dietary cholesterol, long chain fatty acids and fat soluble vitamins
- Transport of triglycerides, cholesterol and fat soluble vitamins from the liver to peripheral tissues and
- Transport of cholesterol from peripheral tissues to the liver and intestine.”

The amount of lipid (which is less dense than water) determines the density of a lipoprotein. Based on their relative densities, lipoproteins are classified into five groups:

- Chylomicrons
- Very low density lipoprotein (VLDL)
- Intermediate density lipoprotein (IDL)
- Low density lipoprotein (LDL)
- High density lipoprotein (HDL)

Chylomicrons, that are the most lipid rich, are the least dense lipoprotein particles; whereas HDLs that have the least lipid content are the most dense lipoproteins.

The proteins associated with lipoproteins called apolipoproteins are required for the assembly, structure, function and metabolism of lipoproteins. Apolipoproteins act as ligands for cell surface receptors and play an important role in the activation of enzymes involved in lipoprotein metabolism.



Types of Apolipoproteins -

ApoB is a very large protein and is the major structural protein of chylomicrons, VLDLs, IDLs and LDLs; one molecule of apoB-48 (chylomicron) or apoB-100 (VLDL, IDL or LDL) is present on each lipoprotein particle. The apoB-48 is derived from the same gene as apoB-100 by mRNA editing. While apoB-100 is synthesized in the human liver, apoB-48 is produced in the intestines.

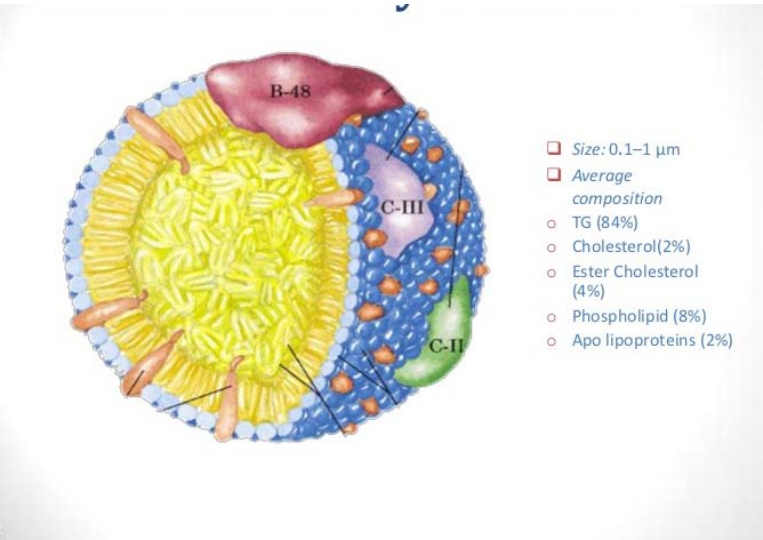
ApoA-1 is a different class of apolipoproteins, that is found virtually on all HDL particles. It is primarily synthesized in the liver and intestine. ApoA-2 is the second most abundant HDL apolipoprotein and is found in approximately 2/3rds of the HDL particles.

ApoC-1, apoC-2 and apoC-3 participate in the metabolism of triglyceride rich lipoproteins. ApoE also plays a critical role in the metabolism and clearance of triglyceride rich particles. Most apolipoproteins, other than apoB, exchange actively among lipoprotein particles in the blood.

Transport of intestinally derived dietary lipids by chylomicrons

One of the key functions of lipoproteins is the efficient transport of dietary lipids from the intestines to the peripheral tissues. The triglycerides present in the diet are first hydrolyzed by lipases within the intestinal lumen and then emulsified with bile acids to form micelles. Likewise, dietary cholesterol is absorbed in the proximal small intestine and esterified to form cholesteryl esters. Longer chain fatty acids are incorporated into triglycerides and packaged with apoB-48, cholesteryl esters, phospholipids and cholesterol to form chylomicrons. The nascent chylomicrons are then secreted into the intestinal lymph and later reach the systemic circulation via the thoracic duct.

CHYLOMICRON



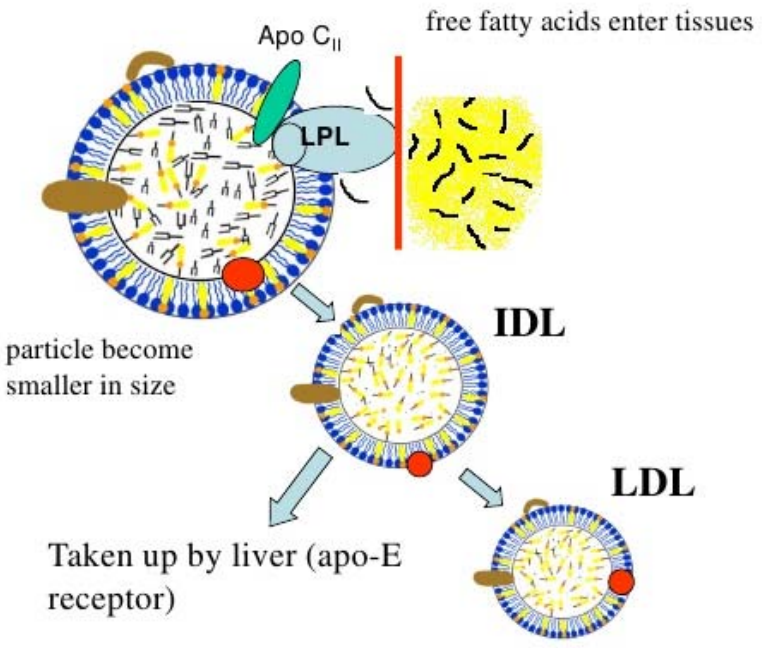
In the systemic circulation the chylomicrons encounter lipoprotein lipase, attached to the endothelial surfaces of capillaries in adipose tissue, heart, and skeletal muscle. The triglyceride of chylomicrons are hydrolyzed by lipoprotein lipase (LPL) and free fatty acids are released. ApoC-2 which is transferred to circulating chylomicrons from HDL, acts as a required co factor for LPL in this reaction. The released free fatty acids are taken up by the adjacent myocytes or adipocytes and are either oxidized to generate energy or re-esterified and stored as triglyceride. As the process gets repeated (hydrolysis of the hydrophobic core) the chylomicron particles progressively shrink in size, creating chylomicrons remnants.

Chylomicron remnants are rapidly removed from the circulation by the liver through a process that requires apoE as a ligand for receptors in the liver. Consequently, few, if any, chylomicrons or chylomicron remnants are generally present in the blood after 12hr-fast, except in patients with certain disorders of lipoprotein metabolism.

Transport of hepatically derived lipids by VLDL and LDL

Another key role of lipoproteins is the transport of hepatic lipids from the liver to the periphery. VLDL resemble chylomicrons in protein composition except that they contain apoB-100 rather than apoB-48 and have a higher ratio of cholesterol to triglyceride. After secretion into the plasma, VLDL acquires multiple copies of apolipoproteins of C and E series by transfer from HDL. As with chylomicrons the triglycerides of VLDL are hydrolyzed by LPL,

especially in muscle, heart and adipose tissue. After the VLDL remnants dissociate from LPL, they are referred to as IDLs and they contain roughly the same amounts of cholesterol and triglycerides. The liver removes approximately 40-60% of IDL by LDL receptor mediated endocytosis via binding to apoE. The remainder of IDL is remodeled by hepatic lipase to form LDL (during the process of remodeling phospholipids and triglycerides in the particle are hydrolyzed, and all lipoproteins except apoB-100 are transferred to other lipoproteins). Approximately 70% of LDL is removed from the circulation by the liver in a similar manner as IDL; however in this case apoB binds with the LDL receptor, rather than apoE.



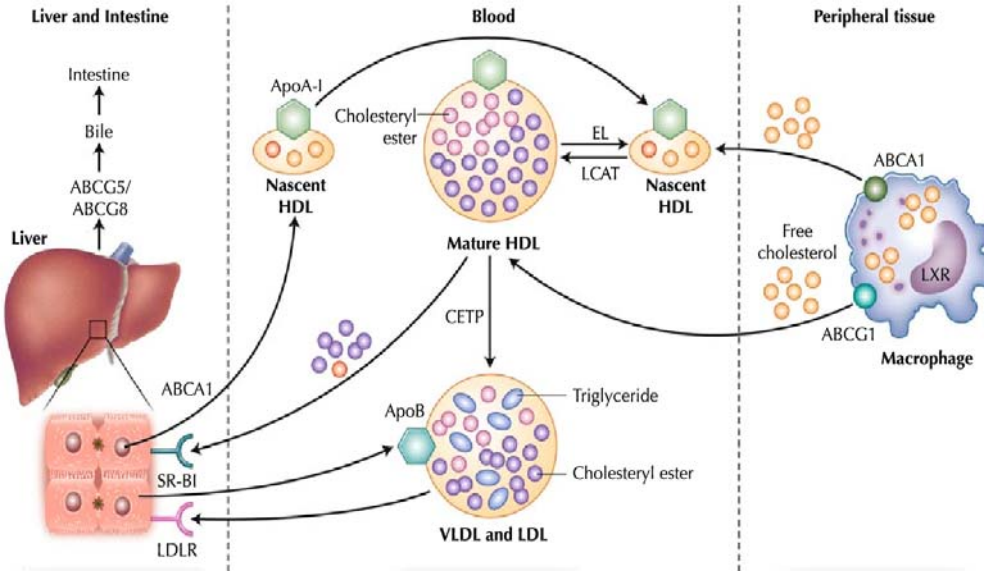
HDL metabolism and reverse cholesterol transport

Though all nucleated cells can synthesize cholesterol, only hepatocytes and enterocytes can effectively excrete cholesterol from the body- into either the bile or the gut lumen. In the liver cholesterol is secreted into the bile either directly or after conversion into bile acids. Cholesterol in peripheral cells is transported to the liver and intestine by a process termed reverse cholesterol transport, that is facilitated by HDL.

Nascent HDL particles are synthesized by the liver and intestines. Newly secreted apoA-1 rapidly acquires phospholipids and unesterified cholesterol from its site of synthesis via efflux promoted by the membrane protein ATP binding cassette protein A1 (ABCA1). This process results in the formation of discoidal HDL particles, which then recruit additional unesterified cholesterol from cells or circulating lipoproteins. Within the HDL particle, the cholesterol is esterified by lecithin cholesterol acyl transferase (a plasma enzyme associated with HDL) and the more hydrophobic cholesteryl ester moves to the core of the HDL particle. As HDL acquires more cholesteryl esters, it becomes spherical and additional apolipoproteins and lipids are transferred to the particles from the surfaces of chylomicrons and VLDLs during lipolysis.

HDL cholesterol is transported to hepatocytes by both an indirect and a direct pathway. HDL cholesteryl esters can be transferred to apoB containing lipoproteins in exchange for triglycerides by the cholesteryl ester transfer

protein. The cholesteryl esters are then removed from the circulation by LDL receptor mediated endocytosis. HDL cholesterol can also be taken up directly by hepatocytes via the scavenger receptor class B1, a surface receptor that mediates the selective transfer of lipid to cells.



FUNCTIONS OF HDL CHOLESTEROL

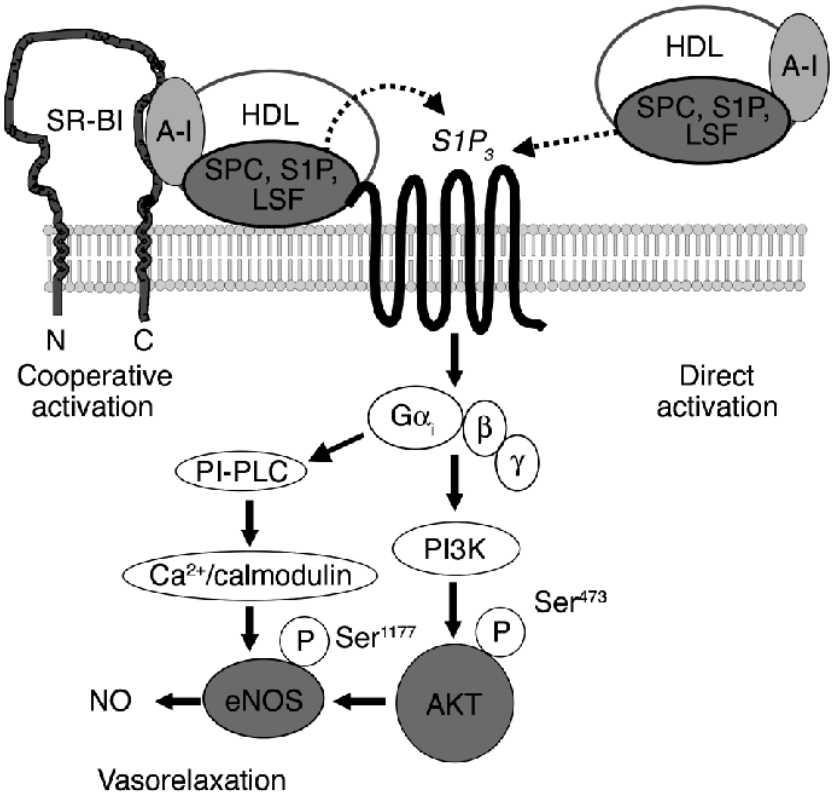
1.Cellular cholesterol efflux

HDL particles remove cholesterol from the cells of the arterial wall, primarily macrophages and macrophage-derived foam cells. This cholesterol efflux is responsible for the clinically relevant atheroprotective effect of HDL that forms the basis of the association between circulating levels of HDL-C and cardiovascular risk(1). Indeed, studies have shown that reduction of plasma HDL-C concentration may accelerate the development of atherosclerosis by impairing the clearance of cholesterol from the arterial wall.(2)

2. Vasodilatory actions

Apart from playing a key role in cholesterol efflux, HDL has other anti-atherogenic properties. The beneficial effect of HDL on the endothelium includes the primary vasodilatory property, which is attributed to its capacity to stimulate NO release by endothelial cells (3) and increase NO bioavailability.

Activation of NO production by HDL involves binding of the HDL particles to SR-BI as an initiating event (4). HDL bound to the extracellular loop of SR-BI initiates signaling in the endothelium that ultimately results in the activation of endothelial nitric oxide synthase (eNOS).(5)



Another pathway participating in vasodilatory effects of HDL in endothelial cells is mediated by ABCG1 and involves efflux of cholesterol and 7-oxysterols which improves the formation of active eNOS dimers and results in decreased ROS production. Diminished cellular production of superoxide, which inactivates NO, can also increase in NO bioavailability and improved vasodilation in the presence of HDL.(6)

Vasodilatory activity of HDL may translate into improved endothelial function; clinical evidence to support this hypothesis is accumulating.

3. Cytoprotective action

HDL particles possess several cytoprotective properties.

HDL protects both macrophages and endothelial cells from apoptosis induced by loading with free cholesterol or by oxidized LDL. HDL-mediated protection from apoptosis induced by loading with free cholesterol or oxidized LDL is mediated by cellular efflux of oxidized cholesterols, primarily of 7-ketosterol (7) . Cytoprotection is also related to intracellular antioxidative actions of HDL, which include reduced cellular generation of superoxide anion and/or hydrogen peroxide secondary to down-regulation of superoxide production by NADPH oxidase and/or mitochondrial electron transport chain.(8)

Another cytoprotective activity of HDL relevant for vasoprotection involves inhibition of apoptosis, cell detachment, and extracellular matrix degradation induced by elastase in human vascular smooth muscle cells. Such an anti-elastase action can be related to the presence in HDL of serpin peptidase inhibitors, including alpha-1-antitrypsin.(9)

4. Anti-inflammatory and anti-oxidative actions

HDL particles display multiple anti-inflammatory actions which collectively contribute to suppression of a chronic inflammatory response in the arterial wall which evolves in response to LDL-derived cholesterol deposition (10). HDL potently decreases adhesion molecule expression on endothelial

cells activated by cytokines and thereby inhibits monocyte adhesion to the endothelium both *in vitro* and *in vivo*. HDL also directly inhibits monocyte activation, reducing expression of chemokines and chemokine receptors via the modulation of nuclear factor kappa B (NFkB) and PPAR gamma.

HDL particles also inhibit stimulation of T-cells by antigen-presenting cells and activation of monocytes by stimulated *T*-cells, thereby inhibiting the production of proinflammatory cytokines and chemokines induced by *T*-cell contact with monocytes. Interaction of HDL with *T*-lymphocytes, which blocks subsequent activation of monocytes by lymphocytes, can account for this effect. HDL can also restore the emigratory process of macrophages and monocyte-derived dendritic cells and thus result in the resolution of inflammatory reactions in atherosclerotic plaques In addition, HDL potently reduces neutrophil activation *in vivo* and *in vitro*. Collectively, these effects constitute an important facet of HDL action on the innate and adaptive immunity.

The multiple effects of HDL on the immune system suggest that several mechanisms of action may be operative. Cellular efflux of non-oxidized and oxidized lipids may form a mechanistic basis for the capacity of HDL to decrease adhesion molecule expression, for direct inhibitory actions of HDL on monocyte and neutrophil activation, and for reductions in myeloid cell proliferation and monocytosis.

Antioxidative properties of HDL are closely linked to its anti-inflammatory potential. Indeed, oxidative modifications of cholesterol-rich lipoproteins, primarily LDL, retained in the arterial wall result in the formation of highly pro inflammatory oxidized phospholipids, such as 1-palmytoyl-2-(5-oxovaleroyl)-sn-glycero-3-phosphocholine and 1-palmitoyl-2-glutaroyl-sn-glycero-3-phosphocholine. The response-to-retention hypothesis of atherosclerosis postulates that these and other products of LDL oxidation, acting together, induce local inflammatory response. HDL-mediated protection of LDL from pathological oxidation may therefore result in the inhibition of inflammation.

HDL can protect LDL and other lipoproteins from oxidative stress *in vitro* induced by various oxidants, which include one- and two-electron species. Removal of oxidized lipids from LDL or cells represents the first step of HDL-mediated protection from oxidative damage induced by free radicals. Indeed, phospholipid hydroperoxides (PLOOHs) are rapidly transferred from oxidized LDL to HDL upon their co-incubation.

Inactivation of oxidized lipids associated with HDL particles represents the second step in this antioxidative pathway. Depending on their structure, oxidized lipids can be reduced (LOOHs) by apoA-I and other redox-active HDL components or hydrolysed (short-chain oxidized phospholipids, lysophosphatidylcholine) by HDL-associated hydrolytic enzymes.

As a consequence, HDL particles constitute a major transport vehicle of LOOH in human plasma and may therefore function as a 'sink' for oxidized lipids which can accumulate in the particle when the LOOH-inactivating capacity of HDL is overwhelmed. Subsequently, CEOOHs and their corresponding hydroxides can be rapidly removed from HDL via selective uptake by the liver mediated by SR-BI.

HDL also inhibits generation of reactive oxygen species (ROS) and decreases intracellular oxidative stress both *in vitro* and *in vivo*. Such antioxidative actions of HDL require interaction with surface receptors, including SR-BI and ABCG1, but do not necessitate direct contact between HDL and prooxidative agents in the extracellular compartment. Diminished cellular production of superoxide and/or hydrogen peroxide may be implicated in the antioxidative effect of HDL in endothelial cells.

5. Protection from infection

Plasma HDL displays several anti-infectious activities which may contribute to the innate immunity. Indeed, HDL binds circulating LPS and participates in its hepatic clearance to the bile, thereby inhibiting LPS-induced cellular activation and endotoxic shock in animal models.(10)

Furthermore, human plasma HDL is a major carrier of specific trypanosome lytic activity, which selectively protects humans from *Trypanosoma brucei brucei*, a parasitic species that causes sleeping

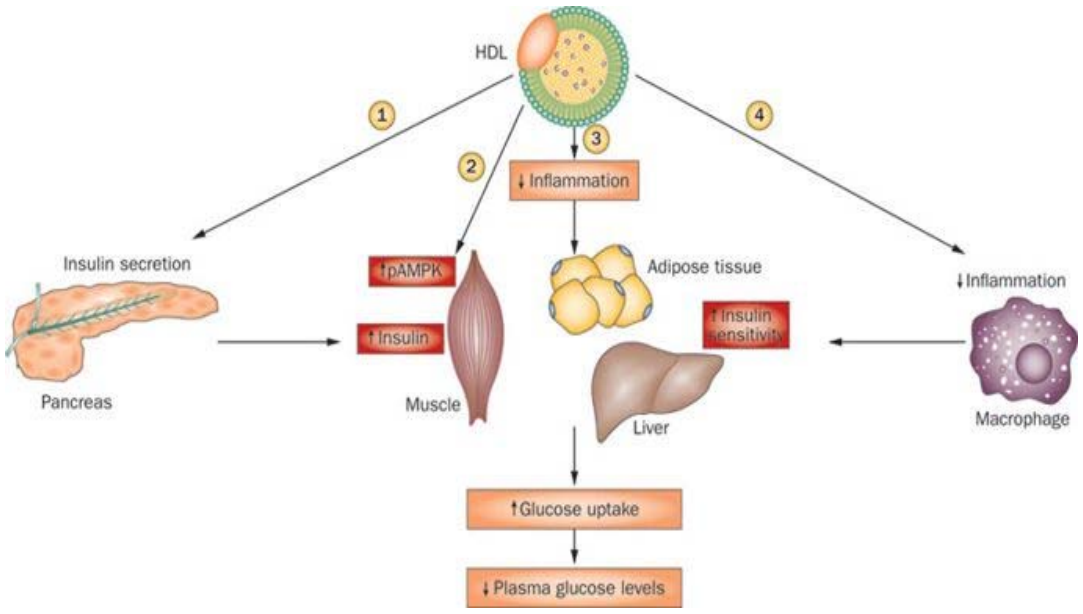
sickness. Trypanosome lytic factor constitutes a minor subpopulation of HDL particles, which is characterized by the ability to kill *T.b. brucei.* HDL-mediated killing of *T. b. brucei* occurs through a unique mechanism of ionic pore formation in endosomal membranes of the parasite.(11)

6. Modulation of glucose metabolism

Human plasma HDL efficiently improves glucose metabolism by multiple mechanisms (12) (13) which include enhanced insulin secretion by pancreatic beta-cells, improved insulin sensitivity and maintenance of cholesterol homeostasis. Indeed, infusions of reconstituted HDL reduce plasma glucose and increase plasma insulin when compared with placebo in patients with Type 2 diabetes. *In vitro* HDL accelerates insulin secretion beta-cells via a mechanism that involves removal of excess cholesterol through ABCA1 (14). Another pathway contributing to improved insulin sensitivity under the action of HDL includes protection of pancreatic beta-cells from apoptosis. Activation of intracellular survival pathways, which underlies this effect, involves altered expression of inducible nitric oxide synthase, Fas, and FLICE-like inhibitory protein (FLIP).

HDL equally improves insulin sensitivity at the level of skeletal muscle (15). Specifically, HDL activates the AMPK pathway via elevated phosphorylation of acetyl-CoA carboxylase 2 (ACC-beta) and increases glucose uptake by cultured skeletal muscle cells. This effect involves binding to

ABCA1 and activation of calcium/calmodulin-dependent protein kinase kinase.



7. Reduction of platelet activation

HDL exerts several anti-thrombotic effects which primarily include reduction of platelet activity (16). *In vitro* anti-thrombotic activities of HDL are expressed as dose-dependent inhibitory actions on agonist-stimulated platelet aggregation, fibrinogen binding, granule secretion, and thromboxane A2 and 12-hydroxy-eicosatetraenoic acid production (17). HDL decreases platelet aggregation mediated by glycoprotein IIb/IIIa in response to thrombin, collagen, ADP, and adrenaline. (18)

The above action is brought about by the following mechanism. Interaction of HDL with the platelet SR-BI receptor may trigger intracellular

signaling cascades, which encompass intracellular release of diacylglycerol from plasma membrane phosphatidylcholine, activation of protein kinase C, stimulation of the Na^+/H^+ antiport, alkalization of the cytoplasm, and inhibition of calcium release from storage sites (19).

In addition to its effects on platelets, HDL exerts anti-thrombotic effects on endothelial cells. The anti-thrombotic effects of HDL towards endothelial cells depends on the stimulation of cellular NO production, which is in turn triggered by the interaction of HDL with platelet SR-BI.

Apart from this HDL may also inhibit factors which promote blood coagulation, including tissue factor and factors X, Va, and VIIIa (20).

9. Regulation of gene expression by miRs

Recently, HDL has been shown to transport small non-coding miRs. Multiple copies of several miRs are transported by circulating HDL in man. miRs are key intracellular regulators of gene expression which post-transcriptionally control cellular cholesterol homeostasis, including cholesterol efflux. HDL carries miRs that control cholesterol metabolism; miR-33 down-regulates expression of ABCA1 and ABCG1 and reduces HDL biogenesis in mice (21). As a corollary, both antagonism and deficiency of miR-33 raise circulating HDL-C levels, enhance macrophage cholesterol efflux, and prevents progression of atherosclerosis. Such anti-atherosclerotic effects suggest

antagonism and down-regulation of miR-33 as a novel strategy for atheroprotection.

10. Cholesterol handling in the brain

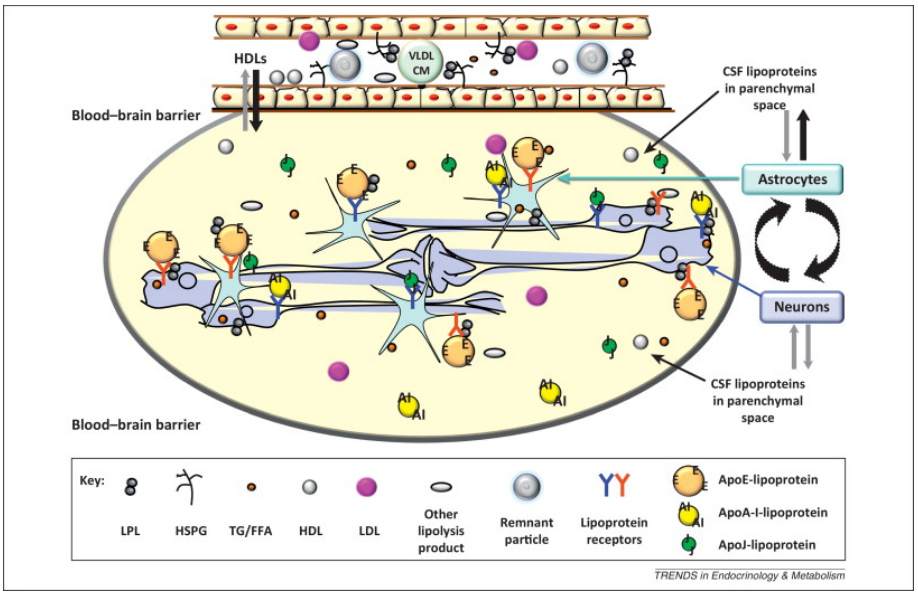
Brain relies heavily on cholesterol supply which is essential for cell membrane synthesis and myelin production. Strikingly, while the central nervous system accounts for only 2.1% of body weight, it contains 23% of the whole body cholesterol pool. Cholesterol transport mediated by lipoproteins has long been thought to underlie the proper functioning of the brain. Indeed, all major types of neuronal cells can bind and internalize lipoproteins present in the extracellular fluid.

Brain lipoproteins appear to be similar to those of cerebrospinal fluid, as a consequence of the passage between the two compartments.

Human cerebrospinal fluid primarily contains spherical lipoproteins of approximately 10–22 nm in diameter with a density of 1.063–1.25 g/mL, which corresponds to the density of HDL and very-HDL of human plasma (22). Lipoprotein concentrations in the cerebrospinal fluid are, however, several hundred-fold lower when compared with the plasma compartment.

ApoE and apoA-I are the major apolipoproteins in human CSF, with slightly higher concentration of the former. ApoE- and cholesterol-rich lipoproteins secreted by astrocytes appear to ensure a continuous supply of cholesterol required for normal functioning of neurons (23).

In addition, HDL obtained from cerebrospinal fluid efficiently effluxes cholesterol from neuronal cells. Similar to the plasma compartment, metabolism of brain lipoproteins is regulated by ABCA1, ABCG1 and other proteins known to participate in plasma lipoprotein metabolism. Available data indicate that metabolism of brain lipoproteins can be impaired in Alzheimer's disease, as occurs in the presence of apolipoprotein E4, the primary genetic risk factor for the sporadic disease (24).



FACTORS INFLUENCING HDL LEVELS(25)

EXERCISE

It has been established that regular aerobic exercise increases the HDL cholesterol level by 3 to 9 percent in healthy individuals, irrespective of the age group. This increase is linked to the frequency and intensity of physical activity. It is expected that with frequent, low-intensity exercise (e.g., five 30-

minute sessions per week vs. three 60-minute sessions) there would be greater increase in HDL cholesterol .However, there is little evidence that walking significantly increases HDL cholesterol levels. HDL cholesterol levels may increase with as little as eight weeks of regular exercise, although changes may not be evident for two years. Exercise may increase HDL cholesterol levels by stimulating the production of pre beta HDL cholesterol and reverse cholesterol transport. Regular exercise yields a greater increase in HDL cholesterol in men with low HDL cholesterol levels, elevated triglyceride levels, and abdominal obesity than in those with isolated low HDL cholesterol levels.

Weight loss may be crucial for an increase in HDL cholesterol to occur. In one randomized, controlled trial, persons who walked or jogged 10 miles per week but did not lose weight did not have different HDL cholesterol levels from those of controls. Nevertheless, it is reasonable to recommend a program of regular, brisk aerobic exercise for 30 minutes on most days of the week.

SMOKING CESSATION

Cigarette smoking is associated with reduced HDL cholesterol, lecithin–cholesterol acyltransferase activity and cholesteryl-ester–transfer protein (CETP) activity.

After smoking cessation, HDL cholesterol increases (by a mean of 4 mg per deciliter [0.10 mmol per liter]), more so in women than in men and in

persons with elevated baseline HDL cholesterol levels (>47 mg per deciliter [1.21 mmol per liter]).

It is necessary to recommend a comprehensive approach (involving pharmacotherapy, nicotine replacement, and counseling) for smoking cessation.

WEIGHT CONTROL

Reduced HDL cholesterol levels and elevated serum triglyceride levels are associated with obesity. A negative correlation exists between HDL cholesterol and body-mass index. A meta-analysis examining the effect of weight loss on HDL cholesterol levels demonstrated that the levels increased by 0.35 mg per deciliter (0.009 mmol per liter) per kilogram of weight reduction in subjects who achieved a stabilized, reduced weight ($P \leq 0.01$) but decreased by 0.27 mg per deciliter (0.006 mmol per liter) in subjects during active weight loss ($P \leq 0.05$).In subjects who maintained a stable weight for six weeks after weight loss, HDL cholesterol levels, lipoprotein lipase levels, and lecithin– cholesterol acyltransferase activity increased; these increases may contribute to enhanced cholesterol esterification and reverse cholesterol transport. A reasonable weight-loss goal for overweight or obese patients is 1 lb (0.45 kg) per week, with a target body-mass index of less than 25.

ALCOHOL INTAKE

Moderate alcohol consumption raises HDL cholesterol levels. A meta-analysis indicated that the consumption of 30 g (1 fluid oz) of alcohol per day

increases HDL cholesterol levels by a mean of 4 mg per deciliter. This has been observed irrespective of the kind of alcohol consumed. Persons who consume one to three drinks daily have higher HDL cholesterol levels and a lower risk of myocardial infarction than do those who drink less, even after adjustment for other likely confounding factors. Thus, for many persons with low HDL mild-to-moderate alcohol consumption (no more than one to two drinks per day) appears to be reasonable. However, the potential risks associated with this recommendation may outweigh the benefits in persons with hepatic dysfunction or the potential for addiction. Alcohol consumption may elevate HDL cholesterol levels by increasing cellular cholesterol efflux and plasma cholesterol esterification.

DIETARY FAT INTAKE

Reduction in the intake of dietary fat leads to decline in plasma LDL and HDL levels. In a study comparing calorically balanced diets (i.e., intake is equal to expenditure) that differed in fat content, subjects who consumed a low-fat diet (19 percent of total calories were from fat) had lower HDL cholesterol and apolipoprotein A-I levels than did subjects who were fed a high-fat diet (50 percent of total calories were from fat) (54 mg per deciliter [1.40 mmol per liter] vs. 63 mg per deciliter [1.63 mmol per liter] and 118 mg per deciliter [3.05 mmol per liter] vs. 127 mg per deciliter [3.28 mmol per liter], respectively; $P<0.005$). The concomitant decrease in LDL cholesterol

that occurs with a diet low in saturated fat may override the effects associated with the decline in HDL cholesterol.

Native Alaskan population that eat a diet rich in n–3 polyunsaturated fatty acids have high HDL cholesterol levels. Although a diet high in monounsaturated fats does not elicit a significant change in HDL cholesterol levels, the dietary glycemic load (which represents the equivalent elevating effect on blood-glucose levels of 1 g of pure glucose or white bread) is negatively correlated with HDL cholesterol levels. Thus, a diet rich in n–3 polyunsaturated fatty acids — sources include oils (olive, canola, soy, flaxseed), nuts (almonds, peanuts, walnuts, pecans), cold-water fish (salmon, mackerel), and shellfish — with limited carbohydrates can be recommended to increase serum HDL cholesterol levels.

LIFESTYLE AND MODIFYING FACTORS

Improvement in HDL cholesterol levels associated with exercise, moderate alcohol consumption, and weight loss is greatest in persons with the highest baseline HDL cholesterol levels (60 mg per deciliter [1.55 mmol per liter] or more); those with low baseline levels have less improvement.

The magnitude of improvement in HDL cholesterol levels associated with lifestyle modifications is influenzed by the interactions between genes and the environment. Specifically, improvement with exercise may depend on individual CETP and endothelial lipase genotypes. However, genetic tests for

these factors are not currently used in routine practice, and lifestyle changes as described above should be recommended routinely, both to raise HDL cholesterol levels and to lower LDL cholesterol levels and improve other cardiovascular risk factors.

MEDICATION

Several classes of medications increase HDL cholesterol levels; these include niacin and fibrates, in particular, and, to a lesser degree, statins. These medications also lower triglycerides and LDL cholesterol levels. Without trials designed to isolate the effects of drugs on changes in HDL cholesterol levels and on coronary outcomes, it is difficult to determine how much of the drugs' benefit in reducing the rates of coronary heart disease are due to changes in HDL cholesterol levels. At present, there is no clear consensus regarding when to use medications for the purpose of raising HDL cholesterol levels, although drugs are most often considered for patients with established coronary disease or for those at high risk.

NIACIN

Niacin (nicotinic acid or vitamin B 3) is the most effective medication to raise HDL cholesterol levels, causing increases of 20 to 35 percent. The Coronary Drug Project demonstrated a significant reduction in the incidence of death and myocardial infarction after five years of niacin treatment among men with a history of myocardial infarction.

Niacin inhibits hepatic uptake of apolipoprotein A-I and increases plasma pre beta HDL cholesterol levels. Niacin therapy is associated with improved endothelial function and nitric oxide synthase activity.

The side effects of niacin therapy include cutaneous flushing, dyspepsia, and elevation of plasma glucose and uric acid levels. Flushing, which is largely mediated by prostaglandins, may be minimized with the use of an extended-release formulation of niacin (not the same as sustained-release niacin); with the concurrent consumption of a low fat snack at bedtime, 30 minutes after ingestion of an aspirin; and with a regimen that begins with a low dose (e.g., 500 mg each night) and increases gradually.

FIBRATES

Fibrate therapy results in an increase in HDL cholesterol levels of 10 to 25 percent. This is brought about by activating peroxisome-proliferator–activated receptor A (PPARa), which in turn results in increased expression of the hepatic apolipoprotein A-I gene.

The Helsinki Heart Study and the Veterans Affairs High-Density Lipoprotein Cholesterol Intervention Trial demonstrated increases in HDL cholesterol (10 percent and 6 percent, respectively) and a large reduction in triglyceride levels in asymptomatic men and those with primary dyslipidemia or coronary heart disease, respectively, who had been randomly assigned to receive gemfibrozil (1200 mg daily), as compared with subjects who received

placebo. Gemfibrozil significantly reduced the risks of coronary events (34 percent) and the combined outcome of coronary death, nonfatal myocardial infarction, and stroke (24 percent).

Subsequent analysis of the Veterans Affairs trial indicated that only the increase in HDL cholesterol levels, not the change in LDL cholesterol or triglyceride levels, significantly predicted a reduced risk of coronary events.

STATINS

In addition to lowering LDL cholesterol levels, statins raise HDL cholesterol levels by 2 to 15 percent by increasing apolipoprotein A-I synthesis.

In a randomized trial comparing the effects of simvastatin and atorvastatin (maximum dose, 80 mg and 40 mg, respectively, titrated to LDL cholesterol levels) among patients with elevated LDL cholesterol levels, there was a moderately greater increase with simvastatin in HDL cholesterol levels (9 percent vs. 7 percent, $P<0.001$) and apolipoprotein A-I levels (6 percent vs. 3 percent, $P<0.001$); clinical outcomes were not assessed. Several trials have documented reduced risks of major coronary events associated with the use of a statin, as compared with placebo, but it is not clear whether the rise in HDL cholesterol levels is an independent predictor of the reduced risk of coronary events with the use of these agents.

COMBINATION THERAPY

In some patients with low HDL cholesterol levels, various lipid-modifying medications may be useful in combination. The HDL Atherosclerosis Treatment Study (HATS) demonstrated that a combination of low-dose simvastatin (10 to 20 mg per day) and high-dose niacin (2 to 4 mg per day) significantly increased HDL cholesterol levels (26 percent), as compared with placebo, in patients with HDL cholesterol levels of 40 mg per deciliter or less, LDL cholesterol levels of 145 mg per deciliter (3.75 mmol per liter) or less, and triglyceride levels of less than 400 mg per deciliter (4.52 mmol per liter). In the simvastatin–niacin treatment group, coronary stenosis, as documented on angiography, moderately regressed over three years (0.4 percent, $P<0.001$ vs. placebo; 3.9 percent increase). The Arterial Biology for the Investigation of the Treatment Effects of Reducing Cholesterol (ARBITER) 2 study, which involved subjects who had established coronary disease, HDL cholesterol levels of less than 45 mg per deciliter (1.16 mmol per liter), and LDL cholesterol levels of less than 130 mg per deciliter (3.36 mmol per liter), showed that the addition of extended-release niacin (1000 mg daily) to existing statin therapy increased mean HDL cholesterol levels by 21 percent (from a mean of 39 to 47 mg per deciliter [1.01 to 1.21 mmol per liter]; $P<0.001$ vs. the change in the placebo group). Medial thickness of the carotid intima significantly increased in the placebo group (mean change, 0.044 mm; $P<0.001$) but not in the placebo group (mean change, 0.014 mm; $P=0.23$);

however, a comparison of changes in intimal thickness over time did not show a significant difference between the two groups (P=0.08).

HDL CHOLESTEROL IN ELDERLY

Worldwide the percentage and absolute number of elderly individuals are greater than ever before. With no historical example of such large and rapidly growing cohorts of older adults, the world is entering uncharted waters. Since 1950s, the number of older people as a share of the global population has increased very gradually, but projections indicate much sharper increase in the coming decades.

The United Nations population Division projects an increase in the number of individuals aged 60+ from about 901 million today (12% of the world population) to 2.1 billion by 2050 (21.5%).The number of oldest old individuals, defined as those aged 80+, is projected to triple from 125 million today (2% of the world population) to 434 million (4.5%) by 2050.

Population ageing (the dominant demographic trend of this century) and the burden of non-communicable diseases will usher in challenges in several areas. Thus proactive responses are needed to mitigate the burden posed by the greying society.

A long and healthy life is universally valued. However there is a considerable variation in the markers of ageing and age related diseases between individuals of a similar age. The starkest inequalities in later life are

how many years of life remain at an older age such as 65+ and how many years of that life remain free from disabilities that impede physical and cognitive functioning to the extent that they limit the sense of valuing one's life.

Several factors influence mental and physical sustainability in later life-birth weight, education, socioeconomic position and lifestyle (diet, physical activity, smoking) etc. The influence of modifiable factors on ageing has been under study in the recent years. Dyslipidemia is one among them.

The atheroprotective effect of HDL, through its reverse cholesterol transport, is well established. Therefore, having low HDL cholesterol concentration predisposes people to atherosclerotic disease and stroke, peripheral vascular disease, lower-extremity amputation, and loss of kidney function, which in turn impair physical performance.

HDL cholesterol not only improves physical performance but also is probably related to preserving the cognitive function, by preventing the formation of amyloid-beta in senile plaques, which constitutes one of the defining hallmarks of Alzheimer's disease (Olesen&Dagø, 2000).

This association of HDL with physical performance and cognition has been evaluated in several studies. To quote a few,

- In the ilSIRENTE study (26), conducted in Central Italy, the relationship of HDL-cholesterol with physical performance in community dwelling older persons aged 80 years and above was studied.

The participants of the study were grouped into three tertiles based on their HDL levels. The parameters like physical performance (SPPB), muscle strength (hand grip), functional status (IADL) and blood measurements (serum albumin, cholesterol (HDL and LDL), C-reactive protein and urea) were assessed in each group and a comparison was drawn. Compared with the participants with the first tertile of HDL-cholesterol, those in the third tertile were younger, more likely to be women and had a higher prevalence of osteoarthritis. On the contrary, subjects in the third tertile of HDL-cholesterol had a lower prevalence of congestive heart failure, diabetes and cerebrovascular diseases, and tended to be more physically active, compared with the other participants. Albumin, urea and LDL-cholesterol increased as HDL cholesterol increased, while C-reactive protein declined with increasing HDL-cholesterol tertiles.

In the unadjusted analysis physical function (as measured by the 4-m walking speed, the SPPB score, the Basic and IADL scales scores), but not hand-grip strength, improved significantly as HDL-cholesterol tertiles increased. After adjustment for potential confounders, which included age, gender, living alone, alcohol abuse, physical activity, congestive heart failure, diabetes, cerebrovascular diseases, osteoarthritis, albumin, urea, C-reactive protein, and LDL cholesterol, the association of HDL-cholesterol tertiles with the 4-m walking speed and the SPPB score was still consistent. The results showed that, in

older persons, physical function declines as serum HDL-cholesterol levels decline. This association was consistent in very old participants and in both gender groups.

- In a study published in Japan Geriatrics Society in 2011 (27), the association between HDL-C levels and physical and cognitive performance was analyzed. Functional status was determined by the Lawton–Brody Index (LI) for instrumental activities of daily living (IADL) and the Barthel Index (BI) for basic activities (BADL).Cognition was assessed using the Spanish version of the Mini-Mental State Examination (MMSE). As per the study results, normal HDL-C levels at baseline were significantly associated with higher BI scores ($P < 0.006$) and a lower number of prescription drugs used ($P < 0.04$). However there was no significant association between HDL levels and MMSE.

- In a study published in the Archives of Gerontology and Geriatrics 71 (28) the association between HDL cholesterol and functional state in the elderly Korean population was studied. Herein, elevated HDL cholesterol concentration was found to be strongly associated with higher MMSE score, ADL scale, and Chair RiseTest speed.

- Yet another study was conducted among Chinese elderly(29) to investigate the association of cholesterol level with cognitive performance. A total of 2000 elderly aged 65 years and over participated in this study. Total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C) and high density lipoprotein cholesterol (HDL-C) concentration were determined and cognitive impairment was defined as Mini-Mental State Examination (MMSE) score≤23.The results of the study showed a significant positive linear association between cholesterol levels and MMSE score in linear regression models. These findings suggest that cholesterol levels within the high normal range are associated with better cognitive performance in Chinese elderly, specifically in the oldest old. With further validation, low cholesterol may serve a clinical indicator of risk for cognitive impairment in the elderly.

- CSI-MCI study, conducted in China in 2016 (30), was a preliminary case-control study of the association of plasma lipids/lipoproteins with MCI in 112 MCI cases and 115 cognitively normal controls. Plasma total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG) levels were measured in fasting blood samples. Multivariable logistic regression was used to evaluate the potential association between MCI and these factors. Statistical models were adjusted for multiple demographic and biological covariates. The results

of this study showed that plasma HDL level was significantly negatively associated with the risk of MCI. There was no association between plasma LDL level and the risk of MCI, adjustment for demographics, vascular disorders did not change this relation.

MATERIALS AND METHODS

STUDY CENTRE:

Geriatric Medicine OPD & ward (male & female),

Rajiv Gandhi Government General Hospital, Chennai- 3.

ETHICAL COMMITTEE APPROVAL:

Approved by the Institutional Ethical Committee of Madras Medical College, Chennai

STUDY DESIGN :

Cross sectional observational study (hospital based)

PERIOD OF STUDY :

1year (August 2017- July 2018)

SAMPLE SIZE :

150 patients

INCLUSION CRITERIA :

Persons aged above 60 years willing to consent for the study

EXCLUSION CRITERIA :

Critically ill patients like advanced cardiac failure, stage 4,5 CKD.

Acute stroke and previous stroke with major residual deficits.

Patients with severe OA knee unable to perform SPPB

Patients unable to comprehend simple instructions.

Patients not willing to consent for the study.

DETAILS OF THE STUDY

As per the above mentioned inclusion and exclusion criteria, people aged 60 years and above attending Geriatric medicine OPD of Rajiv Gandhi Govt. General Hospital, Chennai were selected by Simple random sampling. After getting informed written consent from all the study participants, basic information such as name, age, gender, educational status and contact details were obtained. Then history regarding the presence of comorbid illnesses like hypertension, diabetes mellitus, COPD/bronchial asthma, CAD, CVA was collected. Their habitual history like smoking and alcohol intake were also made note of.

Then anthropometric measures like height, weight and BMI were calculated for the subjects and blood samples obtained for lipid profile.

Based on the HDL levels patients were stratified into three tertiles (<40mg/dl; 40-49mg/dl; >49mg/dl). The physical performance and cognitive function of subjects in each group was assessed using short physical performance battery score (SPPB) and mini mental status examination (MMSE) respectively.

SHORT PHYSICAL PERFORMANCE BATTERY (SPPB)

The short physical performance battery (SPPB) is a group of measures that combines the results of the gait speed, chair stand and balance tests (Guralnik et al., 2000). It has been used as a predictive tool for possible disability and can aid in the monitoring of functional status in older people. The scores range from 0 (worst performance) to 12 (best performance). The SPPB has been shown to have predictive validity showing a gradient of risk for mortality, nursing home admission, and disability.

Equipments Required

Chair with arms

Stopwatch

Measuring tape

Procedure

1.REPEATED CHAIR STANDS

Explain “I want to see how long it takes for you to stand up and sit down as quickly as possible 5 times without stopping. After standing up each time, sit down and then stand up again. Keep your arms folded across your chest. Please watch while I demonstrate. I’ll be timing you with a stopwatch”

DEMONSTRATE TO THE PATIENT.

Ask the participant if they are ready. If so, begin timing as soon as they bend forward at the hips. Count out loud the number of sits the participant has

performed. Stop the stop watch when they have sat down having completed the 5th stand. Also stop if the participant starts to use their arms, or after 1 minute they have not completed the test. Stop if the participant cannot complete 5 rises, and if you are concerned about the participant's safety. Record the number of seconds and the presence of imbalance.

Scoring the Repeated Chair Test

Unable to complete 5 chair stands or

Completes stands in >60 sec : ❒0 points

Chair stand time - 16.70 sec or more: ❒1 points

Chair stand time - 13.70 to 16.69 sec : ❒2 points

Chair stand time - 11.20 to 13.69 sec: ❒3 points

Chair stand time - 11.19 sec or less: ❒4 points

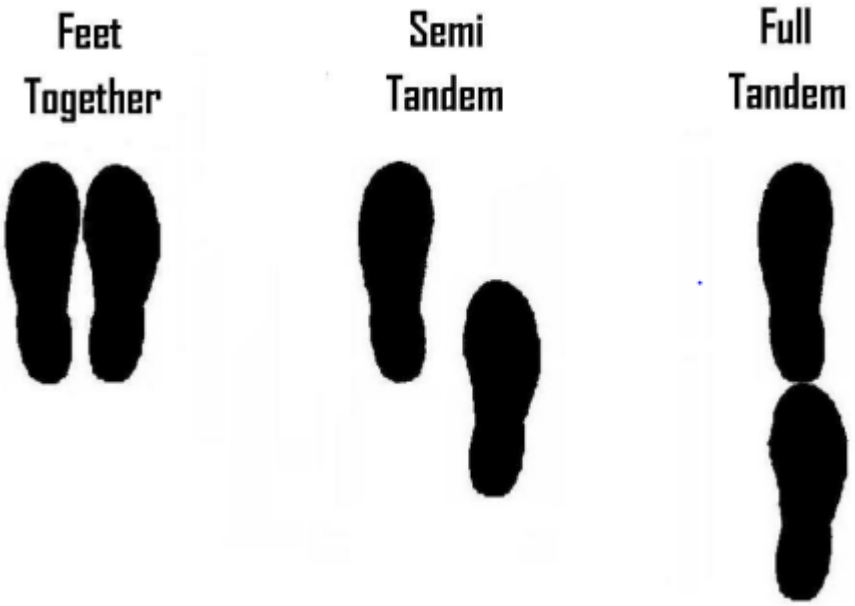
2. BALANCE TESTING

Explain "We will now look at your standing balance. We want to know if you can stand unsupported for 10 seconds with your feet in a certain position".

Explain "Begin with feet together beside each other. I want you to try to stand with your feet together, side by side, for about 10 seconds. Please watch while I demonstrate. You may use your arms, bend your knees, or move your body to maintain your balance, but try not to move your feet. Try to hold this position until I tell you to stop". Stand next to the participant to help him or her

into the side-by-side position. Allow participant to hold onto your arms to get balance. Begin timing when participant has feet together and let go of your arm. If they are able to complete 10 seconds progress to semi-tandem stand.

Repeat in semi tandem stand (heel of one foot placed by the big toe of the other foot). Explain "Now I want you to try to stand with the side of the heel of one foot touching the big toe of the other foot for about 10 seconds. You may put either foot in front, whichever is more comfortable for you. Please watch while I demonstrate". Begin timing when participant has feet in position and let's go of your arm. The test is stopped when the participant moves their feet, grasps the interviewer for support, or when 10 seconds has elapsed. Record time on Case Report Form If they are able to complete 10 seconds progress to tandem stand.



Tandem Stand (feet directly in front of each other) Explain "Now I want you to try to stand with the heel of one foot in front of and touching the toes of the other foot for 10 seconds. You may put either foot in front, whichever is more comfortable for you. Please watch while I demonstrate". Stand next to the participant to help him or her into the side-by-side position. Allow participant to hold onto your arms to get balance. Begin timing when participant has feet together and let's go of your arm.

SCORING:

Side-by-Side stand

Held for 10 sec ❒1 point

Not held for 10 sec ❒0 points

Not attempted ❒0 points

If 0 points, end Balance Tests

Semi-Tandem Stand

Held for 10 sec ❒1 points

Not held for 10 sec ❒0 points

Not attempted ❒0 points

If 0 points, end Balance Tests

Tandem Stand

Held for 10 sec ❒2 point

Held for 3 to 9.99 sec ❒1 points

Held for < than 3 sec ❒0 points

Not attempted ❒0 points

Total Balance Tests score _____ (sum points)

3. GAIT SPEED:

Mark out the distance with a tape measure and put a cone at either end. Place a chair at the other end if you think the participant might require it. Explain "This is our walking course. If you use a walking aid when walking outside your home, please use it for this test. I want you to walk at your usual pace between the two cones Walk all the way past the cone before you stop. I will walk behind you. We will be doing this test two times".

For 4-Meter Walk:

If time is more than 8.70 sec: ❒ 1 point

If time is 6.21 to 8.70 sec: ❒ 2 points

If time is 4.82 to 6.20 sec ❒ 3 points

If time is less than 4.82 sec: ❒ 4 points

Scoring for SSPB

Total Balance Test score _____ points

Gait Speed Test score _____ points

Chair Stand Test score _____ points

Total Score _____ points (sum of points above)

MINI MENTAL STATUS EXAMINATION

(Tamil version)

The Mini Mental State Examination (MMSE) is a tool that can be used to systematically and thoroughly assess mental status. It is an 11-question measure that tests five areas of cognitive function: orientation, registration, attention and calculation, recall, and language. The maximum score is 30. A score of 23 or lower is indicative of cognitive impairment.

ORIENTATION

Time: Year Season Month Date Time /5

Place: Country Town District Hospital Floor /5

REGISTRATION

Examiner names three objects (e.g. apple, table, penny) and asks the patient to repeat (1 point for each correct) /3

ATTENTION AND CALCULATION

Subtract 7 from 100, then repeat from result. Continue five times: 100, 93, 86, 79, 65. /5

RECALL

Ask for the names of the three objects learned earlier /3

LANGUAGE

Name two objects (e.g. pen, watch). /2

Repeat “No ifs, ands, or buts” /1

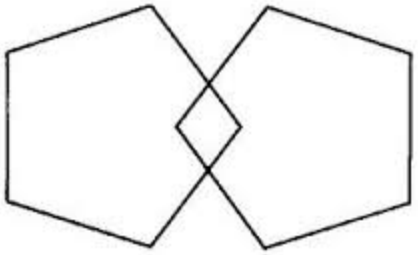
Give a three-stage command.

Score 1 for each stage. (e.g. “Place index finger of right hand on your nose and then on your left ear”). /3

Ask the patient to read and obey a written command on a piece of paper. The written instruction is: “Close your eyes”. /1

Ask the patient to write a sentence. Score 1 if it is sensible and has a subject and a verb. /1

COPYING: Ask the patient to copy a pair of intersecting pentagons /1



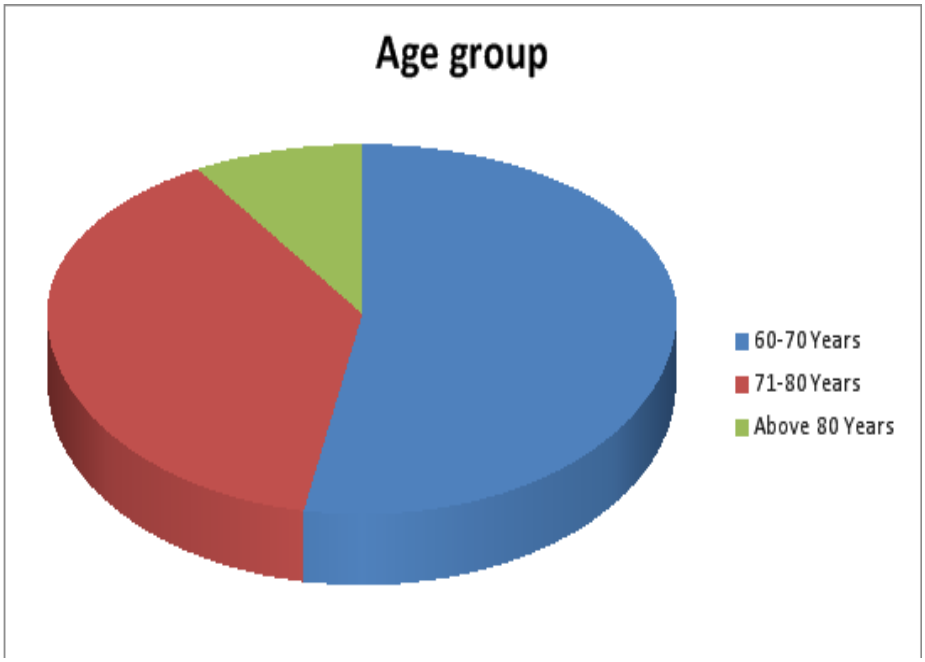
TOTAL SCORE

RESULTS

AGE

Age group	Frequency	Percent
60-70 Years	79	52.7
71-80 Years	56	37.3
Above 80 Years	15	10.0
Total	**150**	**100.0**

Of the 150 participants – 52.7% (n=79) were young old, 37.3% (n=56) old old and the remaining 10% (n=15) belonged to the oldest old category.



In this cross-sectional study, there was no significant association between HDL levels and age.

			Age group			Total
			60-70 Years	71-80 Years	Above 80 Years	
group -	<40mg/dl	Count	27	19	4	50
		% within group	54.0%	38.0%	8.0%	100.0%
	40mg/dl - 49mg/dl	Count	22	20	8	50
		% within group	44.0%	40.0%	16.0%	100.0%
	>49mg/dl	Count	30	17	3	50
		% within group	60.0%	34.0%	6.0%	100.0%
Total		Count	79	56	15	150
		% within group	52.7%	37.3%	10.0%	100.0%

Pearson Chi-Square=4.291 p=0.368

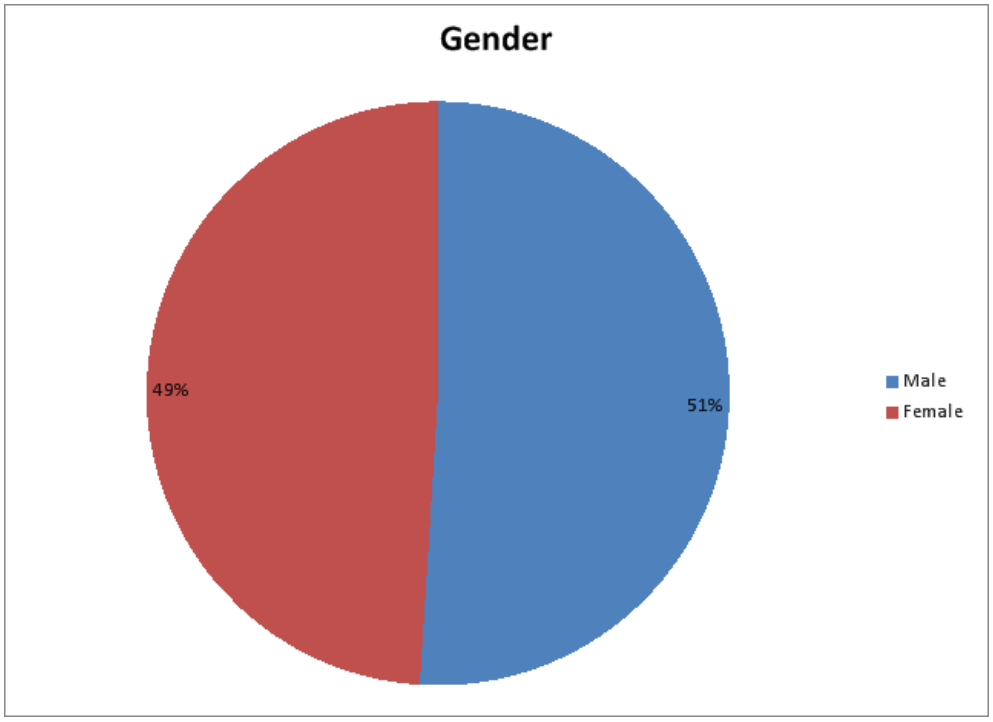
Since results are quite variable in the literature, more prospective studies are recommended to better define the causes and consequences of cholesterol and lipoprotein changes in old age.

GENDER

Gender	Frequency	Percent
Male	77	51.3
Female	73	48.7
Total	**150**	**100.0**

77 males participated in the study as against 73 females.

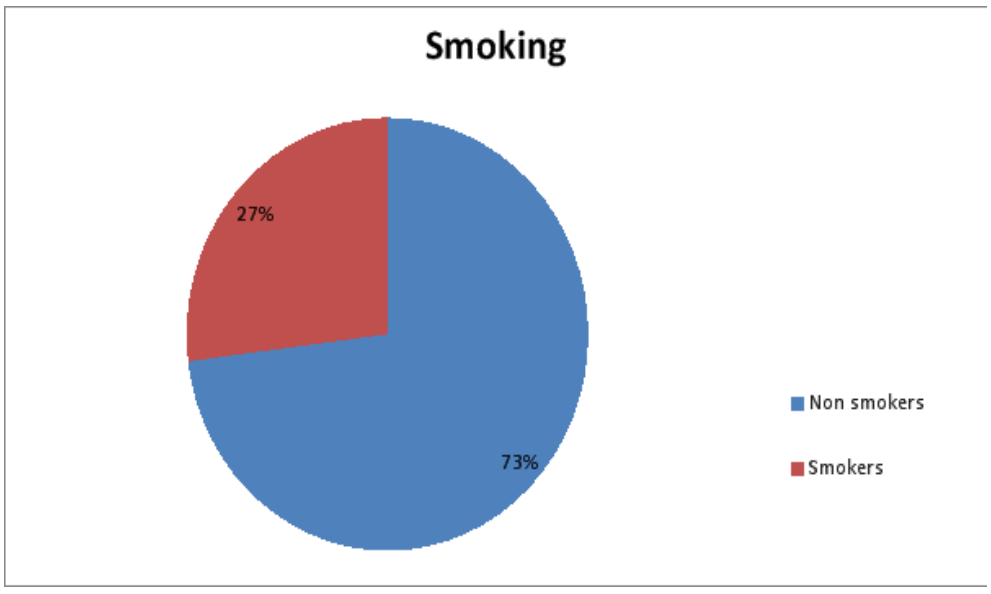
Thus the sex distribution in the study was almost equal.



SMOKING

SMOKING	FREQUENCY	PERCENT
Non- smokers	109	72.7
Smokers	41	27.3
Total	**150**	**100.0**

Of the 150 patients who participated in the study, the majority (72%) were nonsmokers (i.e) did not smoke more than 100 cigarettes in their life-time.



Smoking cigarettes is associated with decreased HDL cholesterol level, lecithin–cholesterol acyltransferase activity and cholesteryl-ester–transfer protein (CETP) activity.

But in this study, there was no significant association found between HDL levels and cigarette smoking. This could be attributed to a small sample size with majority of the study participants being non-smokers.

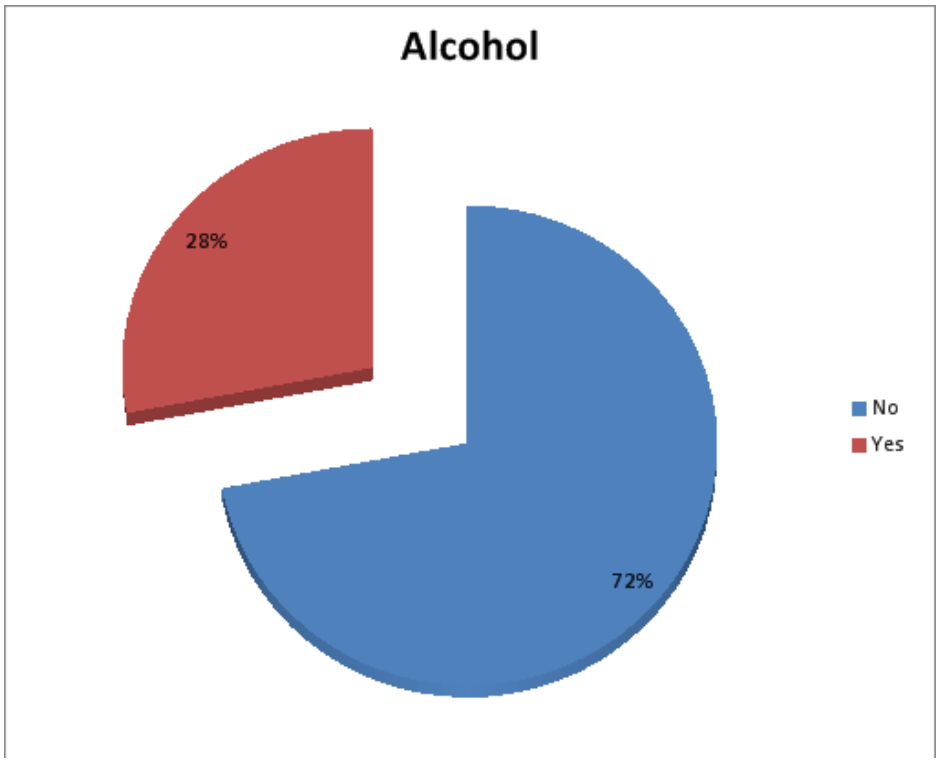
			SMOKING		Total
			No	Yes	
group	<40mg/dl	Count	38	12	50
		% within group	76.0%	24.0%	100.0%
	40mg/dl - 49mg/dl	Count	32	18	50
		% within group	64.0%	36.0%	100.0%
	>49mg/dl	Count	39	11	50
		% within group	78.0%	22.0%	100.0%
Total		Count	109	41	150
		% within group	72.7%	27.3%	100.0%

Pearson Chi-Square=2.887 p=0.236

ALCOHOL

ALCOHOL	FREQUENCY	PERCENT
No	108	72.0
Yes	42	28.0
Total	**150**	**100.0**

In the study group, 42 persons (28%) consumed alcohol while the rest 108 (72%) denied alcohol consumption.

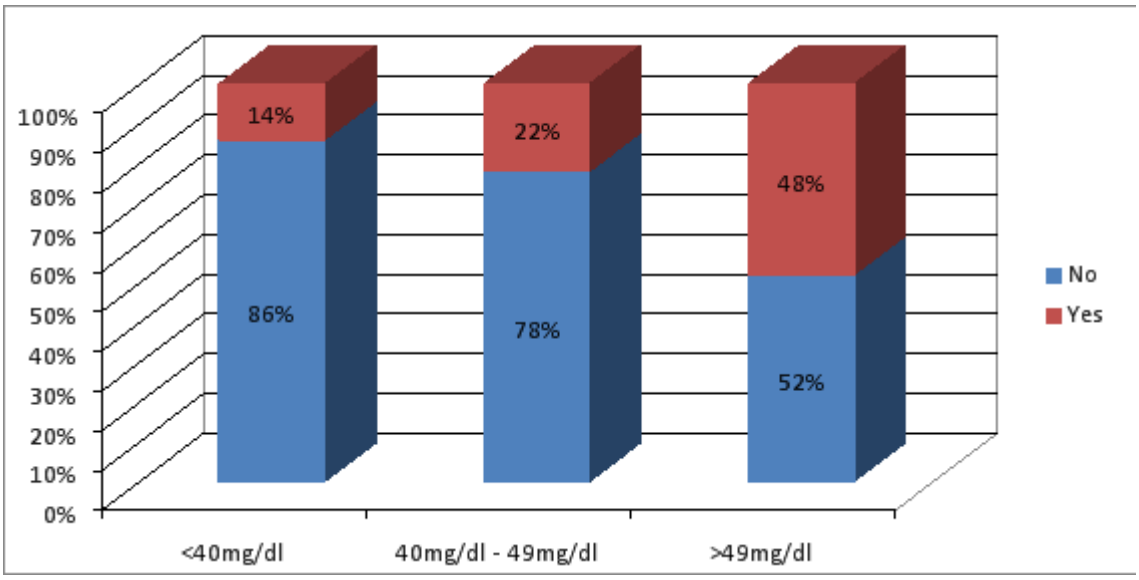


			ALCOHOL		Total
			No	Yes	
group	<40mg/dl	Count	43	7	50
		% within group	86.0%	14.0%	100.0%
	40mg/dl - 49mg/dl	Count	39	11	50
		% within group	78.0%	22.0%	100.0%
	>49mg/dl	Count	26	24	50
		% within group	52.0%	48.0%	100.0%
Total		Count	108	42	150
		% within group	72.0%	28.0%	100.0%

Pearson Chi-Square=16.675** p<=0.001

As depicted above, a positive association is seen between alcohol consumption and HDL levels.

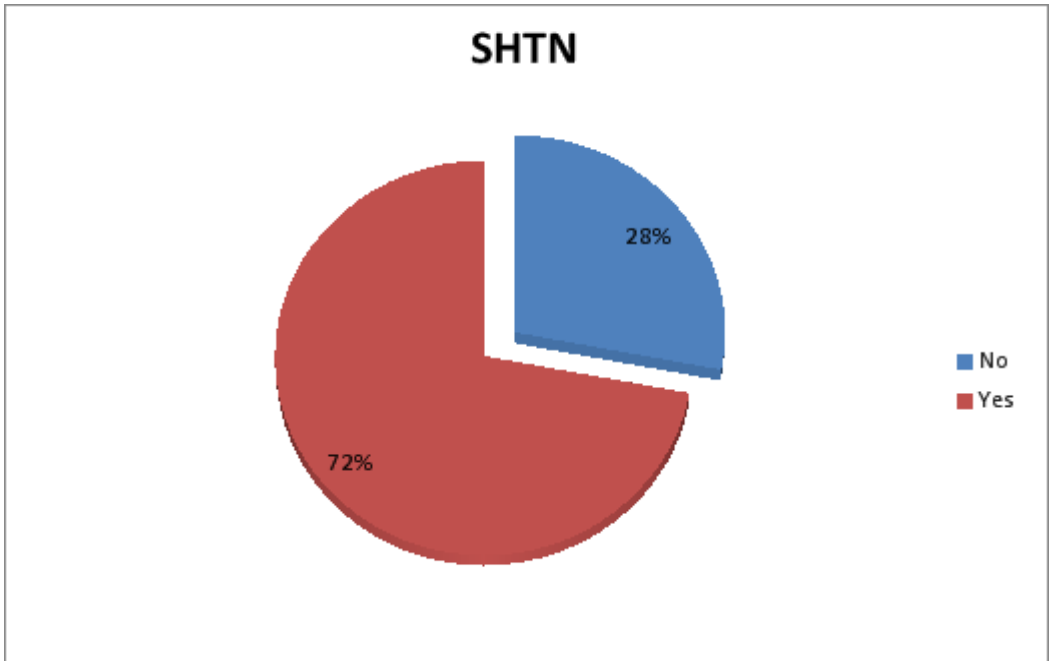
Alcohol consumption may elevate HDL cholesterol levels by increasing the cellular cholesterol efflux and plasma cholesterol esterification as proved in several trials.



HYPERTENSION

SHTN	FREQUENCY	PERCENT
Non-Hypertensives	42	28.0
Hypertensives	108	72.0
Total	**150**	**100.0**

With increasing longetivity, the prevalence of non-communicable diseases like diabetes and hypertension is also increasing in the general population. Of the 150 patients 108 (72%) were hypertensives, that is almost every one in three of the study group.



However there was no significant association between HDL levels and hypertension.

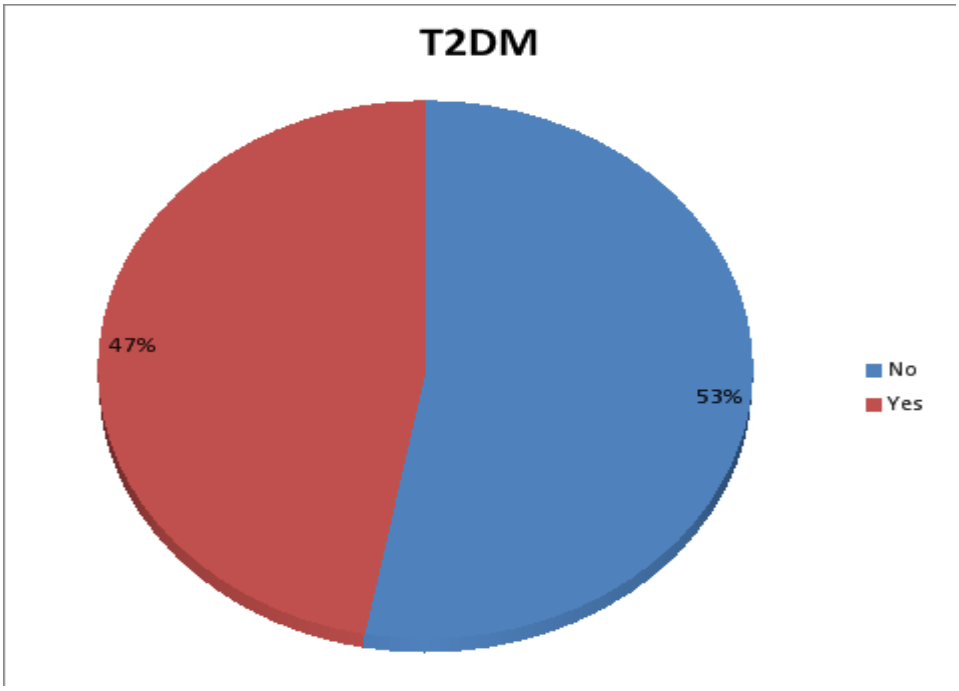
			SHTN		Total
			No	Yes	
group	<40mg/dl	Count	16	34	50
		% within group	32.0%	68.0%	100.0%
	40mg/dl - 49mg/dl	Count	11	39	50
		% within group	22.0%	78.0%	100.0%
	>49mg/dl	Count	15	35	50
		% within group	30.0%	70.0%	100.0%
Total		Count	42	108	150
		% within group	28.0%	72.0%	100.0%

Pearson Chi-Square=1.389 p=0.499

DIABETES MELLITUS

T2DM	FREQUENCY	PERCENT
Non Diabetics	79	52.7
Diabetics	71	47.3
Total	**150**	**100.0**

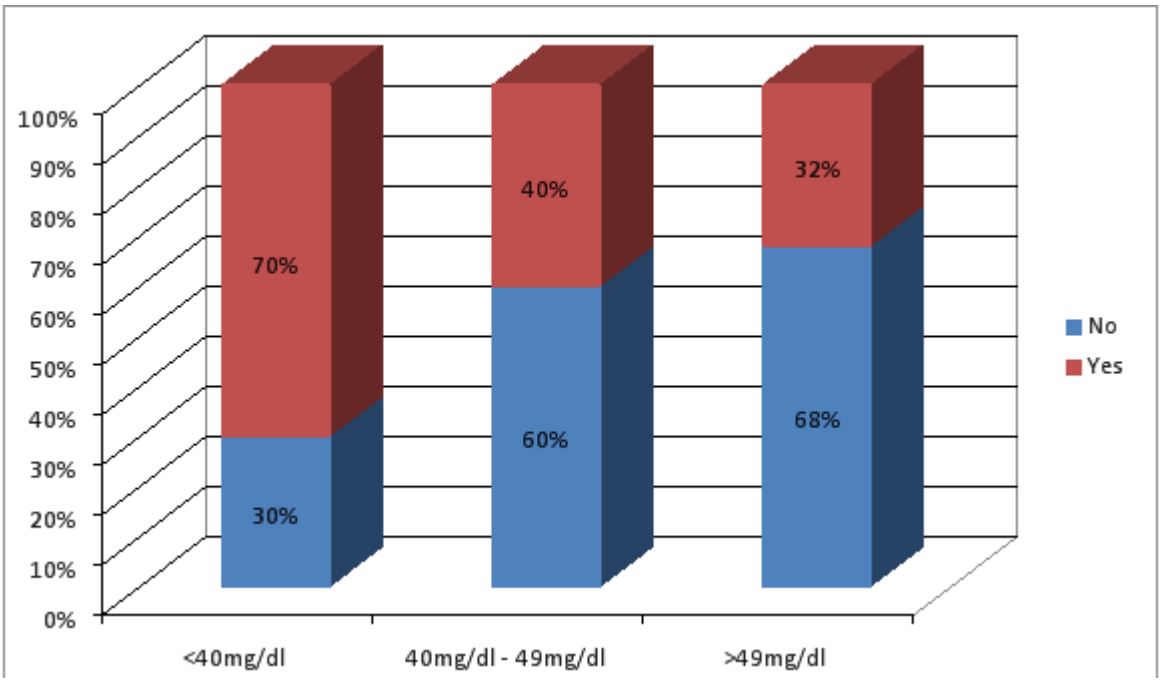
Nearly half of the study population (47.3%) were diabetics and were on oral hypoglycemic agents or insulin.



The prevalence of diabetes mellitus in the individuals in the third tertile was found to be much lower than those in first and second tertiles. This negative association between diabetes and HDL levels was found to be significant.

			T2DM		Total
			No	Yes	
group	<40mg/dl	Count	15	35	50
		% within group	30.0%	70.0%	100.0%
	40mg/dl - 49mg/dl	Count	30	20	50
		% within group	60.0%	40.0%	100.0%
	>49mg/dl	Count	34	16	50
		% within group	68.0%	32.0%	100.0%
Total		Count	79	71	150
		% within group	52.7%	47.3%	100.0%

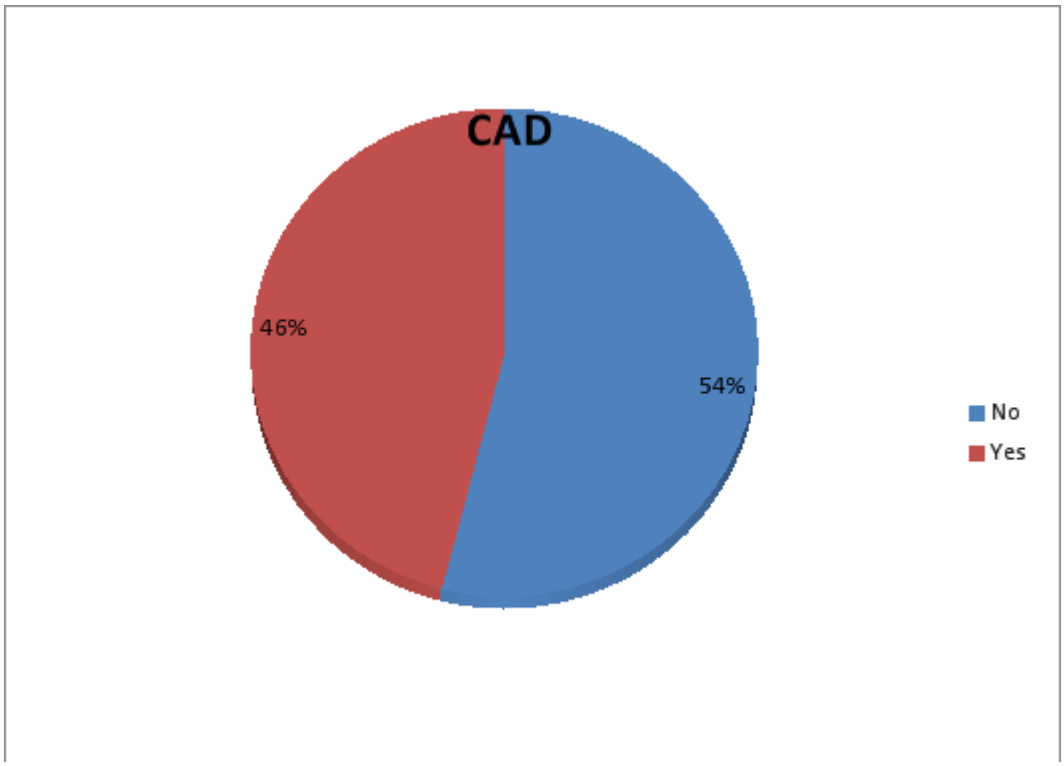
Pearson Chi-Square=16.099** p<0.001



CORONARY ARTERY DISEASE

CAD	Frequency	Percent
No	81	54.0
Yes	69	46.0
Total	150	100.0

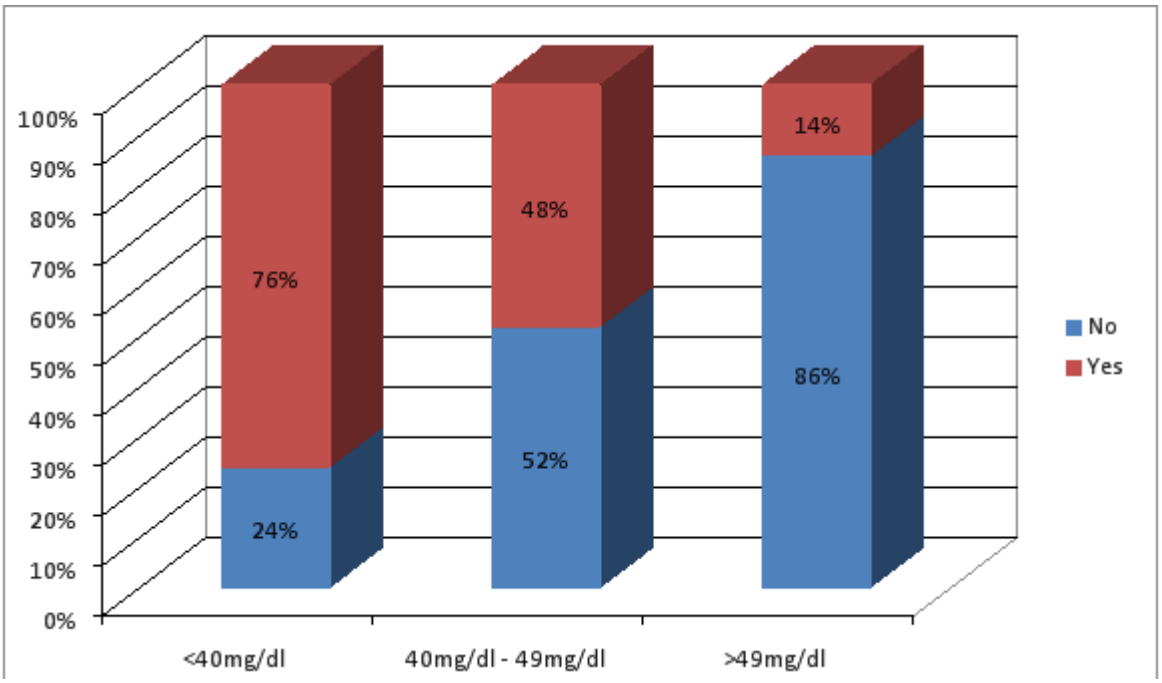
The prevalence of coronary artery disease, which is the leading cause of death worldwide, was found to be 46% (i.e) 69 out of 150.



As proven from several studies, the role of HDL as a negative predictor of coronary events was well established.

			CAD		Total
			No	Yes	
group	<40mg/dl	Count	12	38	50
		% within group	24.0%	76.0%	100.0%
	40mg/dl - 49mg/dl	Count	26	24	50
		% within group	52.0%	48.0%	100.0%
	>49mg/dl	Count	43	7	50
		% within group	86.0%	14.0%	100.0%
Total		Count	81	69	150
		% within group	54.0%	46.0%	100.0%

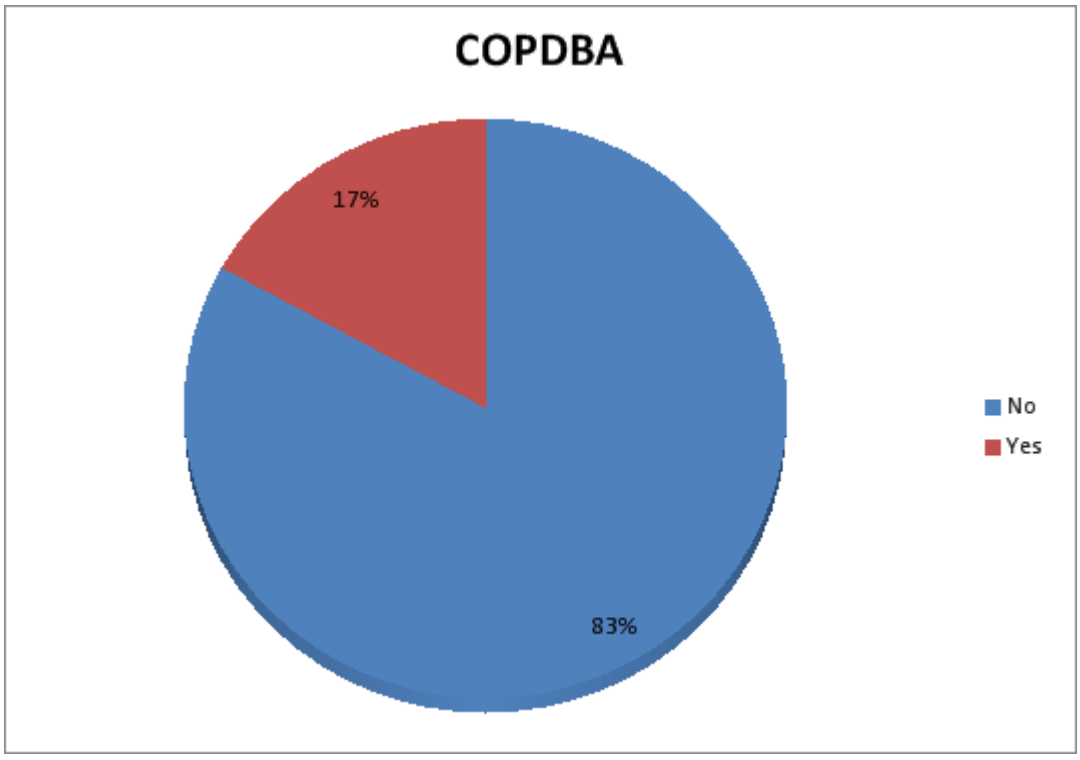
Pearson Chi-Square=38.808** p<0.001



COPD/ BRONCHIAL ASTHMA

COPD/BA	FREQUENCY	PERCENT
No	125	83.3
Yes	25	16.7
Total	**150**	**100.0**

Of the 150 participants , only 25 had obstructive airway pathology in the form of either COPD or bronchial asthma.



HDL did not have a positive or negative influence on the airway disease.

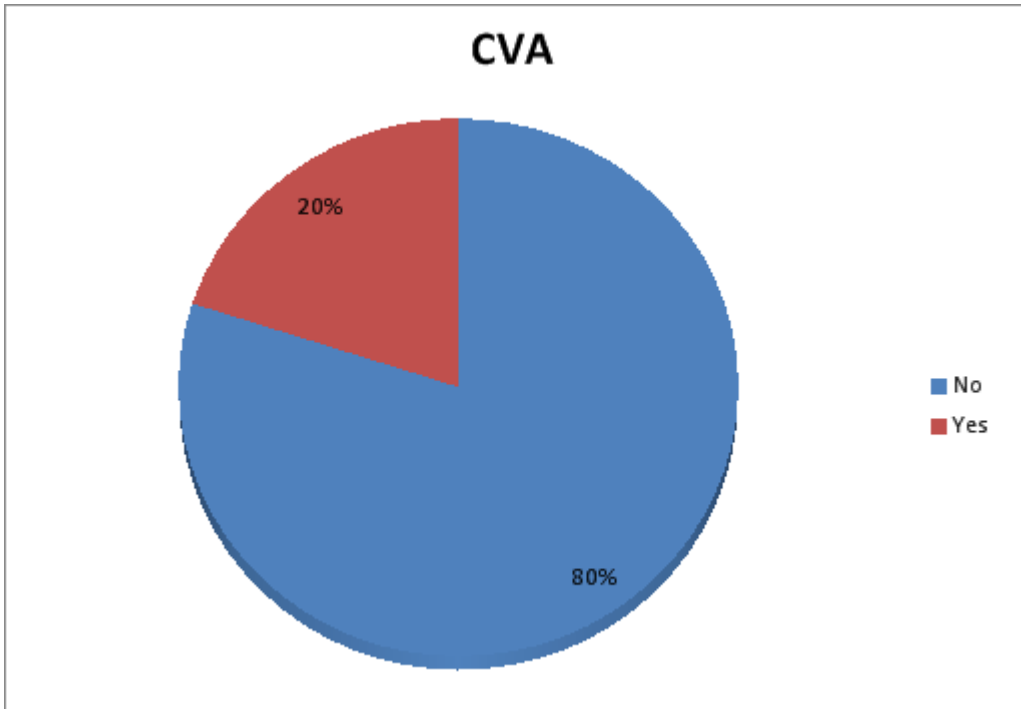
			COPDBA		Total
			No	Yes	
group	<40mg/dl	Count	42	8	50
		% within group	84.0%	16.0%	100.0%
	40mg/dl - 49mg/dl	Count	41	9	50
		% within group	82.0%	18.0%	100.0%
	>49mg/dl	Count	42	8	50
		% within group	84.0%	16.0%	100.0%
Total		Count	125	25	150
		% within group	83.3%	16.7%	100.0%

Pearson Chi-Square=0.096 p=0.953

CEREBROVASCULAR ACCIDENT

CVA	FREQUENCY	PERCENT
No	120	80.0
Yes	30	20.0
Total	**150**	**100.0**

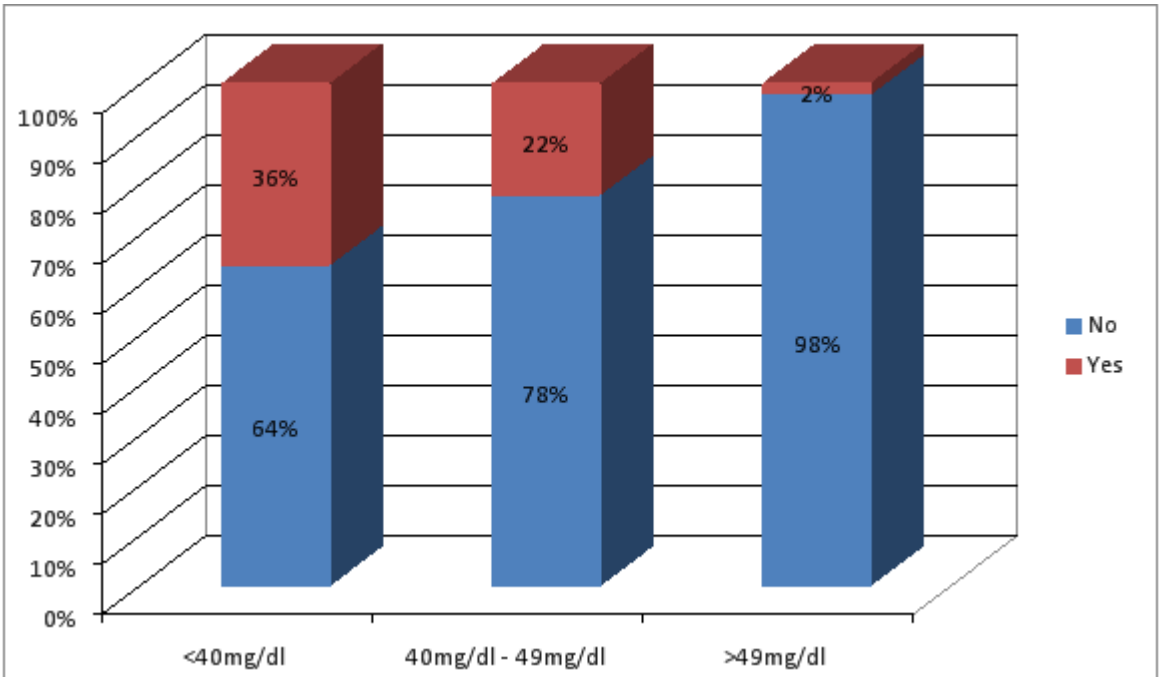
Of the 150 patients, 30 had suffered cerebrovascular accidents- either ischemic or haemorrhagic stroke in the past. This accounts to 20% of the sample size.



As the incidence of stroke decreases with increasing HDL, there seems to be a negative association between HDL levels and stroke.

			CVA		Total
			No	Yes	
group	<40mg/dl	Count	32	18	50
		% within group	64.0%	36.0%	100.0%
	40mg/dl - 49mg/dl	Count	39	11	50
		% within group	78.0%	22.0%	100.0%
	>49mg/dl	Count	49	1	50
		% within group	98.0%	2.0%	100.0%
Total		Count	120	30	150
		% within group	80.0%	20.0%	100.0%

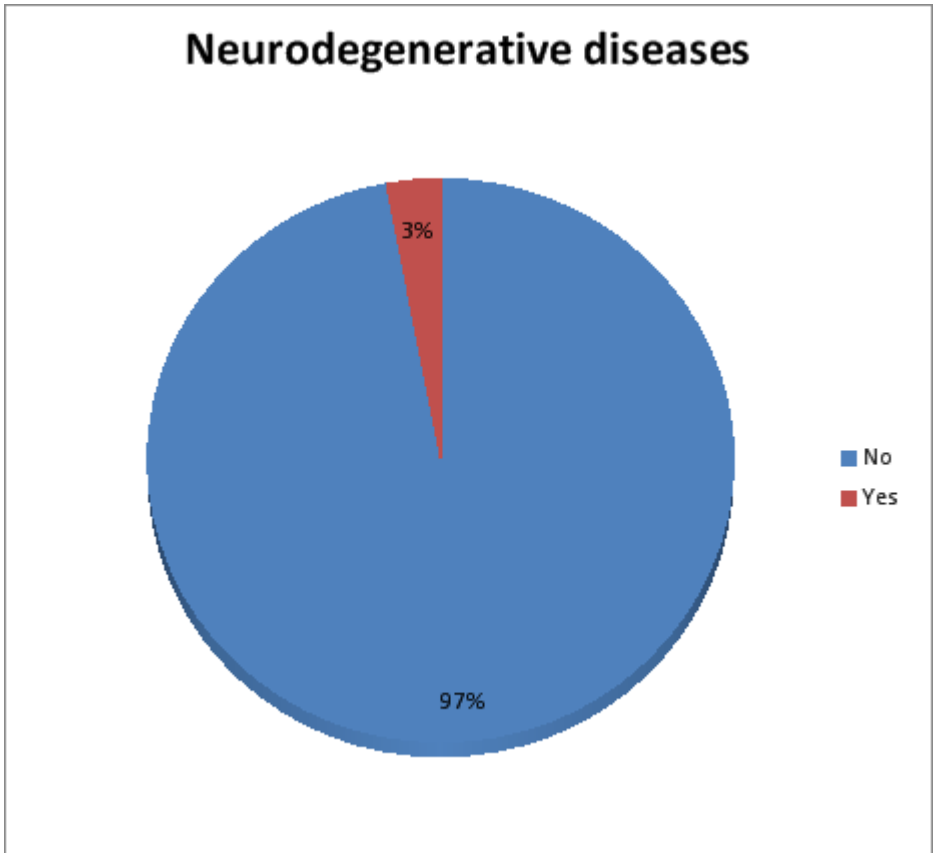
Pearson Chi-Square=18.250** p<0.001



NEURODEGENERATIVE DISEASES

Neurodegenerative diseases	Frequency	Percent
No	146	97.3
Yes	4	2.7
Total	150	100.0

The prevalence of neurodegenerative conditions like Parkinson's disease in the study population was found to be 4%.



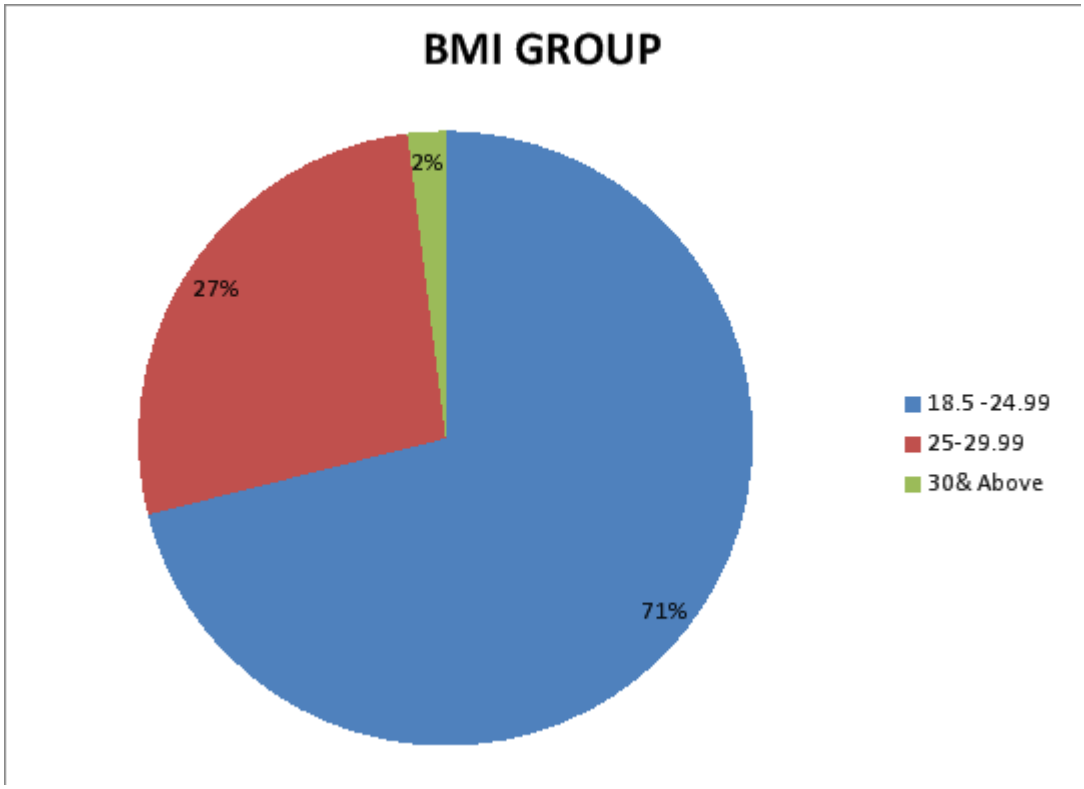
There was no significant association between the two parameters.

			N#DISEASE		Total
			No	Yes	
group	<40mg/dl	Count	47	3	50
		% within group	94.0%	6.0%	100.0%
	40mg/dl - 49mg/dl	Count	50	0	50
		% within group	100.0%	0.0%	100.0%
	>49mg/dl	Count	49	1	50
		% within group	98.0%	2.0%	100.0%
Total		Count	146	4	150
		% within group	97.3%	2.7%	100.0%

Pearson Chi-Square=3.596 p=0.166

BODY MASS INDEX (BMI)

BMI_GROUP	FREQUENCY	PERCENT
18.5 -24.99	107	71.3
25-29.99	40	26.7
30& Above	3	2.0
Total	**150**	**100.0**



Of the 150 participants, 103 were normal weight, 40 were overweight and 3 were obese.No significant association was found between HDL levels and BMI.

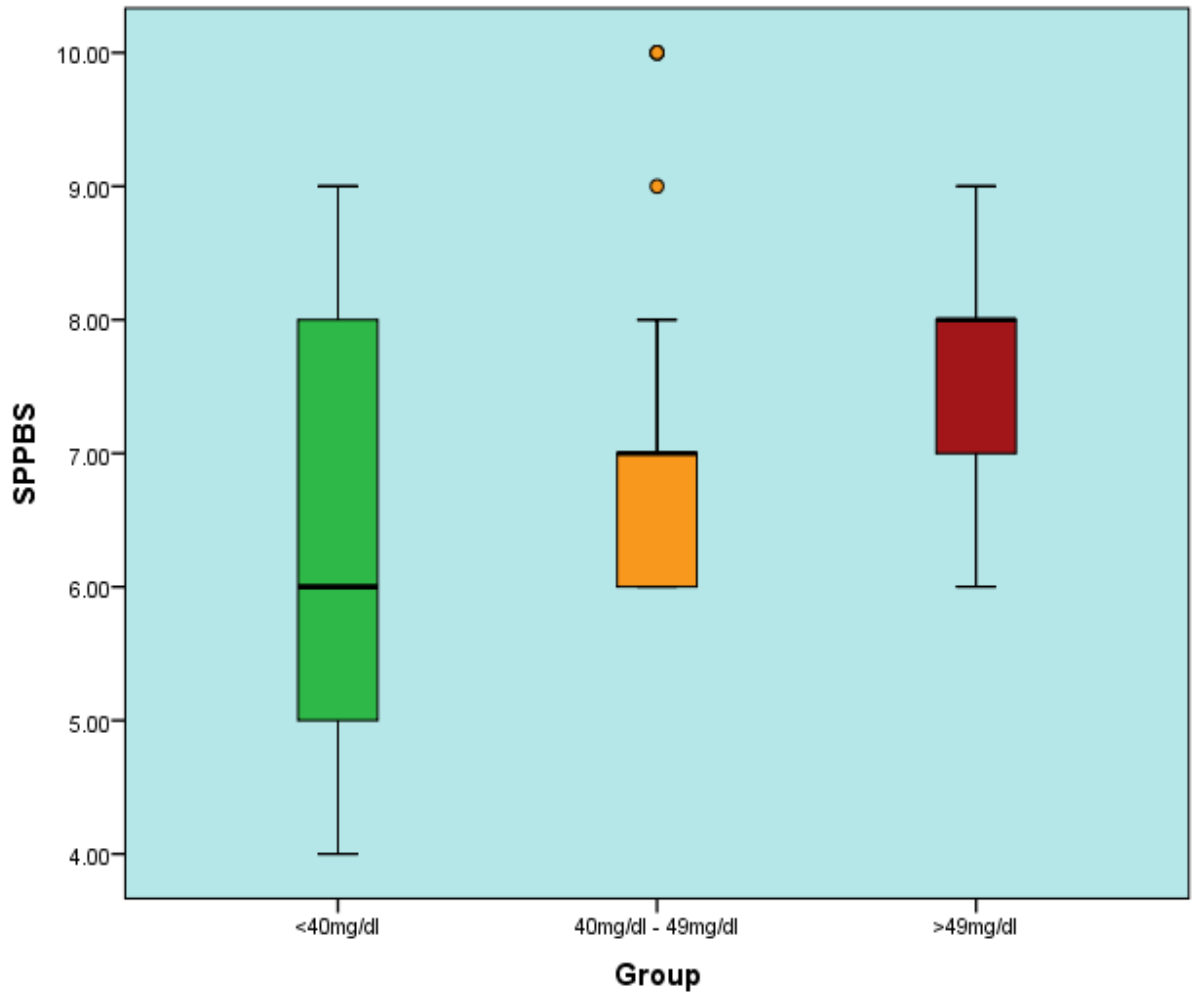
			BMI_Group			Total
			18.5 -4.99	25-9.99	30& above	
group	<40mg/dl	Count	30	19	1	50
		% within group	60.0%	38.0%	2.0%	100.0%
	40mg/dl - 49mg/dl	Count	38	11	1	50
		% within group	76.0%	22.0%	2.0%	100.0%
	>49mg/dl	Count	39	10	1	50
		% within group	78.0%	20.0%	2.0%	100.0%
Total		Count	107	40	3	150
		% within group	71.3%	26.7%	2.0%	100.0%

Pearson Chi-Square=5.014 p= 0.286

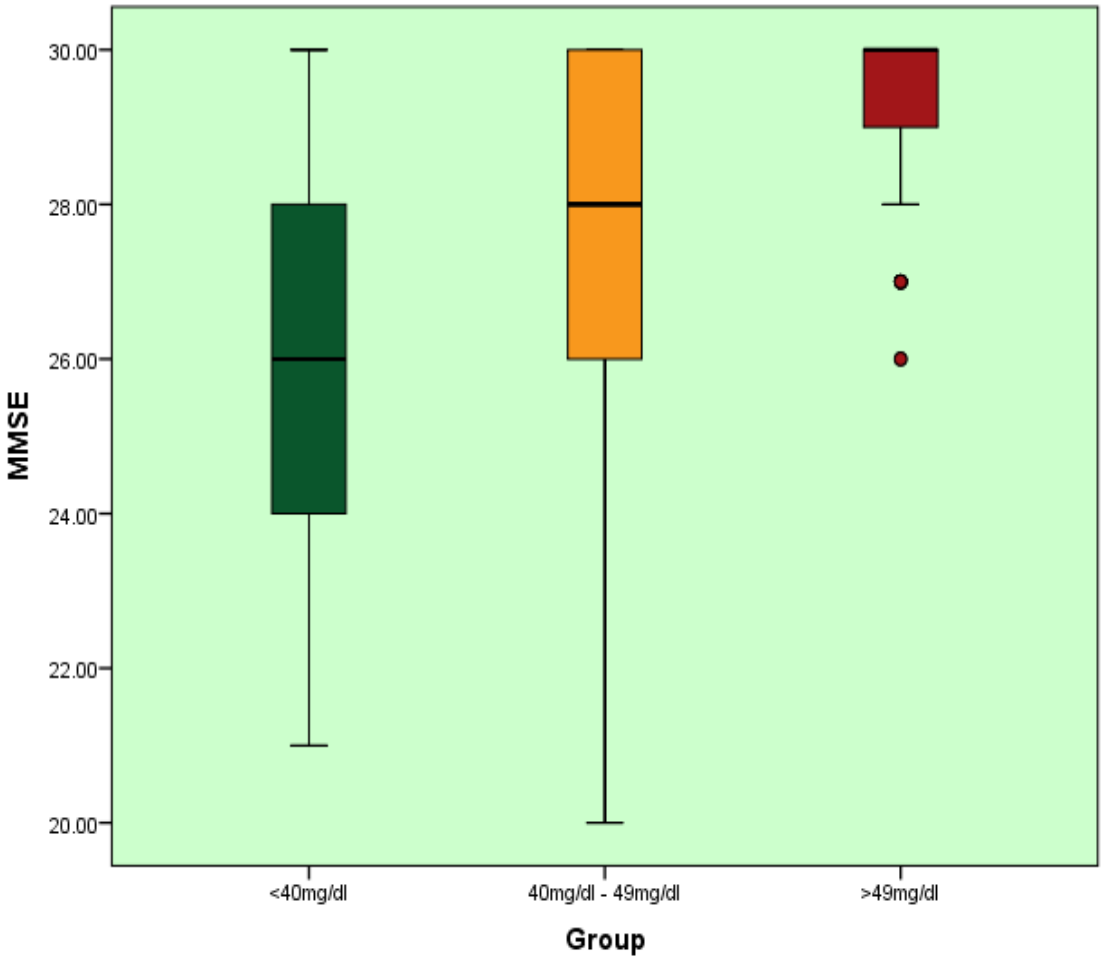
		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum	
						Lower Bound	Upper Bound			F value
SPPBS	<40mg/dl	50	6.4400	1.40204	.19828	6.0415	6.8385	4.00	9.00	17.481**
	40mg/dl - 49mg/dl	50	7.0200	1.03982	.14705	6.7245	7.3155	6.00	10.00	
	>49mg/dl	50	7.7800	.91003	.12870	7.5214	8.0386	6.00	9.00	
	Total	150	7.0800	1.25597	.10255	6.8774	7.2826	4.00	10.00	
MMSE	<40mg/dl	50	26.1000	2.35822	.33350	25.4298	26.7702	21.00	30.00	28.803**
	40mg/dl - 49mg/dl	50	27.8400	2.29783	.32496	27.1870	28.4930	20.00	30.00	
	>49mg/dl	50	29.1800	1.25666	.17772	28.8229	29.5371	26.00	30.00	
	Total	150	27.7067	2.38436	.19468	27.3220	28.0914	20.00	30.00	

**p<0.0001

The mean SPPBS for persons belonging to the first, second and third tertiles are 6.44, 7.02, 7.78 respectively. This goes to prove that there is a positive association between HDL levels and physical performance (i.e) with increasing HDL levels the physical performance (as measured by the short physical performance battery) improves. This association was found to be statistically significant.($p<0.0001$)



The mean MMSE scores for persons belonging to the first, second and third tertiles are 25.42 , 27.18 , 28.82 respectively. This goes to prove that there is a positive association between HDL levels and cognition (i.e) with increasing HDL levels the cognitive function (as measured by the short physical performance battery) improves. This association was found to be statistically significant.($p<0.0001$)



Bonferroni							
Dependent Variable	(I) group	(J) group	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
HDL	<40mg/dl	40mg/dl - 49mg/dl	-10.16000*	.38351	.000	-11.0887	-9.2313
		>49mg/dl	-17.18000*	.38351	.000	-18.1087	-16.2513
	40mg/dl - 49mg/dl	<40mg/dl	10.16000*	.38351	.000	9.2313	11.0887
		>49mg/dl	-7.02000*	.38351	.000	-7.9487	-6.0913
	>49mg/dl	<40mg/dl	17.18000*	.38351	.000	16.2513	18.1087
		40mg/dl - 49mg/dl	7.02000*	.38351	.000	6.0913	7.9487
SPPBS	<40mg/dl	40mg/dl - 49mg/dl	-.58000*	.22731	.035	-1.1305	-.0295
		>49mg/dl	-1.34000*	.22731	.000	-1.8905	-.7895
	40mg/dl - 49mg/dl	<40mg/dl	.58000*	.22731	.035	.0295	1.1305
		>49mg/dl	-.76000*	.22731	.003	-1.3105	-.2095
	>49mg/dl	<40mg/dl	1.34000*	.22731	.000	.7895	1.8905
		40mg/dl - 49mg/dl	.76000*	.22731	.003	.2095	1.3105
MMSE	<40mg/dl	40mg/dl - 49mg/dl	-1.74000*	.40695	.000	-2.7255	-.7545
		>49mg/dl	-3.08000*	.40695	.000	-4.0655	-2.0945
	40mg/dl - 49mg/dl	<40mg/dl	1.74000*	.40695	.000	.7545	2.7255
		>49mg/dl	-1.34000*	.40695	.004	-2.3255	-.3545
	>49mg/dl	<40mg/dl	3.08000*	.40695	.000	2.0945	4.0655
		40mg/dl - 49mg/dl	1.34000*	.40695	.004	.3545	2.3255
*. The mean difference is significant at the 0.05 level.							

DISCUSSION

In this study conducted at RGGGH patients presenting to the hospital were selected by simple random sampling and divided into three tertiles based on their HDL levels. The physical performance and cognitive function of the participants in each tertile was assessed using short physical performance battery and MMSE respectively. The list of co-morbidities and personal history of all the patients was collected.

HDL AND CO-VARIATES

On analyzing the collected data, a statistically significant association was observed between HDL levels and the following factors

- Alcohol
- Diabetes mellitus
- Coronary artery disease
- Cerebrovascular events

In our study, of the 150 participants 42 (28%) consumed alcohol. On comparison, percentage of alcohol consumers was highest in the third tertile and least in the first tertile. This shows that HDL levels in those who consumed alcohol was significantly higher than those who refrained from alcohol abuse. Thus there is a significant positive association between HDL levels and alcohol consumption. This is in accordance with several studies which have

demonstrated that mild to moderate consumption of alcohol rises levels of HDL

Another factor having significant association with HDL levels in our study is diabetes. Unlike in alcohol consumption, the association between HDL and diabetes is a negative one (i.e) HDL acts as a negative predictor of diabetes mellitus. Prevalence of diabetes was found to be less in those with high HDL cholesterol level.(70% in the first tertile, 40% in the second tertile and 32% in the third tertile).This is attributed to increased insulin sensitivity induced by HDL cholesterol. Similar results were obtained in the ilSIRENTE study, conducted in Central Italy

The cardio protective role of HDL is well established in several trials. In our study too, a negative association was observed between HDL and prevalence of CAD. Of the 150 participants, 69 had CAD. Among the 69, 38 persons belonged to the first tertile, 24 to the second tertile and 7 to the third tertile. The negative association between HDL and CAD was statistically significant. Hence the role of HDL as a negative predictor of coronary events was confirmed.

Similarly, a negative association between HDL and CVA was observed among the study participants. This association can be attributed to the athero protective effects of HDL.

There was no significant association between HDL levels and other variables like age and BMI. This is contradiction to the the ilSIRENTE study,

conducted in Central Italy, were a significant association was established with the above factors.

Also smoking, COPD and neurodegenerative conditions like Parkinsons did not have any significant association with HDL.

HDL AND PHYSICAL PERFORMANCE

Physical performance of the study participants was assessed using the short physical performance battery in which the following components was tested - gait speed, repeated chair stand and balance. The mean SPPBS for persons belonging to the first, second and third tertiles was calculated and was found to be 6.44, 7.02, 7.78 respectively. The data revealed a positive association between HDL levels and physical performance (i.e) with increasing HDL levels the physical performance (as measured by the short physical performance battery) improves. This association was found to be statistically significant ($p<0.0001$).The better physical performance in individuals with higher HDL can be probably attributed to the reasons discussed below.

- Cholesterol levels may be associated inversely with the onset and outcomes of specific diseases (31).

- Higher cholesterol levels have been associated with better short-term health outcomes after acute strokes (32).

- Another study found that cholesterol levels were related inversely to morbidity and mortality after surgery (33).

- Finally low cholesterol concentration is often associated with poor health status and impaired disability recovery (34).

Hence even though genetic predisposition and specific metabolic conditions are the most important determinants of serum cholesterol concentration, reduced HDL-cholesterol levels have been hypothesized to be a general marker of poor wellbeing and reduced physical condition.

HDL AND COGNITION

Cognition in the study participants was assessed using Mini Mental Status Examination (MMSE) tamil version. The mean MMSE scores for persons belonging to the first, second and third tertiles was calculated and was found to be 25.42 , 27.18 , 28.82 respectively. This implies that there is a positive association between HDL levels and cognition (i.e) with increasing HDL levels the cognitive function (as measured by MMSE) improves. This association was found to be statistically significant.($p<0.0001$). This result is in concordance with the studies conducted previously in Korea and China. The improved cognition in individuals with higher HDL levels can be attributed to the fact that HDL prohibits the formation of beta amyloid plaques, which constitutes one of the defining hallmarks of neurodegenerative diseases like Alzheimer's disease (35).

STRENGTHS AND LIMITATIONS OF THE STUDY

STRENGTH

- ✓ Almost equal number of men and women were included in the study.
- ✓ There were equal number of study participants in all three categories of HDL.

LIMITATION

- ✓ Small sample size.
- ✓ Hospital based study
- ✓ Hormones which are thought to have a causative role were not tested
- ✓ This study included patients who are on statins.

CONCLUSION

In conclusion, the present study suggests that among the elderly, higher levels of HDL-cholesterol are associated with better physical performance and cognition. This result is of particular importance as improved HDL-cholesterol levels by either lifestyle modification or pharmacological intervention could get better physical performance and cognition in the elderly.

Also the study revealed a statistically significant association between HDL levels and the following factors

- Alcohol (positive association between HDL and alcohol consumption)
- Diabetes mellitus (negative association between HDL and diabetes)
- Coronary artery disease (negative association between HDL and coronary artery disease)
- Cerebrovascular events (negative association between HDL and cerebrovascular events)

This study needs to be validated with further trials about the possible roles of HDL-cholesterol in the pathophysiology of 'geriatric syndromes' as it could have a phenomenal clinical implication.

BIBLIOGRAPHY

1. von Eckardstein A, Nofer JR, Assmann G. High density lipoproteins and arteriosclerosis. Role of cholesterol efflux and reverse cholesterol transport. Arterioscler Thromb Vasc Biol. 2001 Jan;21(1):13–27.

2. Miller GJ, Miller NE. Plasma-high-density-lipoprotein concentration and development of ischaemic heart-disease. Lancet Lond Engl. 1975 Jan 4;1(7897):16–9.

3. Nofer J-R, van der Giet M, Tölle M, Wolinska I, von Wnuck Lipinski K, Baba HA, et al. HDL induces NO-dependent vasorelaxation via the lysophospholipid receptor S1P3. J Clin Invest. 2004 Feb;113(4):569–81.

4. Guo L, Chen M, Song Z, Daugherty A, Li X-A. C323 of SR-BI is required for SR-BI-mediated HDL binding and cholesteryl ester uptake. J Lipid Res. 2011 Dec;52(12):2272–8.

5. Yuhanna IS, Zhu Y, Cox BE, Hahner LD, Osborne-Lawrence S, Lu P, et al. High-density lipoprotein binding to scavenger receptor-BI activates endothelial nitric oxide synthase. Nat Med. 2001 Jul;7(7):853–7.

6. Terasaka N, Yu S, Yvan-Charvet L, Wang N, Mzhavia N, Langlois R, et al. ABCG1 and HDL protect against endothelial dysfunction in mice fed a high-cholesterol diet. J Clin Invest. 2008 Nov;118(11):3701–13.

7. Terasaka N, Wang N, Yvan-Charvet L, Tall AR. High-density lipoprotein protects macrophages from oxidized low-density lipoprotein-induced apoptosis by promoting efflux of 7-ketocholesterol via ABCG1. Proc Natl Acad Sci U S A. 2007 Sep 18;104(38):15093–8.

8. Suc I, Escargueil-Blanc I, Troly M, Salvayre R, Nègre-Salvayre A. HDL and ApoA prevent cell death of endothelial cells induced by oxidized LDL. Arterioscler Thromb Vasc Biol. 1997 Oct;17(10):2158–66.

9. Ortiz-Muñoz G, Houard X, Martín-Ventura J-L, Ishida BY, Loyau S, Rossignol P, et al. HDL antielastase activity prevents smooth muscle cell anoikis, a potential new antiatherogenic property. FASEB J Off Publ Fed Am Soc Exp Biol. 2009 Sep;23(9):3129–39.

10. Pajkrt D, Doran JE, Koster F, Lerch PG, Arnet B, van der Poll T, et al. Antiinflammatory effects of reconstituted high-density lipoprotein during human endotoxemia. J Exp Med. 1996 Nov 1;184(5):1601–8.

11. Pérez-Morga D, Vanhollebeke B, Paturiaux-Hanocq F, Nolan DP, Lins L, Homblé F, et al. Apolipoprotein L-I promotes trypanosome lysis by forming pores in lysosomal membranes. Science. 2005 Jul 15;309 (5733):469–72.

12. High-density lipoprotein modulates glucose metabolism in patients with type 2 diabetes mellitus. - PubMed - NCBI [Internet]. [cited 2018 Oct 11]. Available from: https://www.ncbi.nlm.nih.gov/pubmed/19349317

13. Drew BG, Duffy SJ, Formosa MF, Natoli AK, Henstridge DC, Penfold SA, et al. High-density lipoprotein modulates glucose metabolism in patients with type 2 diabetes mellitus. Circulation. 2009 Apr 21;119(15):2103–11.

14. Fryirs MA, Barter PJ, Appavoo M, Tuch BE, Tabet F, Heather AK, et al. Effects of high-density lipoproteins on pancreatic beta-cell insulin secretion. Arterioscler Thromb Vasc Biol. 2010 Aug;30(8):1642–8.

15. Dalla-Riva J, Stenkula KG, Petrlova J, Lagerstedt JO. Discoidal HDL and apoA-I-derived peptides improve glucose uptake in skeletal muscle. J Lipid Res. 2013 May;54(5):1275–82.

16. Calkin AC, Drew BG, Ono A, Duffy SJ, Gordon MV, Schoenwaelder SM, et al. Reconstituted High-Density Lipoprotein Attenuates Platelet Function in Individuals With Type 2 Diabetes Mellitus by Promoting Cholesterol Efflux. Circulation [Internet]. 2009 Nov 24 [cited 2018 Oct 11]; Available from: https://www.ahajournals.org/doi/abs/ 10.1161/ circulationaha.109.870709

17. Calabresi L, Gomaraschi M, Franceschini G. Endothelial Protection by High-Density Lipoproteins: From Bench to Bedside. Arterioscler Thromb Vasc Biol. 2003 Oct;23(10):1724–31.

18. Holy EW, Besler C, Reiner MF, Camici GG, Manz J, Beer JH, et al. High-density lipoprotein from patients with coronary heart disease loses

anti-thrombotic effects on endothelial cells: impact on arterial thrombus formation. Thromb Haemost. 2014 Nov;112(5):1024–35.

19. Nofer J-R, Brodde MF, Kehrel BE. High-density lipoproteins, platelets and the pathogenesis of atherosclerosis. Clin Exp Pharmacol Physiol. 2010 Jul;37(7):726–35.

20. Nofer J-R, Kehrel B, Fobker M, Levkau B, Assmann G, von Eckardstein A. HDL and arteriosclerosis: beyond reverse cholesterol transport. Atherosclerosis. 2002 Mar;161(1):1–16.

21. Rayner KJ, Moore KJ. MicroRNA control of high-density lipoprotein metabolism and function. Circ Res. 2014 Jan 3;114(1):183–92.

22. Demeester N, Castro G, Desrumaux C, De Geitere C, Fruchart JC, Santens P, et al. Characterization and functional studies of lipoproteins, lipid transfer proteins, and lecithin:cholesterol acyltransferase in CSF of normal individuals and patients with Alzheimer's disease. J Lipid Res. 2000 Jun;41(6):963–74.

23. Pfrieger FW. Outsourcing in the brain: do neurons depend on cholesterol delivery by astrocytes? BioEssays News Rev Mol Cell Dev Biol. 2003 Jan;25(1):72–8.

24. Kontush A. Apolipoprotein Abeta: black sheep in a good family. Brain Pathol Zurich Switz. 2004 Oct;14(4):433–47.

25. Low HDL Cholesterol Levels | NEJM [Internet]. New England Journal of Medicine. [cited 2018 Oct 10]. Available from: https://www.nejm.org/doi/pdf/10.1056/NEJMcp044370

26. Landi F, Russo A, Cesari M, Pahor M, Bernabei R, Onder G. HDL-cholesterol and physical performance: results from the ageing and longevity study in the sirente geographic area (ilSIRENTE Study). Age Ageing. 2007 Sep;36(5):514–20.

27. Formiga F, Ferrer A, Chivite D, Pinto X, Cuerpo S, Pujol R. Serum high-density lipoprotein cholesterol levels, their relationship with baseline functional and cognitive status, and their utility in predicting mortality in nonagenarians. Geriatr Gerontol Int. 2011 Jul;11(3):358–64.

28. Choi H, Kim HC, Stefani KM, Lee JM, Yoon YM, Lee EY, et al. Serum high-density lipoprotein cholesterol concentration and functional state: The Korean Urban Rural Elderly (KURE) Study. Arch Gerontol Geriatr. 2017 Jul 1;71:115–21.

29. Lv Y-B, Yin ZX, Chei C-L, Brasher MS, Zhang J, Kraus VB, et al. Serum Cholesterol Levels within the High Normal Range Are Associated with Better Cognitive Performance among Chinese Elderly. J Nutr Health Aging. 2016 Mar;20(3):280–7.

30. He Q, Li Q, Zhao J, Wu T, Ji L, Huang G, et al. Relationship between plasma lipids and mild cognitive impairment in the elderly Chinese: a case-control study. Lipids Health Dis. 2016 Sep 5;15(1):146.

31. Onder G, Landi F, Volpato S, Fellin R, Carbonin P, Gambassi G, et al. Serum cholesterol levels and in-hospital mortality in the elderly. Am J Med. 2003 Sep;115(4):265–71.

32. Dyker AG, Weir CJ, Lees KR. Influence of cholesterol on survival after stroke: retrospective study. BMJ. 1997 May 31;314(7094):1584–8.

33. Delgado-Rodríguez M, Medina-Cuadros M, Gómez-Ortega A, Martínez-Gallego G, Mariscal-Ortiz M, Martinez-Gonzalez MA, et al. Cholesterol and serum albumin levels as predictors of cross infection, death, and length of hospital stay. Arch Surg Chic Ill 1960. 2002 Jul;137(7):805–12.

34. Onder G, Volpato S, Liperoti R, D'Arco C, Maraldi C, Fellin R, et al. Total serum cholesterol and recovery from disability among hospitalized older adults. J Gerontol A Biol Sci Med Sci. 2006 Jul;61(7):736–42.

35. Olesen OF, Dagø L. High density lipoprotein inhibits assembly of amyloid beta-peptides into fibrils. Biochem Biophys Res Commun. 2000 Apr 2;270(1):62–6.

36. HARRISON'S PRINCIPLES OF INTERNAL MEDICINE

37. OXFORD TEXTBOOK OF GERIATRIC MEDICINE

PROFORMA

THE EFFECT OF HDL ON PHYSICAL PERFORMANCE AND COGNITION IN ELDERLY

Name		Contact information	
Age			
Sex			
Literacy		OP No.	

Smoking	Never		Medical conditions	Y/N	Duration
	Past		Hypertension		
	Current		Diabetes mellitus		
Alcoholism	Never		CAD		
	<1 drink/week		COPD/BA		
	1-4 drinks/week		Old CVA		
	>4 drinks/week		Neurodegenerative disorders		

Anthropometry	
Height	
Weight	
BMI	

HDL Cholesterol level	
Short physical performance battery score	Gait speed Balance test Chair stand test
MMSE score	

SHORT PHYSICAL PERFORMANCE BATTERY

1 .BALANCE TESTS

A. Side-by-Side stand

Held for 10 sec ❒ 1 point

Not held for 10 sec ❒ 0 points

Not attempted ❒ 0 points.

If 0 points, end Balance Tests

B. Semi-Tandem Stand

Held for 10 sec ❒ 1 point

Not held for 10 sec ❒ 0 points

Not attempted ❒ 0 points

If 0 points, end Balance Tests

C. Tandem Stand

Held for 10 sec ❒ 2 point

Held for 3 to 9.99 sec ❒ 1 points

Held for < than 3 sec ❒ 0 points

Not attempted ❒ 0 points

2. GAIT SPEED TEST

For 4-Meter Walk:

If time is more than 8.70 sec: ❒ 1 point

If time is 6.21 to 8.70 sec: ❒ 2 points

If time is 4.82 to 6.20 sec ❒ 3 points

If time is less than 4.82 sec: ❒ 4 points

3. CHAIR STAND TEST

Participant unable to complete 5 chair stands

or completes stands in >60sec: ❒ 0 points

If chair stand time is 16.70 sec or more: ❒ 1 point

If chair stand time is 13.70 to 16.69 sec or more: ❒ 2 points

If chair stand time is 11.20 to 13.69 sec: ❒ 3 points

If chair stand time is 11.19 sec or less: ❒ 4 points

TOTAL SCORE:

MINI MENTAL STATUS EXAMINATION

ORIENTATION

Time: Year Season Month Date Time /5

Place: Country Town District Hospital Floor /5

REGISTRATION

Examiner names three objects (e.g. apple, table, penny) and asks the patient to repeat (1 point for each correct) /3

ATTENTION AND CALCULATION

Subtract 7 from 100, then repeat from result. Continue five times: 100, 93, 86, 79, 65. /5

RECALL

Ask for the names of the three objects learned earlier /3

LANGUAGE

Name two objects (e.g. pen, watch). /2

Repeat “No ifs, ands, or buts” /1

Give a three-stage command.

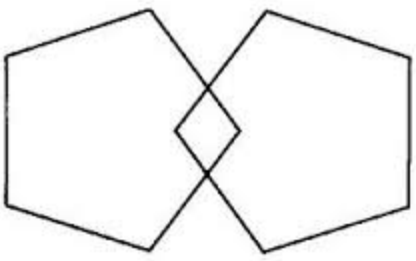
Score 1 for each stage. (e.g. “Place index finger of right hand on your nose and then on your left ear”). /3

Ask the patient to read and obey a written command on a piece of paper. The written instruction is: “Close your eyes”. /1

Ask the patient to write a sentence. Score 1 if it is sensible and has a subject and a verb.

/1

COPYING: Ask the patient to copy a pair of intersecting pentagons /1



TOTAL SCORE

INFORMATION SHEET

We are conducting a study titled **"Effect of HDL on Physical Performance and Cognition in Elderly"** among patients attending Geriatric OPD at Rajiv Gandhi Government General Hospital, Chennai .

We are selecting certain cases and if you are found eligible, we may be using your blood samples to do lipid profile, we will collect your clinical history and will conduct simple office based tests to assess your physical performance and cognition.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of investigator Signature of participant

Date:

Place: Chennai.

PATIENT CONSENT FORM

Study Detail : Effect of HDL on Physical Performance and Cognition

Study Centre : Rajiv Gandhi Govt. General Hospital, Chennai.

Patient's Name :

Patient's Age :

OP. No. :

Patient may check (✓) these boxes:

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

c) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms. ☐

e) I hereby consent to participate in this study. ☐

f) I hereby give permission to undergo clinical examination and biochemical tests. ☐

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To

Dr.Oviya Elango
I Year PG in MD Geriatrics
Department of Geriatric Medicine
Madras Medical College
Chennai 600 003

Dear Dr.Oviya Elango,

The Institutional Ethics Committee has considered your request and approved your study titled **"EFFECT OF HDL ON PHYSICAL PERFORMANE AND COGNITION IN ELDERLY " - NO.22062017(A)**

The following members of Ethics Committee were present in the meeting hold on **20.06.2017** conducted at Madras Medical College, Chennai 3

1. Prof.Dr.C.Rajendran, MD.,	:Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., MMC,Ch-3	: Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	:Member Secretary
4. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
5. Prof.A.Pandiya Raj,Director, Inst. of Gen.Surgery,MMC	: Member
6. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai	: Member
7. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3	: Member
8.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
9.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist
10.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

URKUND

Signature / Thumb Impression Signature of Investigator

Patient's Name & Address:

Dr.OVIYA ELANGO

Urkund Analysis Result

Analysed Document: New final doc.doc (D42480530)

Submitted: 10/12/2018 2:28:00 PM

Submitted By: oviyase@gmail.com

Significance: 2 %

Sources included in the report:

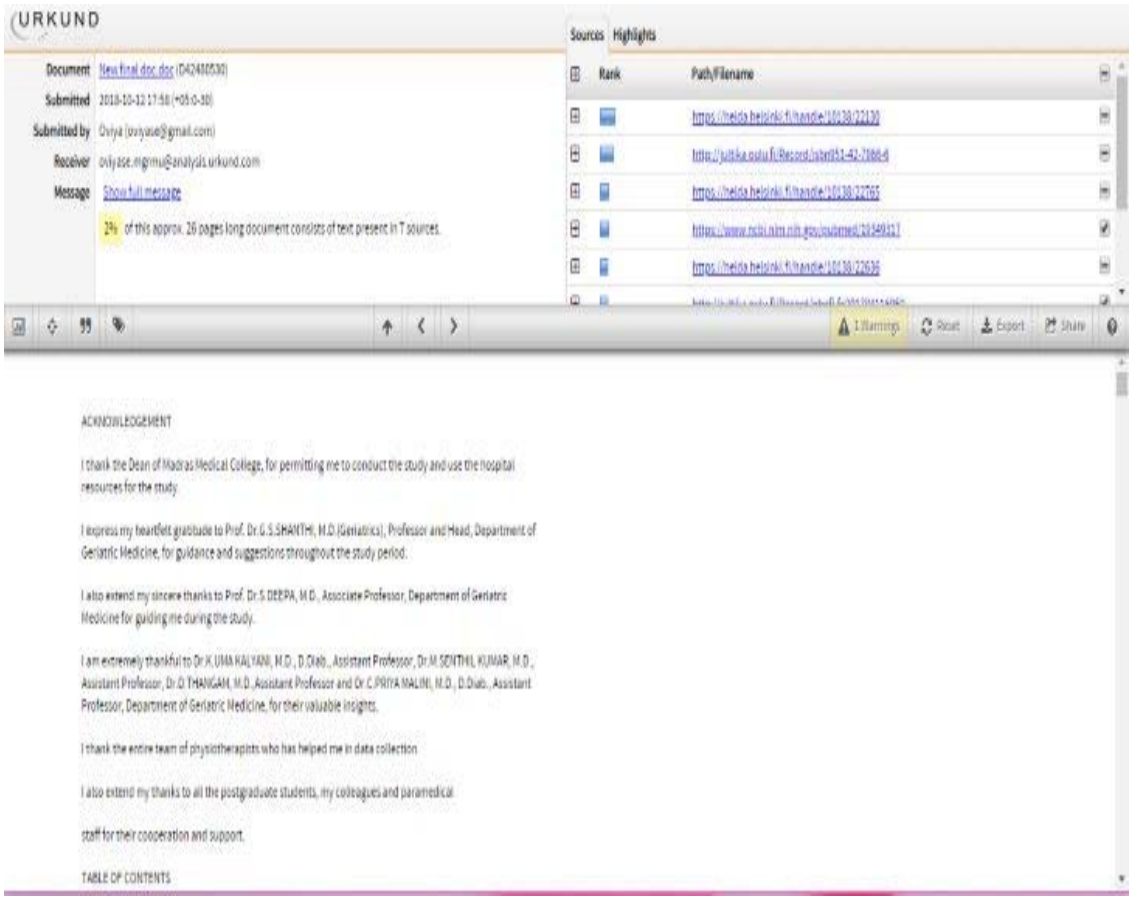
http://jultika.oulu.fi/Record/isbn951-42-7866-6

https://helda.helsinki.fi/handle/10138/22130

https://helda.helsinki.fi/handle/10138/22765

https://helda.helsinki.fi/handle/10138/22636 http://jultika.oulu.fi/Record/nbnfi-fe201704116050 https://openarchive.ki.se/xmlui/handle/10616/45179

https://www.ncbi.nlm.nih.gov/pubmed/19349317 Instances where selected sources appear: 12



ACKNOWLEDGEMENT

I thank the Dean of Madras Medical College, for permitting me to conduct the study and use the hospital resources for the study.

I express my heartfelt gratitude to Prof. Dr.G.S.SHANTHI, M.D.(Geriatrics), Professor and Head, Department of Geriatric Medicine, for guidance and suggestions throughout the study period.

I also extend my sincere thanks to Prof. Dr.S.DEEPA, M.D., Associate Professor, Department of Geriatric Medicine for guiding me during the study.

I am extremely thankful to Dr.K.UMA KALYANI, M.D., D.Diab., Assistant Professor, Dr.M.SENTHIL KUMAR, M.D., Assistant Professor, Dr.D.THANGAM, M.D.,Assistant Professor and Dr.C.PRIYA MALINI, M.D., D.Diab., Assistant Professor, Department of Geriatric Medicine, for their valuable insights.

I thank the entire team of physiotherapists who has helped me in data collection.

I also extend my thanks to all the postgraduate students, my colleagues and paramedical staff for their cooperation and support.

TABLE OF CONTENTS

CERTIFICATE - II

This is to certify that this dissertation work titled EFFECT OF HDL ON PHYSICAL PERFORMANCE AND COGNITION IN ELDERLY of the candidate Dr. OVIYA ELANGO with registration Number 201626003 for the award of M.D., degree in the branch of GERIATRIC MEDICINE.

I personally verified the urkund.com website for the purpose of plagiarism Check.

I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 2 percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

MASTER CHART

NAME	AGE	SEX	EDUCATION	SMOKING	ALCOHOL	SHN	T2M	CAD	COPD/BA	CVA	N.DISEASE	BMI	HDL	SPPBS	MMSE
ABDUL	67	M	UNEDUCATED	Y	N	Y	N	N	N	N	N	21.2	45	7	30
ADHILAKSHMI	66	F	UNEDUCATED	N	Y	Y	N	Y	N	Y	N	22	32	7	26
AKHILANDESWARI	66	F	5TH STD	N	N	Y	N	N	N	Y	N	25.8	44	6	30
AMAVASAI	68	F	UNEDUCATED	N	Y	N	Y	N	N	N	N	21.9	52	9	30
ANAND	66	M	10TH STD	N	N	Y	N	N	N	N	N	21.3	50	9	30
ANBARASAN	65	M	12TH STD	Y	Y	Y	N	N	Y	N	N	21.2	53	8	30
ANGAMMA	66	F	UNEDUCATED	N	N	N	N	N	Y	N	N	26	55	9	30
ANTHONIAMMAL	63	F	8TH STD	N	N	Y	Y	Y	N	N	N	26.5	44	6	30
AROKIYARAJ	65	M	UNEDUCATED	Y	N	N	N	N	Y	Y	N	26	38	9	26
BABU	82	M	UNEDUCATED	N	N	Y	N	Y	N	Y	N	21	33	5	24
BALAMURUGA	68	M	8TH STD	Y	N	Y	N	N	N	N	N	21.1	42	7	29
BALAMURUGAN	68	M	12TH STD	N	N	Y	N	Y	N	N	N	25.5	35	9	30
BALU	78	M	UNEDUCATED	N	N	Y	N	N	N	N	N	25.8	45	6	28
BARATHI	68	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	18.8	37	8	27
BASKAR	72	M	UNEDUCATED	N	Y	N	Y	N	N	N	N	21.8	52	7	30
BHAKIYAM	68	F	UNEDUCATED	N	Y	Y	N	N	N	N	N	23.4	53	8	30
BHUVANESHWARI	68	F	4THSTD	N	N	Y	N	N	N	N	N	22.8	53	8	29
CHANDRA	65	F	4TH STD	N	N	Y	N	N	N	N	N	21.8	51	8	30
CHINNADURAI	83	M	UNEDUCATED	Y	N	Y	N	Y	N	Y	N	22.8	44	6	25
CHITRADEVI	73	F	UNEDUCATED	N	N	Y	Y	N	N	N	N	22.8	48	6	24
DANAPAL	70	M	UNEDUCATED	Y	Y	Y	N	Y	N	N	N	25.2	52	7	29
DAVID	71	M	10TH STD	N	N	N	Y	N	N	N	N	22.8	53	7	30
DHANALAKSHMI	80	F	UNEDUCATED	N	N	Y	Y	N	N	N	N	22.1	53	7	29
DHARMALINGAM	83	M	UNEDUCATED	N	Y	N	Y	Y	N	N	N	26	34	6	26
DINAKAR	67	M	7TH STD	N	Y	N	N	N	N	N	Y	24.2	51	6	30
DURAIRAJ	73	M	5TH STD	Y	N	Y	N	Y	Y	N	N	24	32	7	26
EHWARIAMAL	61	F	UNEDUCATED	N	N	Y	N	N	N	N	N	24.5	49	7	30
ELANGO	69	M	10TH STD	N	Y	Y	N	N	N	N	N	25.2	51	9	30
ELAVARASAN	73	M	UNEDUCATED	Y	N	Y	N	N	N	N	N	21.7	54	7	29
ESHWARI	69	F	5TH STD	N	N	Y	Y	N	N	N	N	27	34	7	23
EZHIL	61	M	B.A	Y	Y	N	N	N	Y	N	N	25.8	53	9	30
GANESAN	68	M	12TH STD	N	N	Y	N	N	N	N	N	21.8	51	8	30
GOPAL	61	M	12TH STD	N	Y	N	Y	Y	N	Y	N	22	35	8	27
GUNASEKARAN	71	M	UNEDUCATED	N	Y	Y	Y	Y	N	Y	N	22.7	45	7	22
GUNASEKARAN	78	M	UNEDUCATED	Y	Y	N	Y	N	Y	N	N	21.1	53	7	27

HARIDOSS	65	M	B.A	Y	Y	Y	Y	N	N	N	N	22.5	52	9	30
JANAKI	66	F	UNEDUCATED	N	N	Y	Y	N	N	N	N	21.8	53	8	30
JESURATHINAM	81	M	UNEDUCATED	N	N	Y	Y	Y	N	N	N	21.21	44	6	26
JOHN	77	M	12THSTD	N	Y	Y	Y	Y	N	N	N	21.4	33	8	29
JOSEPH	68	M	6TH STD	Y	Y	Y	N	Y	Y	N	N	24.8	43	7	30
KALAISELVI	66	F	5TH STD	N	N	Y	N	N	N	N	N	24	52	9	30
KALIDOSS	77	M	10TH STD	N	N	Y	N	N	N	N	N	21.7	51	7	30
KALPANA	79	F	UNEDUCATED	N	N	Y	N	Y	N	N	N	24.7	46	6	26
KAMATCHI	60	F	7TH STD	N	N	N	N	N	Y	N	N	28	39	6	23
KANAKAVALLI	74	F	UNEDUCATED	N	N	N	N	Y	N	Y	N	23.9	48	7	28
KANAMMA	72	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	23.18	44	6	28
KANAMMAL	72	F	UNEDUCATED	N	N	Y	N	N	Y	N	N	21.9	47	6	28
KANAPPAN	74	M	7TH STD	N	Y	N	N	Y	N	N	N	22.5	53	8	30
KAVIARASAN	69	M	8TH STD	Y	N	Y	Y	N	N	N	N	23.1	51	8	30
KESAVAN	62	M	B.A	N	Y	N	Y	N	N	N	N	22.8	52	9	30
KRISHNAMMAL	72	F	UNEDUCATED	N	N	Y	N	Y	N	Y	N	26.6	37	7	29
KRISHNAMOORTHY	72	M	12TH STD	N	N	N	Y	N	N	Y	N	22.6	32	6	28
KRISHNAN	74	M	UNEDUCATED	N	N	N	Y	N	N	N	Y	20.4	33	4	23
KUMAR	78	M	UNEDUCATED	Y	N	Y	N	N	N	Y	Y	28.8	34	5	21
KUMAR	71	M	5TH STD	N	N	N	Y	Y	N	Y	N	22.9	41	6	29
KUMARAVEL	75	M	UNEDUCATED	Y	Y	Y	N	N	N	N	N	22.9	50	9	30
KUPPUSAMY	82	M	UNEDUCATED	N	N	Y	N	Y	N	N	N	21.9	42	6	26
KUTTIAMMAL	70	F	UNEDUCATED	N	Y	Y	N	N	N	N	N	19.8	54	7	27
LAKSHMI	63	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	27	38	6	24
LATHA	64	F	5TH STD	N	N	Y	N	Y	N	N	N	26.5	48	7	30
LOORTHMARY	68	F	10TH STD	N	N	Y	N	Y	N	N	N	25.8	48	6	30
MALAR	76	F	UNEDUCATED	N	N	Y	N	N	N	N	N	21.3	53	8	29
MALLIGA	60	F	7TH STD	N	N	Y	N	Y	N	N	N	23.4	53	8	30
MANICKAM	66	M	5TH STD	N	N	N	Y	Y	N	Y	N	23.6	30	5	27
MANICKAM	74	M	UNEDUCATED	Y	Y	Y	Y	N	Y	N	N	19	31	6	27
MANICKAM	84	M	UNEDUCATED	Y	Y	Y	N	N	Y	N	N	26.5	44	6	26
MANJULA	68	F	4TH STD	N	N	Y	N	N	N	N	N	22.1	51	8	30
MARY	73	F	2ND STD	N	N	Y	N	Y	N	Y	N	24.6	35	5	23
MASILAMANI	77	M	UNEDUCATED	Y	Y	Y	N	N	Y	N	N	25.6	45	6	26
MEENATCHI	71	F	UNEDUCATED	N	N	Y	N	Y	Y	N	N	20	36	7	26
MEERA	70	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	26	38	8	28
MOHAMAD	76	M	3RD STD	Y	Y	Y	N	Y	N	N	N	20.6	44	7	30
MOHANA	76	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	21.5	46	6	28
MURUGAN	64	M	10TH STD	Y	N	Y	N	N	N	N	N	22.9	46	7	28
MURUGAN	65	M	UNEDUCATED	N	N	Y	N	N	N	N	N	23.8	50	7	29

MUTHUKUMARI	71	F	UNEDUCATED	N	N	N	Y	N	N	N	N	26.5	48	7	28
NAGAMMAL	82	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	19.2	52	6	27
NARAYANAN	64	M	7TH STD	N	Y	Y	Y	N	Y	Y	N	26.9	33	6	27
NATESAN	73	M	10TH STD	N	N	N	N	N	N	N	N	22.4	55	9	30
PACHAIYAMMAL	72	F	UNEDUCATED	N	N	N	Y	Y	N	N	N	26.1	37	6	26
PADMANABAN	77	M	9TH STD	Y	N	Y	N	N	N	N	N	24.8	45	7	28
PADMAVATHY	61	F	8TH STD	N	N	N	N	Y	N	N	N	20	36	6	25
PALPANDI	80	M	UNEDUCATED	Y	Y	Y	Y	Y	N	N	N	20.7	42	5	27
PANDU	66	M	UNEDUCATED	Y	Y	Y	Y	Y	N	N	N	24.8	33	6	24
PARVATHY	73	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	24.9	45	6	26
PATTAMAL	69	F	3RD STD	N	N	Y	N	N	N	Y	N	24.8	45	7	28
PITCHAIAMMAL	66	F	UNEDUCATED	N	N	Y	N	Y	N	Y	N	21	37	5	25
PONAMMAL	88	F	UNEDUCATED	N	N	N	N	Y	N	N	N	23.2	45	6	26
PONAPPAN	81	M	UNEDUCATED	N	N	N	N	Y	N	N	N	22.3	51	8	27
PONNI	80	F	UNEDUCATED	N	N	Y	N	N	N	N	N	19.2	50	7	27
PONNUDURAI	86	M	UNEDUCATED	Y	Y	Y	N	Y	Y	Y	N	23	50	6	26
PONUTHAI	77	F	UNEDUCATED	N	N	Y	Y	Y	Y	N	N	23.9	45	6	27
POONGOTHAI	69	F	6TH STD	N	N	Y	Y	N	N	N	N	22.2	53	8	30
PUSHPAVALLI	86	F	UNEDUCATED	N	N	Y	Y	Y	N	Y	N	25.1	45	7	26
RAANI	66	F	6TH STD	N	N	Y	Y	N	N	N	N	22.4	51	9	30
RAJAMMAL	61	F	UNEDUCATED	N	N	Y	Y	Y	N	Y	N	19	36	9	26
RAJASEKAR	71	M	10TH STD	Y	Y	Y	N	N	Y	N	N	21.8	53	7	30
RAJATHI	69	F	UNEDUCATED	N	N	Y	Y	N	N	N	N	26.5	38	8	26
RAJATHI	68	F	UNEDUCATED	N	N	N	N	Y	Y	N	N	24.5	46	7	27
RAJENDRAN	62	M	12TH STD	Y	Y	Y	Y	N	N	N	N	25.1	44	8	30
RAJESWARI	72	F	UNEDUCATED	N	N	Y	Y	Y	N	Y	N	21.8	36	7	27
RAM MOHAN	74	M	7TH STD	N	N	N	N	N	N	N	N	25	53	7	30
RAMAMOORTHY	60	M	10TH STD	Y	N	Y	Y	N	N	N	N	24.9	37	6	30
RAMANI	74	M	UNEDUCATED	Y	Y	Y	Y	Y	N	N	N	22.5	38	8	23
RAMUTHAI	79	F	UNEDUCATED	N	N	N	N	N	Y	N	N	22.6	47	7	27
RAVINDRAN	79	M	7TH STD	Y	N	Y	N	N	N	Y	N	26.1	45	6	30
RENUKA	70	F	UNEDUCATED	N	N	Y	Y	N	N	N	N	22	52	6	27
REVATHY	66	F	6TH STD	N	N	Y	N	N	N	N	N	20	52	8	30
SANTHANAM	66	M	7TH STD	N	N	Y	N	N	N	N	N	26.1	53	9	27
SARASWATHY	72	F	UNEDUCATED	N	N	Y	Y	Y	N	Y	N	20	35	5	24
SARATHAMAL	67	F	UNEDUCATED	N	N	N	Y	Y	N	N	N	27	37	6	26
SARATHY	67	M	7TH STD	N	Y	N	Y	N	N	N	N	19.8	46	8	20
SAROJA	86	F	UNEDUCATED	N	N	N	Y	N	N	N	N	26	36	8	30

SAVITHRI	68	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	21.4	38	7	27
SEETHA	77	F	UNEDUCATED	N	N	Y	Y	Y	N	Y	N	33.9	45	7	27
SEETHALAKSHMI	80	F	UNEDUCATED	N	N	N	N	N	Y	N	N	25	54	7	29
SELVAMUTHU	70	M	UNEDUCATED	Y	N	Y	N	Y	N	N	N	24.8	34	9	28
SELVARAJ	76	M	UNEDUCATED	Y	Y	N	Y	Y	Y	y	N	22.9	30	4	26
SELVI	81	F	UNEDUCATED	N	Y	Y	N	N	N	Y	N	22.8	46	7	26
SELVI	70	F	UNEDUCATED	N	N	N	Y	N	N	N	N	24.5	51	8	29
SENTHIL	61	M	B.Sc	Y	Y	Y	N	N	N	N	N	25	53	8	30
SIVAKAMI	81	F	UNEDUCATED	N	N	N	Y	Y	N	N	N	30	36	8	27
SIVAKUMAR	76	M	4TH STD	N	Y	Y	N	Y	N	N	N	27	36	8	28
SRINIVASAN	82	M	UNEDUCATED	Y	N	N	Y	Y	N	N	N	23.9	46	6	24
SUBAIYA	65	M	12TH STD	N	N	Y	Y	Y	N	N	N	26.9	36	10	30
SUBASH	68	M	N	N	Y	N	Y	N	N	N	N	19.2	36	4	26
SUBRAMANI	72	M	UNEDUCATED	Y	Y	Y	Y	Y	N	Y	Y	26.6	32	5	22
SUBRAMANI	64	M	12TH STD	N	N	N	Y	Y	N	N	N	19.9	43	8	30
SUBRAMANIAN	70	M	12TH STD	N	N	Y	N	N	N	N	N	34.1	54	8	30
SUNDARI	65	F	3RD STD	N	N	Y	Y	N	N	N	N	23.7	46	7	30
SURESH	66	M	10TH STD	Y	Y	N	N	N	N	N	N	21.65	43	7	30
THAMARAI	66	F	UNEDUCATED	N	N	Y	N	N	N	N	N	26.1	53	8	29
THANGAPANDIYAN	76	M	UNEDUCATED	Y	N	Y	N	N	N	Y	N	23.5	44	7	28
THENDRAL	70	F	UNEDUCATED	N	N	N	Y	N	Y	N	N	26.5	50	7	26
THENMOZHI	71	F	UNEDUCATED	N	N	Y	N	N	N	N	N	23	51	8	30
THYAGARAJAN	72	M	UNEDUCATED	N	Y	Y	N	N	N	N	N	19.5	53	7	28
VADIVEL	66	M	UNEDUCATED	Y	Y	N	N	Y	N	N	N	21	32	6	24
VALLI	80	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	19	37	6	24
VALLIAMAL	78	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	19.4	53	7	28
VEERAMANI	80	M	UNEDUCATED	N	N	Y	Y	Y	N	Y	N	24	31	4	21
VEERAMMAL	64	F	5TH STD	N	N	Y	Y	Y	N	N	N	22	33	5	26
VEERAPANDI	64	M	10TH STD	Y	N	N	Y	N	Y	N	N	20.1	42	7	30
VENI	64	F	UNEDUCATED	N	N	Y	Y	Y	N	N	N	26.8	35	7	27
VENKETESAN	73	M	4TH STD	Y	Y	Y	N	N	N	N	N	23.9	44	7	30
VENKIAH	63	M	10TH STD	Y	N	Y	N	Y	N	N	N	24.4	45	8	30
VENTESAN	69	M	UNEDUCATED	Y	N	Y	Y	Y	N	N	N	19.9	35	10	28
VIJAYA	66	F	4TH STD	N	N	Y	N	N	N	Y	N	24.2	46	6	30
VIJAYANTHI	60	F	5TH STD	N	N	Y	Y	Y	Y	N	N	24.21	46	6	30
VIOLET MARY	62	F	8TH STD	N	N	Y	Y	Y	Y	N	N	26.8	36	6	30