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Full Length Research

Changes in serum total and lipoprotein cholesterol level induced by cigarette smoking

Okere O. L.* and Orogu J. O.

Department of Science Laboratory Technology, School of Science and Technology, Delta State Polytechnic Ozoro, Delta State, Nigeria.

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Smoking is associated with high risk of developing various forms of arteriosclerosis. This present study was carried out to investigate the changes in serum total and lipoprotein cholesterol level induced by cigarette smoking. Fifteen smokers and fifteen non-smokers who reside in Abraka town, Delta State, Nigeria were recruited for this research. The subjects' age bracket lies between 20-30 years and they have been smoking for about 2- 10 years. Fasting venous blood was collected and the serum obtained after centrifugation was used for the biochemical analysis. The results shows that there was a significant difference (P<0.05) in the total cholesterol level of smokers (254.13±9.42 mg/dL) as compared with the smokers (263.13±7.80 mg/dL). High density lipoprotein cholesterol of the non- smokers is significantly (P<0.05) higher (28.18±3.57 mg/dL) as compared with the smokers group (18.24±2.94 mg/dL). The ratio of HDL: LDL (a cardiovascular risk marker) is higher in non-smokers (0.130) compared to smokers (0.08). This shows that smokers may be prone to various form of arteriosclerosis. It was therefore concluded that smokers are at high risk of developing cardiovascular disease and recommended that the habit of smoking should be discouraged.

Key words: Smokers, non smokers, serum, cholesterol level, lipoprotein, arterioscleriosis.

INTRODUCTION

Smoking is socially accepted in various communities and cigarette consumption has drastically increased around the world (WHO, 2003). Recent study carried out by a local research group indicated an increased in the rate of teenage smokers. Harmful effects of smoking appear at an early age, seriously affecting the brain, gastrointestinal systems, immune functioning and respiratory systems (Aoshida et al., 2003).

Dependency on smoking also develops very rapidly, which might not be expected by new consumers (Russell, 1990). It has been estimated that smoking will kill about 10 million people by the year 2020, if the current trend of smoking persists (WHO, 2005). Smoking is associated with many unwanted effects and can predispose one to a multitude of disease such as atherosclerosis (Kannel, 1981) and dyslipidaemia (Sharma et al., 2005).

A lipoprotein is a biochemical assembly that contains both proteins and lipids whose function is to transport water-insoluble lipids in the water-based bloodstream. The lipids of their derivatives may be covalently or non-covalently bonded to proteins. Many enzymes, transporters, structural proteins, antigens, adhesions and toxins are lipoproteins. Examples include the High density (HDL) and the low density (LDL) lipoproteins which enable fats to be carried in the bloodstream, the transmembrane proteins of mitochondrion and chloroplast, and bacteria lipoprotein (Skipski, 1972).

Cholesterol is a waxy steroid metabolite found in the cell membranes and transported in the blood plasma of all animals (Leah, 2009). It is an essential structural component of mammalian cell membrane, where it is required to established proper membrane permeability

*Corresponding author. E-mail: tsetimi@gmail.com

and fluidity. In addition, cholesterol is an important component for the manufacture of bile acids steroid hormones, and fat soluble vitamins including vitamin a, Vitamin D, Vitamin E, and Vitamin K. Cholesterol is the principal sterol synthesized by animals, but small quantities are synthesized in other Eukaryotes, such as plants and fungi. It is almost completely absent among prokaryotes which include bacteria (Pearson et al., 2003). Although cholesterol is an important and necessary molecule for animals, a high level of serum cholesterol is an indicator for diseases such as heart disease. Cigarette smoking is also a potent resource of free radicals (Ross, 1993) which can also deplete scavengers of reactive oxygen species (RDS) accentuating oxidative damage (Finkel et al., 2000). The free radicals produce physiologically, have the propensity of oxidizing low density lipoprotein (LDL) thereby increasing risk of atherogenicity (Chisolm, 1991). Smokers generally demonstrate increase level of pseudo halide thiocyanate (SCN) (Vos et al.,1998), a major risk factor of cardiovascular diseases. SCN can in turn enhance the activity of mycloperoxidase by acting as a catalyst at the same time and thereby leading to the oxidation of low density lipoprotein (LDL) (Exner et al., 2004). Therefore, this study was carried out to evaluate the changes of serum total and lipoprotein cholesterol level induced by cigarette smoking and to estimate the serum total cholesterol level, determine serum HDL cholesterol level and estimate serum low density lipoprotein (LDL) levels in young male adult smoking cigarettes.

METHODOLOGY

Thirty male free-living undergraduates in apparent good health were selected at random for the study. They were divided into two groups of fifteen each (that is, 15 smokers and 15 non-smokers). All participants reside in Abraka town, Delta State, Nigeria.

Five milliliters of venous whole blood was collected from each volunteer with the aid of a syringe into a clean sterile plain tube. The blood sample was allowed to clot and the serum was obtained after centrifugation at 1200 × g for five minutes at room temperature. The serum was decanted into another sterile plain tube and was kept frozen at -20°C until analyzed.

Biochemical analysis

Estimation of serum total cholesterol (Tarbutton and Gunter, 1974)

Principle: The enzymatic reaction sequence employed in the assay of cholesterol is as follows:

$$\text{Cholesterol Esters} \xrightarrow{\text{C. Esterase}} \text{Cholesterol + fatty acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{C. oxidase}} \text{Cholesten-3-one} + H_2O_2$$

$$2H_2O_2 + 4\ \text{Aminoantipyrine} + \text{P-HBS} \xrightarrow{\text{H peroxidase}} \text{Quinoneimine} + 2H_2O$$

(Red dye)

Label test tubes: blank, standard, control and test. Pipette 1.0 ml of reagent to all tubes and pre-warm at 37°C for at least 2 min. Add 0.01 ml (10ul) of sample to respective tubes, mix and return to 37°C. Zero spectrophotometer with reagent blank at 520 nm. Read and record absorbances of all tubes.

Calculations:

A (test) * conc. Of standard+ Concentration of test (mg/dl)
A standard (mg|dl)

Determination of serum HDL-cholesterol (Tietz, 1986)

Principle: When serum is reacted with polyethene glycol reagent, all the low and very low density lipoprrptein (LDL and VLDL) are precipitated. The HDL fraction remains in the supernatant. The supernatant is then treated as a sample for cholesterol assay.

Procedure:

Reagents and supernatants were allowed to stand at room temperature as shown in Table 1. The content of each tube was thoroughly mixed and allowed to stand for 5 min. at 37°C. Absorbance was recorded at 520 nm against reagent blank.

Calculation:

$$\text{HDL Cholesterol (mg/dl)} = \frac{A(\text{test})}{A(\text{standard})} * \text{conc of standard} * 1.1$$

Estimation of serum low density lipoprotein (LDL) (Friedwald et al., 1972)

LDL- cholesterol was estimated with Friedwald formular.
LDL- cholesterol= Total cholesterol- HDL-1/5 TAG.

RESULTS AND DISUSSION

The results of the biochemical parameters measured which include; total cholesterol, high density lipoprotein cholesterol (HDL-cholesterol) and low density lipoprotein cholesterol (LDL-cholesterol) are presented in Table 2.

Table 1. Reagents and supernatants test.

	Blank	Standard	Test	Control
Reagent	1 ml	1 m	1 ml	1 ml
Distilled water	25 µl			
Standard		25 µl		
Supernatant			25 µl	25 µl

Table 2. Levels of total cholesterol, high density lipoprotein cholesterol, low density lipoprotein cholesterol and HDL: LDL ratio of smokers and non-smokers.

Groups	Total cholesterol (mg/dl)	HDL-cholesterol (mg/dl)	LDL-cholesterol (mg/dl)	Ratio of HDL:LDL
Smokers	254.4±9.42[a]	18.24±2.94[a]	219.19±13.23[a]	0.08
Non-smokers	263.13±7.80[b]	28.18±3.57[b]	216.54±16.51[a]	0.130

The values are expressed as mean ± SD (with n=15). Means not sharing a common superscript letter down a column differ significantly at P<0.05.

The results in Table 2 indicate that smokers, which expressed significantly (P<0.05) reduced total cholesterol level (254.43±9.42) as compared with non-smokers group is significantly (P<0.05) higher (28.18±3.75) than the smokers group (18.24±2.94). The results also show that the low-density lipoprotein cholesterol level of both smokers and non-smokers were comparable (that is, P>0.05) and the ratio of HDL: LDL was lower in smokers when compared with non-smokers.

Smoking is socially accepted in various communities and cigarette consumption has drastically increased around the World (WHO, 2003). The harmful effects of smoking appear at an early age, seriously affecting the brain, gastrointestinal systems, immune functioning and respiratory systems (Aoshida et al., 2003).

Smoking has been reported to lower the ratio of high density lipoprotein cholesterol to low density lipoprotein cholesterol as this according to Narkiewicz et al. (2005) predispose smokers to risk of developing various forms of arteriosclerosis. The result of this study is in line with the aforementioned statement. The ratio of HDL: LDL for smokers and non-smokers is 0.08:0.130. This implies that smokers may be prone to various forms of arteriosclerosis when compared with non-smokers.

Conclusion

It can therefore be concluded from this research that smokers are at high risk of developing cardiovascular disease when compared to non-smokers.

RECOMMENDATION

Since cigarette smoking has been reported to be a potent source of free radicals (Ross, 1993), which can also deplete scavengers of reactive oxygen species, It is recommended that further studies of this nature should focus on the antioxidant levels of the study groups and smoking should be discouraged since it can lead to the development of cardiovascular disease.

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