

Fasting versus Nonfasting Lipids for Cardiovascular Disease Risk Estimation among healthy adults in Ibadan, Nigeria: A cross-sectional study

Nonfasting lipids for CVD risk assessment

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Abstract

Objective: Risk assessment at the individual level requires incorporating lipid profile results into several cardiovascular disease (CVD) risk estimating equations. Traditionally, fasting is usually required before the lipid profile studies. Recent guidelines have recommended the acceptability of non-fasting lipids for this assessment based on reports from several countries. We aimed to compare the agreement between 10-year risk estimates obtained using fasting and non-fasting lipids from apparently healthy Nigerians.

Methods: This was a cross-sectional study of 111 participants. Serum blood lipids were measured after a 12-hour overnight fast and after a standard local Nigerian meal. Risk estimations with the pooled cohort equations (PCE) and the Framingham risk score (FRS) equation were done with fasting and non-fasting lipid results. Correlations were done with Pearson's coefficient and agreement of proportions with McNemar's test.

Results: Comparing fasting versus non-fasting values, total cholesterol was within 10% for 58 (52.3%), triglycerides were >30% for 65 (58.6%), and high-density lipoprotein cholesterol was <30% in 60 (70.0%) participants. An increase in Low-density lipoprotein cholesterol was seen in 93 (82.8%) participants. With the PCE, 3 (2.7%) persons, had borderline risk with both their fasting and non-fasting samples. With the FRS, 1 participant who was categorised as moderate risk with the fasting sample was classified as low risk with the non-fasting sample. There was no significant difference in risk categorisation by the equations, $p = 1.0$.

Conclusion: Risk categorisation by two (2) CVD risk estimating equations was not significantly affected by the fasting or non-fasting status of a healthy population.

Keywords: Lipids, Nonfasting, Cardiovascular, Risk Assessment

Plain English Summary

People are usually requested to fast for twelve hours before their blood samples may be collected for cholesterol tests. The results of such tests are then used to calculate their risk of developing diseases such as heart attack and stroke. This study shows that fasting may not be required before samples are collected for blood cholesterol tests in apparently healthy individuals as it has minimal effect on the calculations done to determine their risk of the aforementioned diseases.

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Introduction

Atherosclerotic cardiovascular diseases (ASCVD) like ischemic heart disease, stroke and peripheral artery disease are the largest contributors to the increasing cardiovascular disease (CVD) burden in sub-Saharan Africa (1). According to a country report, a Nigerian aged between 30 and 70 years of age has a 22% chance of dying from a major noncommunicable disease, with CVD accounting for over half of that risk (2). Primary prevention at the individual level has made important contributions to the reduced incidence rates that are responsible for the declining mortality trends from CVD over the past 2 decades in high-income countries (3). At the very heart of primary prevention is the identification of individuals at risk of atherosclerotic cardiovascular disease. The use of estimation equations to predict ASCVD risk is a major advance in the older practice of identifying and treating individual risk factors, such as raised blood pressure and raised blood cholesterol. Inputting individual risk factors into any of the several cardiovascular risk estimation equations is the approach recommended by the World Health Organization to stratify patients into mild, moderate and high risk of ASCVD (4). Management options aimed at risk factor reduction may then be implemented to prevent or delay the onset of CVD in moderate and high-risk patients.

Several hundred CVD risk prediction equations have been published (5). There are notable variations concerning populations used for their development and validation, the specific 10-year risk reported, the population in which they are applicable and the parameters required for the execution of the equation. The most widely used equations for assessing risk in the general population include as a required parameter one or more components of a standard plasma lipid profile (6). The American College of Cardiology/American Heart Association pooled cohort equations (PCEs) require plasma total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C), the European Society of Cardiology's Systematic COronary Risk Evaluation (SCORE) requires TC, HDL-C and low-density lipoprotein cholesterol (LDL-C) while the German Prospective Cardiovascular Münster risk score requires LDL-C, HDL-C, and triglycerides (TG) (7, 8, 9).

Historically and traditionally, lipid profile measurements have required a sample collected after a 12-hour fast. This requirement was deemed necessary by the proponents of the now widely used Friedewald formula, $LDL-C = TC - (HDL-C + TG/5)$, which allowed the accurate calculation of LDL-C from TC, HDL-C and TG, without the use of the laborious preparative

ultracentrifugation process (10). They observed that the triglyceride to cholesterol ratio in Very Low-Density Lipoprotein (VLDL) is relatively constant in normal subjects and nearly all patients with dyslipidaemia at about 5:1 as well as that when chylomicrons are not detectable in the blood, most of the triglyceride in plasma is from VLDL (10). The inference from these observations is that when chylomicrons are not detectable in the blood, as occurs in the fasting state, most of the triglyceride in plasma is from VLDL and the ratio and formula apply. Following on from this, when chylomicrons are detectable in the blood, in the post-prandial state, most of the triglyceride in plasma will not be from VLDL alone, the ratio will not be 5:1 and the formula should not apply. Thus, to use the Friedewald formula, a 12-hour fast has been required to ensure that all the dietary sourced chylomicrons have been cleared from circulation (10).

Large population studies over the last 2 decades have shown that serum lipid levels show only minor variation between the fasting and the postprandial state (11, 12). From the reports, postprandial serum triglyceride levels increased by about 20%, at most. This is within the scope of biological variation estimates for TG at 20 – 30% (13, 14). Differences between fasting and post-prandial values for TC, HDL-C and LDL-C have been reported to be less than 5%. The implications of these variations have also been explored on CVD risk estimation. Using the 2013 PCEs equation, Mora et al demonstrated nearly 94% concordance in the ASCVD risk scores obtained for the same individual using fasting and non-fasting samples (15).

There are no known local studies that have investigated the impact of the use of non-fasting lipids on CVD risk estimation in Nigeria. This study aims to examine the degree of agreement or differences between CVD risk estimates obtained using fasting versus non-fasting samples.

Materials and Methods

Study site and participants

This was a cross-sectional study of one hundred and eleven (111) apparently healthy staff of the University College Hospital, Ibadan aged between 30 and 65 years. Pregnant and lactating women were excluded from the study as well as those on any hypolipidemic medication or a special diet. The University College Hospital is a tertiary health facility located in the heart of Ibadan, Oyo state. It is a 1,000-bed hospital with over 5,000 staff.

Data/Sample Collection Protocol

A semi-structured questionnaire was used to collect information on demographic, social and

clinical history. Blood pressure (BP) was measured before the fasting sample was collected. BP measurement was taken after the participant was seated for about 10 minutes with hospital-provided Welch Allyn 767 Mobile Aneroid Sphygmomanometer (Baxter Incorporated, Alabama, United States of America). Participants were told to fast overnight for a minimum of 12 hours from 8 pm the previous day to 8 am the next day. A fasting blood sample was taken at 8 am and a standard meal (477 grams (9.5 tablespoon servings)) of jollof rice prepared according to Oguntano protocol (16) was served along with 5 pieces of fried plantain and 2 pieces of beef. Another sample was drawn after 4 hours for a postprandial lipid profile. Both samples were collected into plain bottles. Samples were allowed to clot and retract before centrifuging at 3000g for 15 minutes using Uniscope Laboratory centrifuge, model SM112 (Surgifriend Medicals, England) to obtain sera which was stored at -20°C in plain bottles. The centrifuge was validated using a strobe tachometer. Analysis was carried out within 1 week of collection.

Laboratory Analyses

Plasma total cholesterol, HDL-C and TG were assayed using the enzymatic method on the automated chemistry platform Landwind C100 plus (Shenzhen Landwind Biomedical Technology Co., Ltd, Shenzhen 518040, Guangdong, China). LDL-C was calculated using Martin-Hopkins equation (17).

CVD risk estimation

The 10-year risk for ASCVD was estimated using 2 equations. The first equation was the American College of Cardiology/American Heart

Association pooled cohort equations (PCEs) accessed at www.msmanuals.com/professional/multimedia/clinical-calculator/cardiovascular-risk-assessment-10-year-revised-pooled-cohort-equations-2018. The 10-year risk for ASCVD is categorized as low-risk (<5%), borderline risk (5% to <7.5%), intermediate risk (7.5% to <20%), and high risk (≥20%) (18). The other equation was the Framingham risk score equation was accessed at <https://www.mdcalc.com/calc/38/framingham-risk-score-hard-coronary-heart>. The 10-year risk for ASCVD is categorized as low-risk (<10%), moderate risk (10 to 20%), and high-risk (>20%) disease (19).

Data Analysis

Statistical analysis was done using IBM SPSS version 23 (IBM Corp., Armonk, NY, USA). Qualitative variables were presented as frequencies (percentages), while quantitative variables were presented as mean (SD). Pearson's correlation was used to check for association between quantitative variables. Agreement of risk categorization between estimates from fasting versus non-fasting results was assessed using McNemar's test for paired nominal data. Significance was set at $p < 0.05$.

Results

A total of one hundred and eleven (111) persons were recruited for the study with a mean age of 42 (7.3) years. There were 59 (53.2%) female participants with a mean age of 41.7 (6.9) years, which was not statistically different from that of the male participants at 42.3 (7.7) years, $p = 0.689$. Table 1 shows specific clinical characteristics of the study population.

Table 1: Clinical characteristics of the participants

Variables	Male	Female	Total
n (%)	52 (46.8%)	59 (53.2%)	111 (100%)
Age (yrs)	42.3 (7.7)	41.7 (6.9)	42 (7.3)
SBP (mmHg)	120.8 (12.4)	121.7 (13.6)	121.3 (13)
DBP (mmHg)	72.9 (10.1)	76.4 (9.9)	74.8 (10.1)
TC (mmol/L)	4.42 (0.49)	4.63 (0.48)	4.53 (0.49)
TG (mmol/L)	1.13 (0.25)	1.07 (0.21)	1.10 (0.23)
HDL-C (mmol/L)	1.25 (0.19)	1.27 (0.24)	1.26 (0.22)
LDL-C (mmol/L)	2.66 (0.49)	2.86 (0.49)	2.76 (0.50)
nTC (mmol/L)	4.46 (0.59)	4.57 (0.59)	4.52 (0.59)
nTG (mmol/L)	1.54 (0.38)	1.49 (0.33)	1.51 (0.35)
nHDL-C (mmol/L)	1.40 (0.25)	1.49 (0.27)	1.45 (0.26)
nLDL-C (mmol/L)	2.45 (0.58)	2.47 (0.57)	2.46 (0.57)

Values are mean (standard deviation); SBP – Systolic Blood Pressure, DBP – Diastolic Blood Pressure, TC- Total Cholesterol, TG – Triglycerides, HDL-C – High-density lipoprotein cholesterol, LDL-C – Low-density

lipoprotein cholesterol, nTC- non-fasting Total Cholesterol, nTG – non-fasting Triglycerides, nHDL-C – non-fasting High-density lipoprotein cholesterol, nLDL-C - non-fasting Low-density lipoprotein cholesterol.

Ten (9%) of the participants had a fasting TC \geq 200mg/dL, with 4 (3.6%) of these persons also having a postprandial TC \geq 200mg/dL. One of the participants had post-prandial TG of >200mg/dL but <400mg/dL. Thirty-one (52.5%) of the female participants had an HDL-C value \geq 50 mg/dL,

while 48 (92.3%) of the male participants had an HDL-C value \geq 40 mg/dL. The highest LDL-C values in the fasting and postprandial state were 168mg/dL and 167mg/dL respectively.

Table 2 shows a classification of the percentage change in the measured lipid profile parameters.

Table 2: Percentage change in serum lipid profile following the standard meal

	Percentage change							
	< -50%	- 20 to - 49.9%	-10 to -19.9%	0 to -9.9%	0 to 9.9%	10 to 19.9%	20 to 49.9%	> 50%
TC	-	2 (1.8%)	19 (17.1%)	37 (33.3%)	36 (32.4%)	9 (8.1%)	7 (6.3%)	1 (0.9%)
TG	-	-	1 (0.9%)	2 (1.8%)	6 (5.4%)	18 (16.2%)	54 (48.6%)	30 (27.0%)
HDL-C	1 (0.9%)	33 (29.7%)	32 (28.8%)	16 (14.4%)	15 (13.5%)	6 (5.4%)	5 (4.5%)	30 (2.7%)
LDL-C	-	1 (0.9%)	9 (8.1%)	10 (9.0%)	26 (23.4%)	20 (18.0%)	40 (36.0%)	5 (4.5%)

TC- Total Cholesterol, TG – Triglycerides, HDL-C – High-density lipoprotein cholesterol, LDL-C – Low-density lipoprotein cholesterol

The majority (73, 65.8%) of the values obtained for TC in the non-fasting state were within 10% of the fasting value. A decline in TC was observed in 58 (52.3%) participants. Almost all (102, 91.8%) of the participants had at least a 10% increase in the level of TG. Sixty-five (65, 58.6%) had greater than a 30% increase in the postprandial value over the fasting value. Most (87, 78.4%) of the participants had a reduction in the HDL-C cholesterol value. Sixty (70.0%) of these participants had less than a 30% decline in the value of HDL-C. Similar to TG, a majority (93, 82.8%) of the participants had an increase in their LDL-C value. Three (2.7%) of these had LDL-C values greater by at least 50%.

ASCVD risk estimates using the PCE from the fasting and non-fasting samples ranged from 0.1 – 7.4% and 0.1 – 6.5%, respectively. There was a strong correlation between the 2 estimates with a Pearson correlation score of 0.988, $p < 0.001$. The number (percentage) of persons with risk estimates less than 1% was 49 (44.1%) and 53 (47.7%) for the fasting and non-fasting samples, respectively. One hundred and eight (97.3%) participants of the study population had low risk, with estimates of less than 5% from both fasting and non-fasting samples. The remaining three

(2.7%) persons, had borderline risk. The values ranged from 5.0 – 7.4% and 5.1 – 6.5% for fasting and non-fasting, respectively.

ASCVD risk estimates using the Framingham equation from the fasting and non-fasting samples ranged from 0.0 – 11.5% and 0.0 – 7.7%, respectively. There was a strong correlation between the 2 estimates with a Pearson correlation score of 0.930, $p < 0.001$. The number (percentage) of persons with risk estimates less than 1% was 80 (72.1%) and 85 (76.6%) for the fasting and non-fasting samples, respectively. One hundred and ten (110, 99.1%) and 111 (100%) participants of the study population had low risk with estimates of less than 10% from results from the fasting and non-fasting samples, respectively. The single participant who had moderate risk with the fasting sample had a post-prandial decline of 30mg/dL (14.9%) in TC and an increase of 16mg/dL (39.0%) in HDL-C.

Table 3 shows the distribution of the participants into risk categories. There was no significant difference in the distribution, comparing fasting estimates with non-fasting estimates for either equation.

Table 3: Agreement between Nonfasting and Fasting Lipid Measurements for Classifying Participants into Categories of ASCVD Risk

Equation	10-y risk category by fasting lipid measurements	10-y risk category by nonfasting lipid measurements		p
		Low Risk	Borderline Risk	
Pooled Cohort Equation	Low Risk	108 (97.3%)	0 (0%)	1.00
	Borderline Risk	0 (0%)	3 (2.7%)	
Framingham Risk Score Equation	Low Risk	110 (99.1%)	0 (0%)	1.00
	Intermediate Risk	0 (0%)	1 (0.9%)	

Discussion

Requesting lipid profile studies in apparently healthy persons is primarily done for the assessment of risk for cardiovascular disease. This will involve the use of any of the severally available CVD risk estimating equations. Using two (2) very popular equations we have demonstrated that there is significant agreement in CVD risk estimates made from fasting and non-fasting lipid profile sample results for a largely low-risk population. For the participants who might have required some intervention based on their risk estimate from the PCE equation (10-year risk >5%), there was 100% agreement. Mora et al also demonstrated similarly high concordance between PCE estimates from fasting versus non-fasting lipids among those deemed low risk. Of the 1247 participants deemed low risk by fasting sample measurement in their population, 98.6% (1230) participants were also deemed low risk by non-fasting samples results (20). The agreement between the fasting and non-fasting sample estimates may be explained by the degree of variation in the specific lipid parameter, TC and HDL-C, used in the estimation equations. The mean of the maximal postprandial change in TC and HDL-C reported by several large prospective studies across several countries was 8mg/dL and 4 mg/dL respectively (11, 12, 21). Karmani et al conducted a systematic examination of the PCE to determine the risk factor levels of each variable in the equation required to exceed risk thresholds. For TC, using the entire range of values permitted by the risk assessment tool, increments of 10 mg/dl were made while holding all the other variables constant. The resulting changes in the CVD risk estimate for African Americans were modest (22).

The key attraction for the use of non-fasting samples for lipid profile studies is that it is convenient and resource-saving. The process of

blood sampling would be simplified for the patients and the laboratories. The inconvenience as well as the resources required for returning on a separate date are avoided by the patient while the laboratory can conserve resources spent early in the day for more phlebotomy services to cope with a surge of patients needing a fasting sample. The former reason may enhance patient compliance with testing. There is also the patient safety component for persons who have diabetes mellitus who no longer have to face the risk of hypoglycaemia from an overnight fast. Non-fasting sampling may also be more convenient for children and the elderly (23, 24).

Beyond the convenience of the non-fasting sample, is that it may indicate CVD risk not obviously estimated from the fasting sample. This was the suggestion from a United States study of 26,509 women (fasting and non-fasting) enrolled in the Women's Health Study and followed up for about 14 years. Non-fasting triglyceride levels were independently associated with incident cardiovascular events while fasting triglyceride levels showed little independent relationship (25). This was similarly demonstrated in another prospective study of about 14,000 men and women in Denmark followed up for about 20 years. Increased levels of non-fasting TG were linked with increased risk of ASCVD and mortality in both genders (26). This association between non-fasting triglycerides and CVD risk is important to identify as most individuals will be in the postprandial phase for between 16 – 18 hours a day. Information in the postprandial phase may therefore be more reflective of the environment to which the cells and tissues are constantly exposed (27).

Given the above, it is unsurprising that the number of national societies and guidelines endorsing the use of non-fasting lipids for CVD risk assessment is increasing (28, 29). This study supports the adoption of this position as part of

the primary preventive efforts to reduce the burden of ASCVD among Nigerians.

Study limitations

Our population of apparently healthy persons was selected from staff of a tertiary hospital who may be more aware of their health status than the general population.

Conclusion

We suggest that the inclusion of non-fasting lipids as an option for ASCVD risk assessment for apparently healthy Nigerians should be strongly considered. This will have a significant public health impact as it may improve compliance with and uptake of requests for CVD risk assessment.

List of Abbreviations

ASCVD:	Atherosclerotic cardiovascular diseases
BP:	Blood pressure
CVD:	Cardiovascular diseases
DBP:	Diastolic blood pressure
FRS:	Framingham risk score
HDL-C:	High density lipoprotein cholesterol
LDL:	Low density lipoprotein
LDL-C:	Low density lipoprotein cholesterol
nHDL-C:	nonfasting high-density lipoprotein cholesterol
nLDL-C:	nonfasting low-density lipoprotein cholesterol
nTC:	non-fasting total cholesterol
nTG:	non-fasting triglycerides
PCE:	Pooled cohort equations
SBP:	Systolic blood pressure
SCORE:	Systematic COronary Risk Evaluation
SD:	Standard deviation
TC:	Total cholesterol
TG:	Triglycerides
UI:	University of Ibadan
UCH:	University College Hospital
VLDL:	Very low-density lipoprotein cholesterol

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the University of Ibadan/University College Hospital (UI/UCH) ethical review board with UI/EC/21/0035. The study adhered to strict guidelines on responsible conduct of research with human subjects as spelt out by the Helsinki declaration.

Consent for publication

All the authors give consent for the publication of the work under the Creative Commons Attribution-Non-Commercial 4.0 license.

Availability of data and materials

The data sets used and analyzed during the current study are available from the corresponding author on request.

Competing interests.

The authors declare no competing interests.

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Authors' contributions

Conceptualization:	KMA, EBJ, BOT, OOA
Data acquisition:	EBJ, OOA
Data analysis:	KMA, OOA
Article writing:	KMA, OOA
Manuscript review:	KMA, EBJ, BOT, OOA
Supervision:	EBJ
Final approval:	KMA, EBJ, BOT, OOA

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References

1. Gouda HN, Charlson F, Sorsdahl K, Ahmadzada S, Ferrari AJ, Erskine H, Leung J, Santamauro D, Lund C, Aminde LN, Mayosi BM. Burden of non-communicable diseases in sub-Saharan Africa, 1990–2017: results from the Global Burden of Disease Study 2017. *The Lancet Global Health*. 2019 Oct 1;7(10):e1375-87. [https://doi.org/10.1016/S2214-109X\(19\)30374-2](https://doi.org/10.1016/S2214-109X(19)30374-2)
2. Sani MU, Ogah OS, Scholtz W, Nel G, Fourie JM, Scarlatescu O. PASCAR and WHF Cardiovascular Diseases Scorecard project. *Cardiovascular Journal of Africa*. 2020 Jul 1;31(4):s19-26. <https://doi.org/10.5830/CVJA-2020-043>
3. Unal B, Critchley JA, Capewell S. Explaining the Decline in Coronary Heart Disease Mortality in England and Wales Between 1981 and 2000. *Circulation* 2004 109(9):1101-7. <https://doi.org/10.1161/01.CIR.0000118498.35499.B2>
4. World Health Organization. Prevention of cardiovascular disease: guidelines for assessment and management of total cardiovascular risk. World Health Organization; 2007.
5. Damen JA, Hooft L, Schuit E, Debray TP, Collins GS, Tzoulaki I, Lassale CM, Siontis

- GC, Chiocchia V, Roberts C, Schlüssel MM. Prediction models for cardiovascular disease risk in the general population: systematic review. *bmj*. 2016 May 16;353. <https://doi.org/10.1136/bmj.i2416>
6. Sofogianni A, Stalikas N, Antza C, Tziomalos K. Cardiovascular Risk Prediction Models and Scores in the Era of Personalized Medicine. *J Pers Med*. 2022; 12(1180):1 – 11. <https://doi.org/10.3390/jpm12071180>
 7. Yadlowsky S, Hayward RA, Sussman JB, McClelland RL, Min Y-I, Basu S. Clinical Implications of Revised Pooled Cohort Equations for Estimating Atherosclerotic Cardiovascular Disease Risk. *Ann Intern Med*. 2018; 169(1):20-9. <https://doi.org/10.7326/M17-3011>
 8. Hageman SH, Petitjaen C, Pennells L, Kaptoge S, Pajouheshnia R, Tillmann T, Blaha MJ, McClelland RL, Matsushita K, Nambi V, Klungel OH. Improving 10-year cardiovascular risk prediction in apparently healthy people: flexible addition of risk modifiers on top of SCORE2. *European Journal of Preventive Cardiology*. 2023 Oct;30(15):1705-14. <https://doi.org/10.1093/eurjpc/zwad187>
 9. Assmann G, Cullen P, Schulte H. Simple Scoring Scheme for Calculating the Risk of Acute Coronary Events Based on the 10-Year Follow-Up of the Prospective Cardiovascular Münster (PROCAM) Study. *Circulation*. 2002; 105(3):310-5. <https://doi.org/10.1161/hc0302.102575>
 10. Friedewald WT, Levy RI, 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. <https://doi.org/10.1093/clinchem/18.6.499>
 11. Sidhu D, Naugler C. Fasting Time and Lipid Levels in a Community-Based Population. *Arch Intern Med*. 2012; 172(22):1707-10. <https://doi.org/10.1001/archinternmed.2012.3708>
 12. Mora S, Rifai N, Buring JE, Ridker PM. Fasting Compared with Nonfasting Lipids and Apolipoproteins for Predicting Incident Cardiovascular Events. *Circulation*. 2008; 118(10):993-1001. <https://doi.org/10.1161/CIRCULATIONAHA.108.777334>
 13. Costongs GM, Janson PC, Bas BM, Hermans J, van Wersch JW, Brombacher PJ. Short-term and long-term intra-individual variations and critical differences of clinical chemical laboratory parameters. *J Clin Chem Clin Biochem*. 1985; 23:7–16. <https://doi.org/10.1515/cclm.1985.23.1.7>
 14. Friedlander Y, Kark JD, Stein Y. Variability of plasma lipids and lipoproteins: the Jerusalem Lipid Research Clinic Study. *Clin Chem*. 1985; 31(7):1121-6. <https://doi.org/10.1093/clinchem/31.7.1121>
 15. Mora S, Chang CL, Moorthy MV, Sever PS. Association of Nonfasting vs Fasting Lipid Levels with Risk of Major Coronary Events in the Anglo-Scandinavian Cardiac Outcomes Trial–Lipid Lowering Arm. *JAMA Intern Med*. 2019; 179(7):898-905. <https://doi.org/10.1001/jamainternmed.2019.0392>
 16. Oguntona CRB, Odunmbaku JA, Ottun BO. Proximate composition of ten standardized Nigerian dishes. *Nutr Food Sci*. 1999; 99(6):295-302. <https://doi.org/10.1108/00346659910290466>
 17. Martin SS, Blaha MJ, Elshazly MB, Toth PP, Kwiterovich PO, Blumenthal RS, Jones SR. Comparison of a novel method vs the Friedewald equation for estimating low-density lipoprotein cholesterol levels from the standard lipid profile. *Jama*. 2013 Nov 20;310(19):2061-8. <https://doi.org/10.1001/jama.2013.280532>
 18. Grundy SM, Stone NJ, Bailey AL, Beam C, Birtcher KK, Blumenthal RS, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2019; 139(25):e1082-143. <https://doi.org/10.1161/CIR.0000000000000698>
 19. Wilson PWF, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of Coronary Heart Disease Using Risk Factor Categories. *Circulation*. 1998; 97(18):1837-47. <https://doi.org/10.1161/01.CIR.97.18.1837>
 20. Mora S, Buring JE, Ridker PM. Discordance of low-density lipoprotein (LDL) cholesterol with alternative LDL-related measures and future coronary events. *Circulation* 2014 129(5):553-61. <https://doi.org/10.1161/CIRCULATIONAHA.113.005873>
 21. Langsted A, Freiberg JJ, Nordestgaard BG. Fasting and Nonfasting Lipid Levels. *Circulation*. 2008; 118(20):2047-56. <https://doi.org/10.1161/CIRCULATIONAHA.108.804146>
 22. Karmali KN, Goff DC, Ning H, Lloyd-Jones DM. A Systematic Examination of the 2013 ACC/AHA Pooled Cohort Risk Assessment Tool for Atherosclerotic Cardiovascular Disease. *J*

- Am Coll Cardiol. 2014; 64(10):959-68. <https://doi.org/10.1016/j.jacc.2014.06.1186>
23. Langsted A, Nordestgaard BG. Nonfasting versus fasting lipid profile for cardiovascular risk prediction. *Pathology*. 2019; 51(2):131-41. <https://doi.org/10.1016/j.pathol.2018.09.062>
24. Darras P, Mattman A, Francis GA. Nonfasting lipid testing: the new standard for cardiovascular risk assessment. *Can Med Assoc J*. 2018; 190(45): E1317-8. <https://doi.org/10.1503/cmaj.180804>
25. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting Compared with Nonfasting Triglycerides and Risk of Cardiovascular Events in Women. *JAMA*. 2007; 298(3):309-16. <https://doi.org/10.1001/jama.298.3.309>
26. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting Triglycerides and Risk of Myocardial Infarction, Ischemic Heart Disease, and Death in Men and Women. *JAMA*. 2007; 298(3):299-308. <https://doi.org/10.1001/jama.298.3.299>
27. Higgins V, Adeli K. Postprandial Dyslipidemia: Pathophysiology and Cardiovascular Disease Risk Assessment. *EJIFCC*. 2017; 28(3):168-84
28. Jellinger PS, Handelsman Y, Rosenblit PD, Bloomgarden ZT, Fonseca VA, Garber AJ, Grunberger G, Guerin CK, Bell DS, Mechanick JI, Pessah-Pollack R. American Association of Clinical Endocrinologists and American College of Endocrinology guidelines for management of dyslipidemia and prevention of cardiovascular disease. *Endocrine Practice*. 2017 Apr 1;23:1-87. <https://doi.org/10.4158/EP171764.APPGL>
29. Nordestgaard BG, Langsted A, Mora S, Kolovou G, Baum H, Bruckert E, Watts GF, Sypniewska G, Wiklund O, Borén J, Chapman MJ. Fasting is not routinely required for determination of a lipid profile: clinical and laboratory implications including flagging at desirable concentration cut-points—a joint consensus statement from the European Atherosclerosis Society and European Federation of Clinical Chemistry and Laboratory Medicine. *European Heart Journal*. 2016 Jul 1;37(25):1944-58. <https://doi.org/10.1093/eurheartj/ehw152>