Effect of Diet on Serum Lipid Profile in Healthy Nigerians

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Abstract

Background: Serum lipid is a screening tool in different disease conditions such as diabetes mellitus, renal disease and ischemic heart disease. Its determination is traditionally done after an overnight fast. However, there is a growing argument that fasting blood specimen may not be absolutely necessary for a plasma lipid analysis. In order to test this concept, this study was undertaking to evaluate the effect a standard Nigerian meal will have on serum lipid at 30 min and 2 hr postprandial, and also determined the cardiovascular risk ratio (CRR) and the artherogenic index (AI) at fasting, 30 min and 2hr postprandial. Methods: A cross-sectional study that involved fifty healthy subjects aged 20-29 years ((male and female). Participants with medical illness were excluded. The subjects were educated on the study and consent form administered. Subjects fasted over night for 12 hour. Blood was taken from the peripheral veins at the 12th hour after fasting and were thereafter served standard Nigerian meal (rice and beef) that weighed 450 g with (250 ml) water. At 30 min and 2 hr blood was collected for lipid analysis. Serum lipid assays were performed using Reagent kits manufactured by Randox (England) and LDL-C was determined by Friedewald Calculation.

Results: There was a significant (p<0.05) increase in total cholesterol at 30 min and 2 hr postprandial compared to fasting, while serum triglyceride was significantly (p<0.05) reduced at 30 min and 2 hr. There were no changes in serum HDL-cholesterol at fasting, 30 min and 2 hr. The LDL-C and non-HDL-C were significantly (p<0.05) increased at 30 min and 2 hr when compared to fasting. The cardiovascular risk ratio at 30 min and 2 hr postprandial was significantly (p<0.05) reduced when compared to fsating. The artherogenic index ratio were reduced at 30 min and 2 hr postprandial when compared to fasting. **Conclusion:** If the risk assessment of an individual is based on values of serum total cholesterol, LDL cholesterol and non HDL cholesterol, fasting specimen will be most desirable. But if the risk assessment is to be considered objectively using the CRR and AI, a fasting blood specimen will not be absolutely necessary especially when the physician needs to take an urgent decision about a patient's clinical condition.

Keywords: Fasting, diet, postprandial, lipid profile, cardiovascular risk ratio, artherogenic index

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No conflicts of interest have been declared by the authors

Introduction

Lipid profile is a group of tests that are often required to determine the possible risk of coronary heart disease and other cardiovascular risks. They are tests that have been shown to be good indicators of the possibility of developing heart attack or stroke caused by blockage of blood vessels or hardening of the arteries. These lipids include total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), triglyceride (TG), low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C). For many years the determination of a routine lipid profile (total cholesterol, LDL, HDL cholesterol and triglycerides) has been done routinely in the clinical laboratory using a blood specimen that is collected in the fasting state. However, because most of each person's lifetime is spent in the postprandial state, the wisdom of collecting a fasting sample to determine future risk of cardiovascular has been challenged¹. Only a few studies have compared fasting and non-fasting specimens in the context of classification of risk of atherosclerotic cardiovascular diseases (ASCVD) and related health outcomes¹. An increase in the plasma concentration of triglyceride is an established risk factor for cardiovascular disease², most likely the cholesterol content of the triglyceriderich lipoproteins or remnant cholesterol is causally associated with ischemic heart disease^{2,3}.

There has been an assumption that triglycerides should be measured in the fasting state because the concentrations of fasting triglycerides are lower and possibly less variable from measurement to measurement compared with triglycerides measured in the non fasting state⁴. The current practice of using fasting lipid profiles was challenged in 2007 by two studies that in combination showed that non fasting triglycerides in predicting risk of cardiovascular disease^{5,6}. Lipid profiles are conventionally measured in the fasting state , however, there are advantages to using non fasting samples rather than fasting samples for lipid profile measurement^{7,8}. The most obvious advantage of non fasting rather than fasting lipid profile measurement is that the blood sampling process is simplified for patients as well as for general practitioners and hospitals ⁹.

Materials and Method

It was a cross-sectional study that involved fifty healthy subjects: 25 males and 25 females, aged between 20-29 years. They were selected from students and staff of the University of Benin, Benin City, Nigeria. The study protocol was carefully explained to all of them, before they voluntarily signed a written consent. A questionnaire was used to collect information from all the participants. The information included; age, sex, smoking habit, drinking habit, marital status and occupation. Blood pressure, weight and height measurements were taken and Body mass index (BMI) calculated, to exclude overweight and obese individuals as well as hypertensive individuals.

Ethical Consideration: Ethical approval was obtained from the hospital research and ethics committee. The day preceding the test, participants were made to fast overnight (12 hr). Blood was collected from the peripheral veins in an aseptic procedure using a 10 ml syringe into appropriate specimen containers and this was the fasting blood sample at zero (0) time. This was followed by serving a standard Nigerian meal (rice and beef) that weighed 450g. A measured amount of water (250 ml) was given to the participants to drink and blood collected at 30 min and 2 hr postprandial. The blood samples were centrifuged at 5000 rpm, serum separated and stored at -20 °C for lipid profile analysis.

Serum lipid analysis was by using already standardized and well established methodologies. All the assays were performed using Randox (England) kits. Total cholesterol (TCHL) analysis was by the modified Liebermann-Burchard's method¹⁰, and HDL-C by precipitation method.¹¹ Triglyceride was assayed using enzymatic colorimetric method¹² and LDL-C was calculated by indirect method (Friedewald equation)¹³. All the concentrations are expressed in mg/dl.

Results

The lipid profile (total cholesterol, triglycerides, high density lipoprotein cholesterol and low density lipoprotein cholesterol) of fifty normal subjects were determined. The results in Table 1 showed that there was a significant (p<0.05) increase in serum total cholesterol at 30 min and 2hr postprandial when compared to fasting. However, the total cholesterol at 2 hr

postprandial was significantly (p<0.05) decreased when compared to the 30 min postprandial value. The serum triglyceride was significantly (p<0.05) decreased at 30 min and 2hr when compared to fasting. The LDL cholesterol and non-HDL cholesterol were significantly (p<0.05) increased in the 30 min and 2 hr postprandial when compared to the fasting, though there was decline in these parameters at the 2 hr towards the fasting levels. The serum HDL cholesterol was not significantly different (p>0.05) at fasting, 30 min and 2 hr postprandial. In Table 2 there was a significant (p<0.05) reduction in the



Fig.1: Showing the concentrations of lipid profile at fasting, 30 min and 2 hr postprandial. Values with different superscripts are significantly different when p < 0.05

TCHL = total cholesterol; TG = triglyceride; HDL-C = high density lipoprotein cholesterol; LDL-C = low density lipoprotein cholesterol; Non-HDL-C = non-high density lipoprotein cholesterol

Sample	Fasting	30 min	2 hr					
TCHL (mg/dl)	123.45 ± 4.4^{a}	185.75 ± 6.99^{b}	$166.30 \pm 8.84^{\circ}$					
TG (mg/dl)	114.24 ± 2.64^{a}	90.40 ± 8.04^{b}	92.91 ± 6.20^{b}					
HDL-c (mg/dl)	57.74 ± 3.95^{a}	52.68 ± 3.46^{a}	49.79 ± 2.54^{a}					
LDL-C (mg/dl)	52.88 ± 4.29^{a}	114.99 ± 7.77^{b}	100.43 ± 9.16^{b}					
Non-HDL-C (mg/dl)	65.72 ± 6.05^{a}	133.07 ± 7.54^{b}	116.51 ± 9.68^{b}					

 Table 1: The effect of a standard Nigerian diet on the lipid profile of healthy subjects (Male and female combined) at fasting, 30 min and 2 hr postprandial

Values with different superscripts are significantly different when p < 0.05

TCHL = total cholesterol; *TG* = triglyceride; *HDL-C* = high density lipoprotein cholesterol; LDL-C = low density lipoprotein cholesterol; Non-HDL-C = non-high density lipoprotein cholesterol

Table 2:	The effect of a standard Nigerian diet on the artherogenic index and cardiovascular
risk ratio	n healthy subjects at fasting, 30 min and 2 hr postprandial

	Artherogenic Index Ratio				Cardiovascular Risk Ratio			р
	Fasting	30 min	2 hr	Ρ	Fasting	30 min	2 hr	
Male (M)	2.63±0.37 ^a	2.17±0.35 ^a	2.20±0.32ª	0.580	0.45±0.04ª	0.32±0.04ª	0.35 ± 0.04^{a}	0.067
Female (F) M+ F	2.51±0.25ª	2.75±0.70 ^a	2.02±0.17ª	0.495	0.58 ± 0.08^{a}	0.28 ± 0.03^{b}	0.36 ± 0.05^{b}	0.002
combined	2.57±0.22ª	2.47 ± 0.40^{a}	2.11±0.17ª	0.479	0.52±0.05ª	0.30 ± 0.02^{b}	0.35 ± 0.03^{b}	0.000

Values are significantly different when p < 0.05

cardiovascular risk ratio in the females at 30 min and 2 hr postprandial when compared to fasting. When the male and female were combined the cardiovascular risk ratio was also significantly (p<0.05) reduced at 30 min and 2 hr postprandial when compared to the fasting.

In figure 2 there were significant (p<0.05) increase in the total cholesterol of males and females in the 30 min and 2 hr postprandial when compared to fasting. While in figure 3 the 30 min, 2hr postprandial triglyceride levels in males were reduced compared to the fasting, but in the females there was a significant

(p<0.05) difference between the fasting, 30 min and 2 hr postprandial. The HDL-C represented in figure 4 showed that there were no significant (p>0.05) changes in the male and female concentrations at 30 min and 2 hr when compared to fasting. However, figures 5 and 6 showed LDL and non-HDL cholesterol in the male and female were significantly (p<0.05) increased in the postprandial (30 min and 2 hr) states compared to the fasting. In figure 7 there was a significant (p<0.05) reduction in the cardiovascular risk ratio in the female at 30 min and 2 hr postprandial when compared to fasting.







Fig. 3: The concentration of triglyceride at fasting, 30 min and 2 hr postprandial in males and females. Values with different superscripts are significantly different when p < 0.05



Fig. 4. The concentration of HDL-cholesterol at fasting, 30 min and 2 hr postprandial in males and females. Values with different superscripts are significantly different when p < 0.05



Fig. 5. The concentration of LDL-cholesterol at fasting, 30 min and 2 hr postprandial in males and females. Values with different superscripts are significantly different when p < 0.05









Discussion

For several years the determination of a routine lipid profile (total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol and triglycerides) has routinely be done in the clinical laboratory using blood specimen collected in the fasting state. A person's life is spent in the postprandial state, hence the rationale for collecting a fasting sample to determine future risk of cardiovascular disease has been challenged¹. In this study we found a significant (P<0.05) increase in serum total cholesterol in 30 min and 2 hr compared to the fasting, though there was a significant (p < 0.05) decrease in 2 hr postprandial value of cholesterol when compared to 30 min. This may imply that the reduction may be time dependent. After normal food intake, concentrations of lipids, lipoproteins and apolipoprotein differ only minimally from the fasting to the non fasting state 7,14.

Non fasting triglyceride as a marker of increased remnant cholesterol appear to be better for predicting risk of myocardial infarction, ischaemic stroke and early death. ^{7,14,15} It has also been reported that in normal individuals, plasma triglycerides increased only slightly after normal food intake compared with fasting concentrations because most of a person's life time is spent in the postprandial state, this triglyceride concentration on average only increase by 0.2-0.4 mmol/L 2-6 hr after eating normal meals and these increases are clinically unimportant ^{9,16,17}.

A superficial analysis suggests that the ingestion of a meal sets in train a series of coordinated changes in fat metabolism that tend both to suppress the entry of endogenous triglycerides and to increase triglycerides disposal from the plasma¹⁸. Our study showed that the plasma triglyceride was significantly (p < 0.05) reduced at 30 min and 2 hr when compared to fasting concentration which seemed to agree with other studies¹⁸. In our opinion serum triglyceride can be determined at 30 min and

2 hr postprandial, as this will not make the attending physician asking his or her patient to come for triglyceride measurement another day fasting as noted in this study.

A study by the US National Health and Nutrition Examination Survey found an average change from fasting concentrations for triglycerides, total and LDL cholesterol after food intake, with no major changes in concentration of HDL cholesterol ^{6,7}. This study showed that there was no significant (p>0.05) changes between the HDL cholesterol of fasting, 30 min and 2 hr postprandial, which is in agreement with the reports of Nordestgaard et al.,⁶ and Langsted et al.,⁷. Remnant cholesterol combined with LDL cholesterol can be assessed as either non-HDL cholesterol or apolipoprotein B². The advantages of using non-HDL-C as a screening tool include the fact that it requires measurement of only total cholesterol and HDL-C, both of which can be measured reasonably accurately in a nonfasting sample.¹⁹ In men, an increase in non-HDL-C has been shown to be associated with an increase in cardiovascular disease mortality.²⁰

We found that there was a significant increase (p<0.05) in LDL cholesterol and Non-HDL cholesterol in 30 min and 2hr when compared to the fasting concentration, though the latter was gradually decreasing towards the fasting level. The arguments that are often presented in favour of use of fasting concentrations, are that triglyceride concentrations are more stable in the fasting than non-fasting state, however, scientific evidence documenting fasting concentrations as better than non-fasting is not available 6,21,22. The serum LDL cholesterol that is calculated according to the original Friedwald equation was developed with fasting individuals and that fasting concentrations have always been used for these measurements and calculations.²³ However, lipid profile is now the only blood test that need a fasting status, because even fasting blood glucose concentrations are being replaced by glycated

haemoglobin (HbA₁c) concentrations¹⁶. In this study we also found that cardiovascular risk ratio were significantly reduced in the 30 min and 2 hr postprandial when compared to the fasting values which may also support the use of postprandial specimens for determining lipid profile on routine clinical visits by patients.

Conclusion

If the risk assessment of an individual is based on values of serum total cholesterol, LDL cholesterol and non HDL cholesterol, fasting specimen will be most desirable. But if the risk assessment is to be considered objectively using the CRR and AI, a fasting blood specimen will not be absolutely necessary especially when the physician need to take an urgent decision about the patient clinical condition.

References

- Rifai N, Young IS, Nordestgaard BG, Anthony S, Wierzbicki AS, Vesper H, Mora S, Stone NJ, Genest J and Miller G. Nonfasting Sample for the Determination of Routine Lipid Profile: Is It an Idea Whose Time Has Come? Clinical Chemistry 2016; 62:3 428-435
- Nordestgaard BG and Varbo A. Triglycerides and cardiovascular disease. Lancet 2014; 384: 626 –635.
- Varbo A, Benn M, Tybjaerg-Hansen A, Jorgensen AB, Frikke-Schmidt R and Nordestgaard BG. Remnant cholesterol as a causal risk factor for ischemic heart disease. J Am Coll Cardiol 2013; 61: 427–436.
- Langsted A and Nordestgaard BG. Nonfasting Lipid Profiles: The Way of the Future. Clinical Chemistry 2015; 61 (9): 1123–1125.
- Bansal S, Buring JE, Rifai N, Mora S, Sacks FM and Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. JAMA 2007; 298: 309 –316

- Nordestgaard BG, Benn M, Schnohr P and Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA 2007; 298: 299 –308
- Langsted A, Freiberg JJ and Nordestgaard BG. Fasting and nonfasting lipid levels: influence of normal food intake on lipids, lipoproteins, apolipoproteins, and cardiovascular risk prediction. Circulation 2008; 118: 2047–2056.
- Nordestgaard BG, Langsted A and Freiberg JJ. Nonfasting hyperlipidemia and cardiovascular disease. Curr Drug Targets 2009; 10: 328 –335.
- Langsted A and Nordestgaard BG. Nonfasting Lipids, Lipoproteins, and Apolipoproteins in Individuals with and without Diabetes: 58 434 Individuals from the Copenhagen General Population Study. Clinical Chemistry 2011; 57: 482–489
- 10. Abell LL, Levy BB, Brodie BB and Kendall FE. A simplified method for the estimation of total cholesterol in serum and demonstration of its specificity. J Biol Chem 1952; 195: 357-366
- Warwick GR, Cheung MC and Albers J. Comparison of current methods for high density lipoprotein cholesterol quantitation. Clin Chem 1979; 25: 596-603
- 12. Jacob NJ and VanDenmark PJ. Enzymatic determination of serum triglyceride. Arch. lochem. Biophy. 1960; 88: 250-255
- 13. Friedewald WT, Levy RI and Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972; 18: 499-502.
- 14. Mora S, Rifai N, Buring JE and Ridker PE. Fasting compared with nonfasting lipids and apolipoproteins for

predicting incident cardiovascular events. Circulation 2008;118: 993– 1001.

- Freiberg JJ, Tybjaerg-Hansen A, Jensen JS and Nordestgaard BG. Nonfasting triglycerides and risk of ischemic stroke in the general population. JAMA 2008; 300: 2142–2152.
- 16. Nordestgaard BG and Tybjærg-Hansen Anne. "Genetic determinants of LDL, lipoprotein(a), triglyceride-rich lipoproteins and HDL: concordance and discordance with cardiovascular disease risk." Current opinion in lipidology 2011; 22 (2): 113-22. doi:10.1097/MOL.0b013e32834477d2.
- 17. Sidhu D and Naugler C. Fasting time and lipid levels in a community based population: a cross-sectional study. Arch Intern Med 2012; 17 (2): 1707– 1710.
- Frayn KN. Insulin resistance and lipid metabolism. Curr Opin Lipidol 1993; 4: 197–204.
- 19. Havel RJ. McCollum Award Lecture, 1993: triglyceride-rich lipoproteins and atherosclerosis — new perspectives. Am J Clin Nutr 1994; 59:795-799.

- 20. Cui Y, Blumenthal RS, Flaws JA, Whiteman MK, Langenberg P, Bachorik PS and Bush TL. Non-high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. Arch Intern Med. 2001; 161: 1413–1419.
- 21 Chapman MJ, Ginsberg HN, Amarenco P, Andreotti F, Boren J, Catapano AL, *et al.* Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: evidence and guidance for management. Eur Heart J 2011; 32:1345–1361
- 22. Hegele RA, Ginsberg HN, Chapman JM, *et al.* The polygenic nature of hypertriglyceridaemia: implications for definition, diagnosis, and management. Lancet Diabetes Endocrin 2013;13: 70191–70198.
- Mora S, Rifai N, Buring JE and Ridker PM. Comparison of LDL cholesterol concentrations by Friedewald calculation and direct measurement in relation to cardiovascular events in 27 331 women. Clin Chem 2009; 55: 888– 894.