

Liver Function and Lipid Profile in adult Men in Eastern Zone of Kogi State, Nigeria

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Abstract

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are enzymes found mainly in the liver. When the liver is diseased or damaged, additional aspartate aminotransferase and alanine aminotransferase are released into the blood stream, causing the levels of the enzymes to rise. Therefore, the amount of aspartate aminotransferase and alanine aminotransferase in the blood is directly related to the extent of the tissue damage. Alkaline phosphatase (ALP) in mammals is found mainly in the liver. Increase in serum alkaline phosphatase level is associated with malignant biliary obstruction, primary biliary cirrhosis, primary sclerosing cholangitis, hepatic lymphoma and sarcoidosis. Cholesterol is component of plasma membranes. Triglycerides are found in adipose tissue, some triglycerides are stored in the blood to provide fuel for the muscles. This work was aimed at providing information on the values of liver function indices and lipid profile in apparently healthy ageing men residing within Kogi Eastern zone of Nigeria. This is as a result of the curiosity to know and make comparison between their parameters with those of the standard/reference or normal laboratory values. The subjects were divided into different groups 25 – 75 years and the mean and standard deviation of each parameter was calculated. Test for Liver function and lipid profile were carried out to determine the level of alkaline phosphatase, AST, ALT, TG, LDL, HDL and VLDL. There was a significant increase ($p < 0.05$) in the alkaline phosphatase of group 6 (75 years and above) when compared to the standard while the AST and ALT showed no significant difference. The lipid profile showed no significant difference as they were in tandem with the reference values.

Keywords: Low Density Lipoprotein, High Density Lipoprotein, Triacylglycerides, Very Low Density Lipoprotein, Aspartate Aminotransferase, Alanine Aminotransferase

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INTRODUCTION

Ageing affects different organs, tissues, and cell types in the same organism in different ways; that is, the extent of age-associated perturbations of structure and function is site-specific. The effects of aging on the mammalian liver have not been clearly resolved. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are enzymes found mainly in the liver, but also found in the red blood cells, heart cells, muscle tissue, pancreas and kidneys. When body tissues or an organ such as the liver or heart is diseased or damaged, additional aspartate aminotransferase and alanine aminotransferase are released into the blood stream, causing the levels of the enzymes to rise. Therefore, the amount of aspartate aminotransferase and alanine aminotransferase in the blood is directly related to the extent of the tissue damage (Sampson *et al.*, 1980). Alkaline phosphatase (ALP) in mammals is found mainly in the liver and bones. Increase in serum alkaline phosphatase level known as hyperalkaline-phosphatasemia is associated with malignant biliary obstruction, primary biliary cirrhosis, primary sclerosing cholangitis, hepatic lymphoma and sarcoidosis.

The lipids (total cholesterol, triglycerides, low-density lipoprotein [LDL] cholesterol, and high-density lipoprotein [HDL] cholesterol) levels are similar in both genders in all age groups before adolescence (Bagatell & Bremner, 1995). In boys, after puberty, the levels of total cholesterol, LDL cholesterol, and triglycerides increase slightly whereas the plasma level of HDL cholesterol decreases (Bagatell & Bremner, 1995). This negative influence of androgens in men and the positive influence of estrogens in women are responsible, at least in part, for higher impact of atherogenesis in men compared with women (Bagatell & Bremner, 1995; Zmuda *et al.*, 1997). Aging in men enhances this negative influence of androgens on the lipid profile (Khaw & Barrett-Connor, 1991; Zmuda *et al.*, 1997). The low levels of testosterone and high levels of free androgens index (defined as testosterone (nmol/L)/SHBG (nmol/L) × 100) are associated with the atherogenic lipid profile in men (Andersen *et al.*, 2000; Khaw & BarrettConnor, 1991; Zmuda *et al.*, 1997).

Cholesterol is component of plasma membranes; it is used to synthesize hormones and forms bile acids essential for digestion. Triglycerides are found in adipose tissue, some triglycerides are stored in the blood to provide fuel for the muscles (Sidhu and Naugler, 2012). High density lipoprotein (HDL) carries cholesterol in the blood and is considered beneficial because it removes excess cholesterol from the circulation and arterial walls and disposes of it; hence HDL is known as the “good cholesterol” The higher the level of HDL, the lower the risk of heart disease. Low density lipoprotein (LDL) carries cholesterol in the blood, but instead of disposing of excess cholesterol, LDL deposits excess in blood vessel walls. It is also known as the “bad cholesterol”. Very low density lipoprotein (VLDL) carries triglycerides in the body. Increased VLDL level is associated with increased risk of arteriosclerosis and coronary artery disease.

This work was aimed at providing information on the variations of Liver function parameters and lipid profile among age men of Kogi East senatorial district.

MATERIAL AND METHODS

Experimental Design

The study was carried out in Kogi east comprising of Idah, Dekina and Ankpa local government areas. Blood samples were collected from volunteered donors (men) aged 25-75 years. Questionnaire was used to collect the demographic data from the subjects for

analysis. Sixty five questionnaires were given out; five were considered invalid due to errors in filling the form by the respondents. Ten (10) questionnaires were given to control subjects from people aged 25 – 34 years old. All the questionnaires were accepted and used as being valid for the analysis.

Sample Preparation

Blood samples were collected from the volunteered donors; men aged 25 years and above through venipuncture, into EDTA coated tubes and plain tubes for the biochemical test. The samples were centrifuged at 4000 rpm for 5 minutes using uniscope laboratory centrifuge model SM 112 to obtain the serum required for analysis.

Determination of activities of serum Alkaline Phosphatase

The method described by Englehardt (1970) was used to determine the activity of alkaline phosphatase. 1ml of reconstituted working reagent (diethanolamine buffer, MgCl₂ and p-nitrophenylphosphate) was added to 0.02ml of sample mixed and timer started simultaneously. The absorbance was read after 1, 2 and 3 minutes at 405 nm.

Calculation

ALP activity (U/L) = $2760 \times \Delta A_{405\text{nm}}/\text{min}$

Determination of the activities of serum Alanine Aminotransferase (ALT)

According to Reitman and Frankel, (1957), 0.1 ml of sample was added to 0.5 ml of working reagent R1 (phosphate buffer, L-aspartate, α -oxoglutarate) after this, mixed and incubated at 37°C for 30 minutes with 0.5 ml of working reagent 2 (R2) (2,4-dinitrophenylhydrazine). This was mixed and allowed to stand for 20 minutes at room temperature of between 20°C-25°C. The reaction was terminated with the addition of 5 ml of NaOH and the absorbance was read after 5 minutes at 546 nm.

Calculation

AST activity (U/L) = Change in Absorbance X concentration

Determination of the activities of serum Aspartate Aminotransferase (AST)

The procedure described by (Reitman and Frankel, 1957) was used for the determination of Aspartate amino transferase activity. To 0.1 ml of sample, 0.5 ml of working reagent R1 (phosphate buffer, L-aspartate, α -oxoglutarate) was added, mixed and incubated at 37°C for 30 minutes with 0.5 ml of working reagent 2 (R2) (2,4-dinitrophenylhydrazine). This was mixed and allowed to stand for 20 minutes at room temperature of between 20°C-25°C. The reaction was terminated with the addition of 5 ml of NaOH and the absorbance was read after 5 minutes at 546 nm.

Calculation

AST activity (U/L) = Change in Absorbance X concentration

Determination of serum cholesterol

The method of Friedewald *et al.* (1972) was used to determined serum total cholesterol concentration.

Determination of High Density Lipoprotein:

The method of Friedewald *et al.* (1972) was used to determined serum total cholesterol concentration.

Determination of Serum Triglyceride Concentration:

The method of Jacobs and Van Denmark (1960) was employed in the determination of serum Triglyceride concentration.

Determination of Low density lipoprotein

Values for low density lipoprotein were calculated using Friedwald equation below,
 $LDL\ Cholesterol = Total\ cholesterol - Triglycerides - HDL\ Cholesterol$

Statistical Analysis

Data were initially sorted out manually and tabulated and then entered into the computer using bonferon comparison test of graph pad with statistical software and the mean value determined, and the differences were considered significant if $p < 0.05$. Comparison of mean values in relation to age distribution was done using analysis of variance (ANOVA).

RESULTS

Variation of the serum alkaline phosphatase (ALP) with age

There were increased serum alkaline phosphatase in groups 3, 5 and 6 compared to the control group 1 while decreased in groups 2 and 4, though not significant. This could indicate a slight damage to the liver.

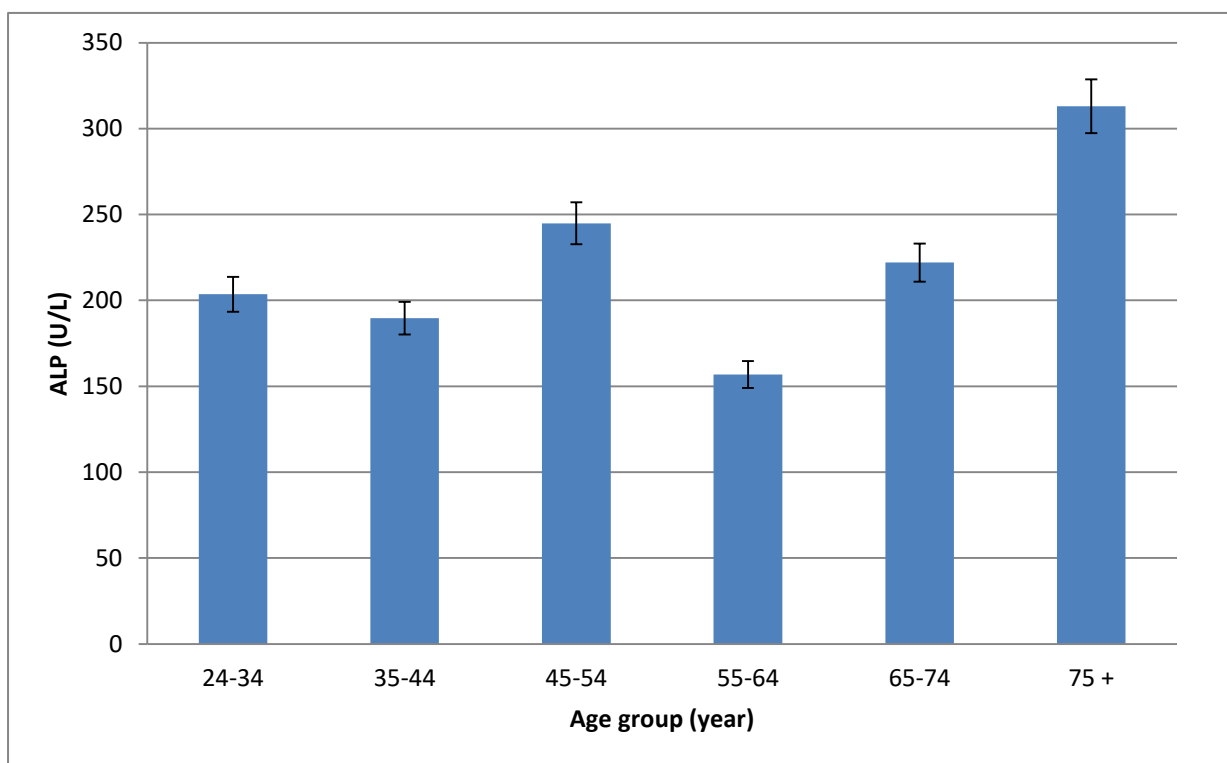


Fig. 1: Barchart showing Alkaline Phosphatase activity in aged men, n=60

Variation of the serum Alanine transaminase (ALT) with age

Figure 2 shows significant increase in serum ALT in all groups. Group 2 and 4 have the highest concentration compared to control group 1.

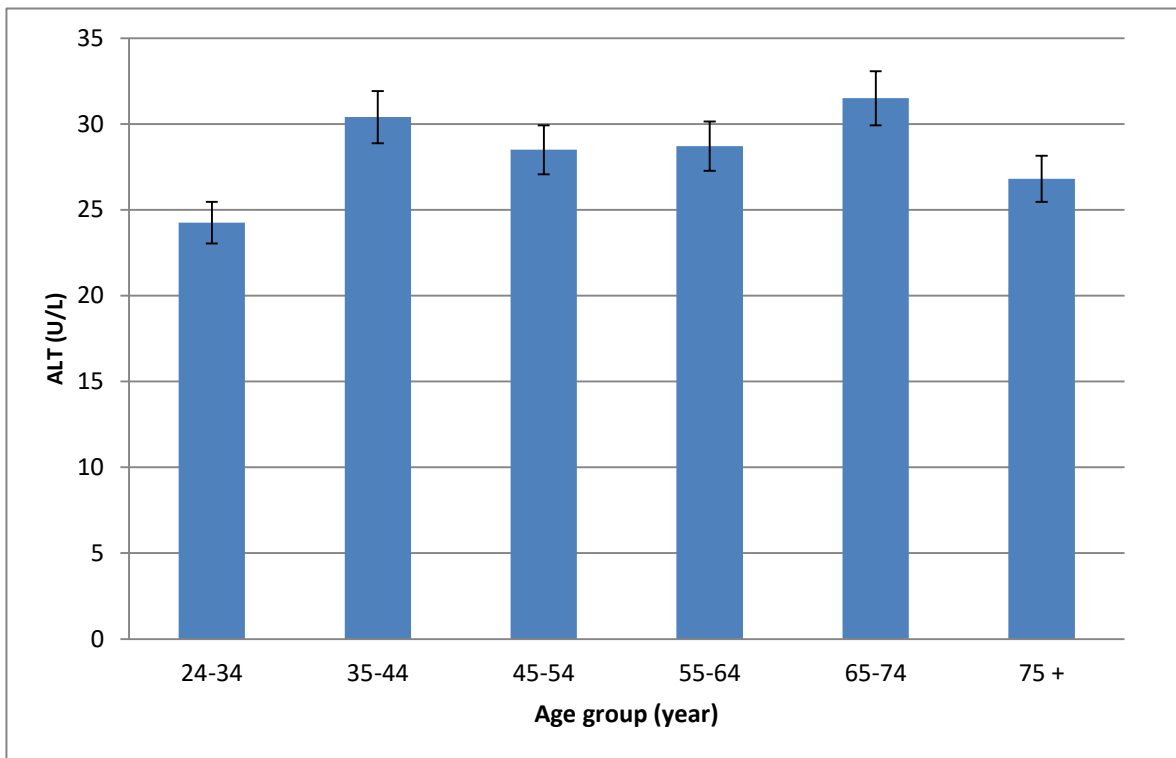


Fig. 2: Bar chart showing Alanine Aminotransferase activity in aged men, n=60

Variation of the total cholesterol with age

Figure 3 shows increases in the total cholesterol in all groups. Group 3 (45-54 year) have a significant increase ($p < 0.05$).

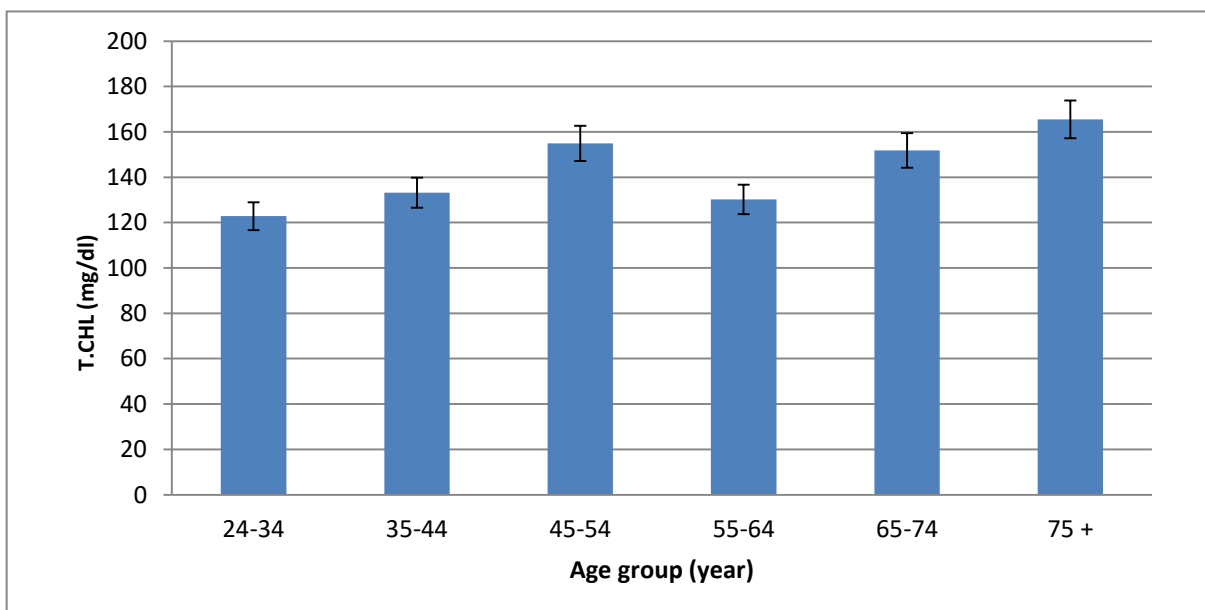


Fig. 3: Bar chart showing total cholesterol concentration in aged men, n=60

Variation of the high density lipoprotein with age

In fig.4, there is a steady significant increase ($p < 0.05$) in HDL in group 3 and 4 when compared to the control group 1 while a significant decrease also occurred in group 5 ($p < 0.05$).

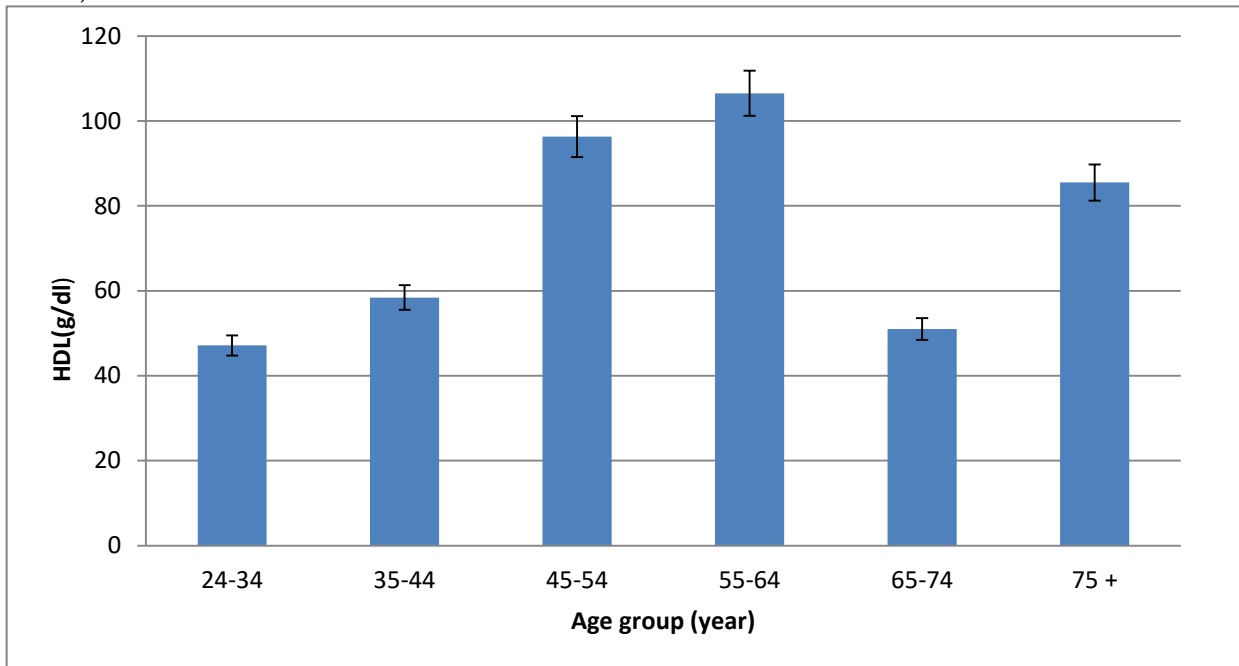


Fig. 4: Bar chart showing High Density Lipoprotein in aged men, n=60

Variation of the low density lipoprotein with age (LDL)

Figure 5 shows significant increase in LDL in group 3 and 5 ($p < 0.05$) while group 2 and 4 shows decrease. These increases could be as a result of the social and nutritional lifestyle such as eating food rich in saturated fats, smoking, etc.

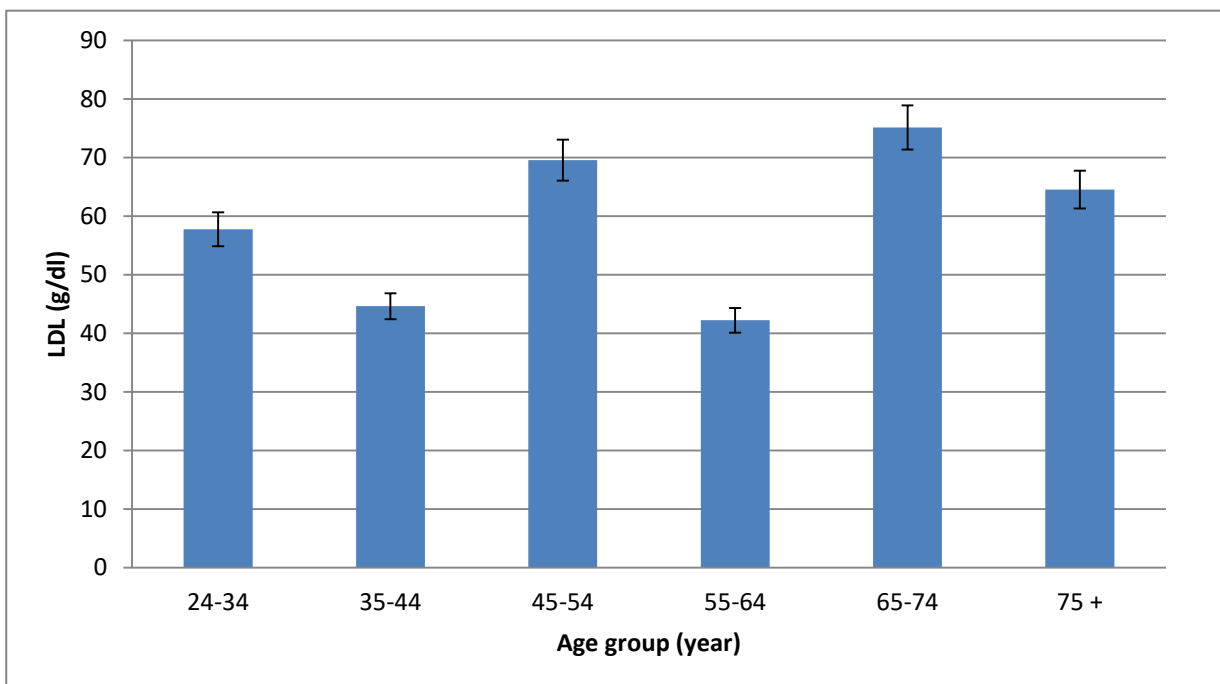


Fig. 5: Bar chart showing Low Density Lipoprotein in aged men, n=60

Variation of the very low density lipoprotein with age

Figure 6 shows a significant increase ($p < 0.05$) in VLDL in group 2 and 4 and decreases in group 5 and 6 showing that VLDL increases with age. The significant increase of VLDL in group 4 could be as a result of high saturated fatty food content in the diets.

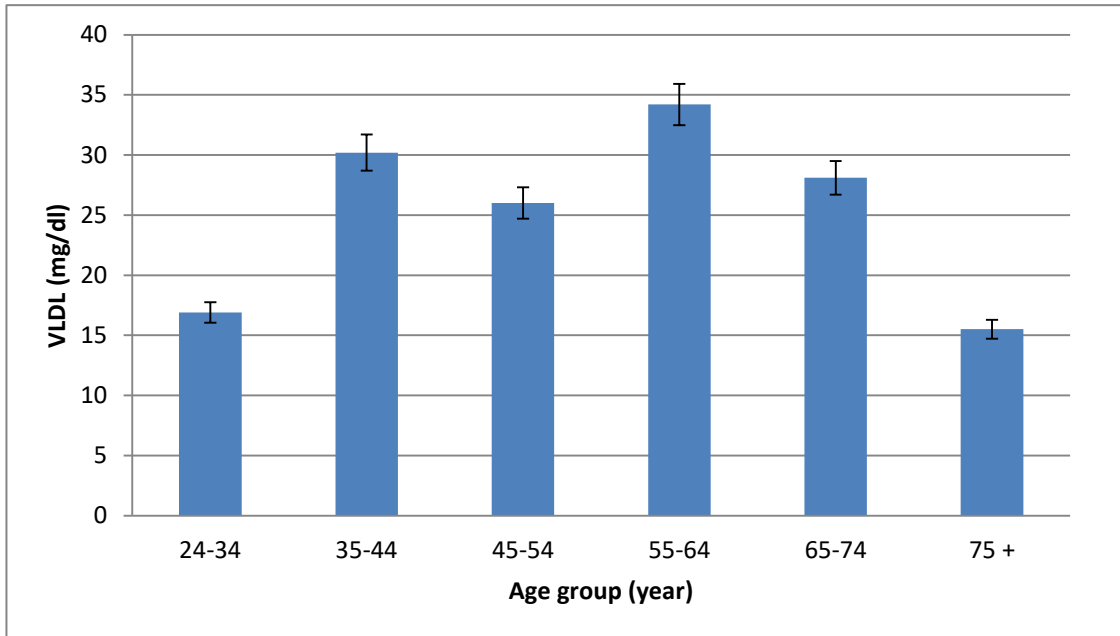


Fig.6: Bar chart showing Very Low Density Lipoprotein in aged men, n=60

Variation of the triglyceride with age

Figure 7 shows an increase in all groups except group (75- above) which shows a decrease in triglyceride when compared to the control group 1. This could be as a result of consumption of high fat calorie content.

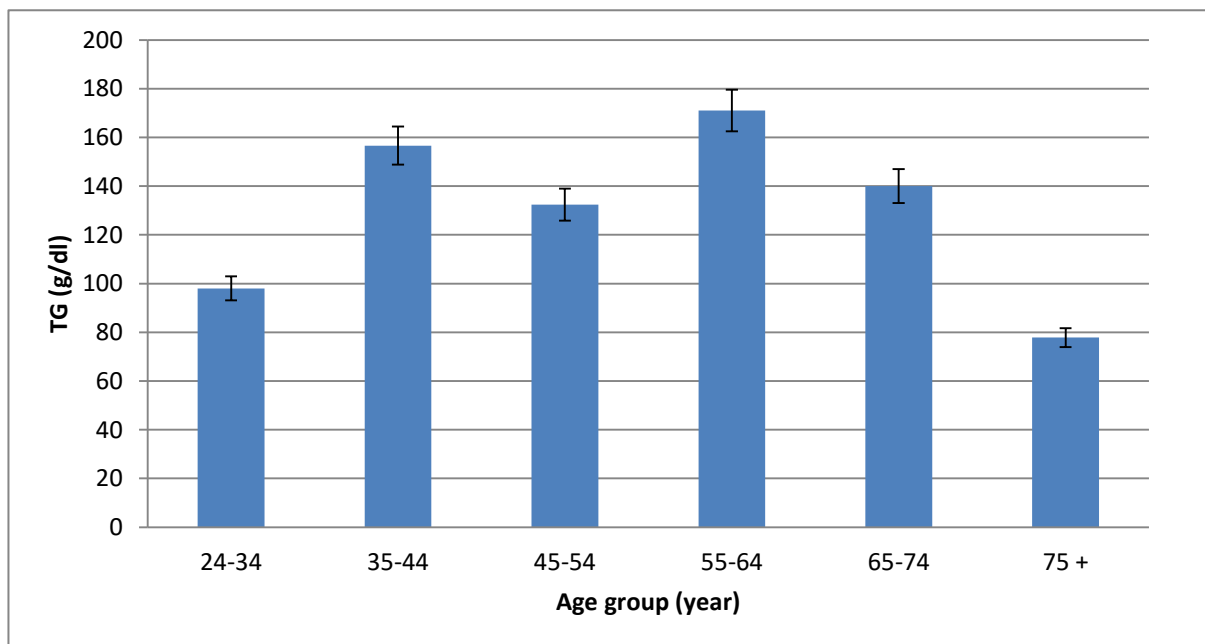


Fig. 7: Bar chart showing triglycerides concentration in aged men, n=60

DISCUSSION

Ageing affects different organs, tissues, and cell types in the same organism in different ways; that is, the extent of age-associated perturbations of structure and function is site-specific. Aging in men enhances this negative influence of androgens on the lipid profile (Khaw & Barrett-Connor, 1991; Zmuda et al., 1997). The low levels of testosterone and high levels of free androgens index (defined as testosterone (nmol/L)/SHBG (nmol/L) × 100) are associated with the atherogenic lipid profile in men (Andersen et al., 2000; Khaw & Barrett-Connor, 1991; Zmuda et al., 1997).

A significant increase in the high density lipoprotein occurred between the ages of 55 - 64 and then decreased. HDL is also known as "good cholesterol". This probably indicated that the subjects in the age range were not at the risk of coronary heart disease. HDL helps to prevent atherosclerosis. Low density lipoprotein (bad cholesterol) helps to transport the contents of lipid molecules into the arterial walls, attract macrophages and thus drive atherosclerosis, while very low density lipoprotein showed no significant difference ($p < 0.05$). Variation occurred in the triglyceride concentration with age as there was an increase between ages 55 - 64 while a decreased occurred as the age increased. High triglycerides could be as result of poorly controlled diabetes, obesity, drinking of alcohol and regularly eating more calories than one can burn.

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