OPEN ACCESS

Saudi Journal of Biomedical Research

Abbreviated Key Title: Saudi J Biomed Res
ISSN 2518-3214 (Print) | ISSN 2518-3222 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com

Original Research Article

Suitability of Routine Sample Containers, Time of Sample Collection, and Diet Types on Lipid Profile Estimation

Eni-Yimini Solomon Agoro[1]*, Harlims Okiemute Ofomola[2], Jane Ugochi Chinedu-Madu[3]

[1]Department of Biochemistry, Faculty of Science, Federal University Otuoke, Bayelsa State, Nigeria
[2]Department of Clinical Biochemistry, Automated Laboratory, NHS University Hospitals of Liverpool Group. Aintree Site University Kingdom
[3]Department of Haematology and Blood Transfusion, Federal University Otuoke, Bayelsa State, Nigeria

DOI: https://doi.org/10.36348/sjbr.2025.v10i09.002 | **Received:** 04.07.2025 | **Accepted:** 02.09.2025 | **Published:** 06.09.2025

***Corresponding author:** Eni-Yimini Solomon Agoro
Department of Biochemistry, Faculty of Science, Federal University Otuoke, Bayelsa State, Nigeria

Abstract

Background**:* Sampling time and containers used for sample collection are crucial preanalytical processes, dictating the accuracy and precision of lipid profile results. We compared the effect of sampling time, routine containers, and diet types on lipid profiles. ***Methods: This cross-sectional study was conducted among final-year students of the Department of Biochemistry at the Federal University Otuoke, Bayelsa State, in 2024. A total of 150 participants were recruited for this study. Fasting, starvation, and random blood samples were collected from each patient into plain containers, lithium heparin, K_2EDTA, and fluoride oxalate containers simultaneously on daily staged structures. Samples in anticoagulant tubes were immediately centrifuged, and plasma was obtained while samples in plain containers were made to clot before centrifugation to obtain serum. The plasma and serum obtained were used to estimate the concentrations of total cholesterol (TC), triglyceride (TG), low-density lipoprotein (LDL-c), and high-density lipoprotein (HDL-c) using an automated chemistry analyzer. ***Results:*** The study shows a significant mean increase ($P<0.05$) in TC and LDL concentrations in the fasting and starvation groups compared to the control and random samples. Similarly, the anticoagulants used revealed a significant mean increase ($P<0.05$) in the concentrations of TC and LDL in lithium heparin and fluoride oxalate compared to EDTA and plain containers. Furthermore, the findings revealed a significant increase in triacylglycerol concentration in the lipid diet group when compared to other groups, whereas other parameters were stable. ***Conclusions**:* Random sampling in lipid profile estimation could be more suitable as a choice condition, comparable to fasting or starvation sampling. In a similar vein, the suitability index of sample containers placed K_2EDTA and plain containers as a better choice for lipid profile estimation, comparable to lithium heparin and fluoride oxalate.

Keywords: Anticoagulants, lithium heparin, K_2EDTA, fluoride oxalate, plain container, lipid profile.

INTRODUCTION

Lipids are a broad group of organic compounds that include fats, waxes, sterols, fat-soluble vitamins, monoglycerides, triglycerides, phospholipids, and others. The functions of lipids include storing energy, signalling, and acting as structural components of cell membranes [1]. Lipids such as cholesterol and triacylglycerol are crucial in the efficiency of most systemic mechanisms of the body [2].

Lipid parameters such as total cholesterol, triacylglycerol, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) form the strings of investigation called lipid profile or panel. The profile is an important biomarker for assessing cardiovascular health. Accurate analysis and monitoring of lipid parameters are critical in evaluating and treating various health conditions, especially those related to cardiovascular disease [3].

Laboratory analysis of lipid profile involves pre-analytical, analytical, and post-analytical stages that culminate in the generation of accurate results used for the disease diagnosis and management of patients. However, the pre-analytic stage involving the choice of blood container (anticoagulant or plain) is crucial in generating accurate and precise laboratory results. Containers such as lithium heparin, plain, ethylenediaminetetraacetic acid (EDTA), and fluoride oxalate are the most routinely used in the laboratory. The preference for these containers for collecting blood

samples for lipid profile determination varies with diverse perspectives [4]. Whereas some prefer EDTA, others are supportive of plain containers.

In a similar vein, the literature is not decisive on the time of sample collection. However, fasting samples are believed to be the most suitable for lipid profile estimation [5]. This stance is strictly adhered to in Nigeria, as almost all the health facilities insist on the use of fasting samples for lipid profile estimation. On the other hand, the nations and researchers are advancing the preference for random or postprandial samples [6-8]. They based their argument on the scientific fact that the postprandial state predominates during most of the day, and the patient is more exposed to the lipid levels in this condition when compared with the fasting state [9].

These gaps discussed above formed the basis of this study, as the study detailed the accuracy and precision resulting from the various routine containers used in sample collection for lipid profile estimation.

MATERIALS AND METHODS

Study location

The study was conducted at Federal University Otuoke (FUO), located in Otuoke, Bayelsa State of Nigeria. This institution is a leading university, known for its advanced research facilities and a strong focus on Biochemistry. Bayelsa State is the cradle of oil exploration and exploitation in Nigeria, hosting Otuobagi, the first place where oil was struck in commercial quantity in Nigeria [10-14]

Research Design

Quantitative experimental design was the chosen research design for the study. The design establishes the cause-and-effect relationship among the various groups (blood) that make up the study [15].

Population Size

A sample size was calculated using a formula proposed for studies where the population is less than 10,000 [16]. The formula is stated and the components defined below:

$S = N/ [1 + N(d^2)]$

Where:

S =sample size
N=population of study =135
d= level of error confidence level at 95%=0.05

$S=135/[1 + 135(d^2]$
$S=135/[1+135 (0.0025]$
$S = 135[1 + 0.3375]$
$S=100$

The blood samples were collected in 10 mL syringes into plain, EDTA, lithium heparin, and fluoride oxalate containers.

Selection Criteria

The Blood was obtained from students of the Department of Biochemistry, Federal University of Otuoke, for the study. The subjects used were healthy and active, as confirmed and approved by the University Physician. Subjects exhibiting signs or symptoms of ill health or on medications were excluded from the study.

Ethical Approval

The ethical approval was granted by the Directorate of Research and Quality Assurance (DR&QA) of the Federal University Otuoke, following the laid-down requirements and processes with a registry number of DR&QA/101/2024.

Sample Collection

The blood samples were collected randomly from a hundred (100) students of the Department of Biochemistry into the various sample containers. The containers used for the study include plain, K_2EDTA, lithium heparin, and fluoride oxalate. This was followed by centrifugation at 1500 rpm and the extraction of the serum/plasma into sterile containers for the lipid profile estimation. Biochemical parameters estimated under the profile included total cholesterol (TC), triacylglycerol (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL).

Laboratory Procedures

The lipid profile parameters (TC, TG, HDL, LDL) were estimated using Aggape (UK) test kits, which involve enzymatic and the end-point is measured with semi semi-chemistry analyser (Contec-China).

Statistical Analysis

The results were expressed as mean ± standard deviation. One-way Anova (Post hoc) was calculated at a 95% confidence interval, and statistically significant results pegged at ≤ 0.05 on an SPPS version (18-21).

RESULTS

Table 1: Age Range Demographics Presentation

Age	Frequency	Percentage (100%)
18-25	68	68%
26-35	32	32%
Total	**100**	**100%**

Table 2: Demographics presentation gender status

Gender	Frequency	Percentage
Male	47	47%
female	53	53%
Total	**100**	**100%**

Table 3: Collection time effect on lipid profile concentrations in plain container sampled blood

Parameters (mmol/L)	Control (n=100)	Fasting (n=100)	Starvation (n=100)	Random (n=100)	F-value	P-value
TC	2.78±0.03	3.78±0.90[a]	3.72±0.56[a]	3.49±0.82[a]	4.54	0.01
TG	0.80±0.02	0.91± 0.33	0.93±0.24	1.22±0.74	1.78	0.17
HDL	1.52± 0.03	1.62±0.33	1.43±0.66	1.73±0.69	4.99	0.68
LDL	0.9± 0.05	1.75±0.03[a]	1.87± 0.23[a]	0.78±0.09[b,c]	5.90	0.04

Table 4: Collection time effect on lipid profile concentrations in lithium heparin container sampled blood

Parameters (mmol/L)	Control (n=100)	Fasting (n=100)	Starvation (n=100)	Random (n=100)	F-value	P-value
TC	3.40 ± 0.01	8.33±1.40[a]	5.51 ± 1.66 [a]	9.30±1.17[a]	14.18	0.00
TG	0.70 ± 0.01	1.31±0.82[a]	1.07 ± 0.31 [a]	1.59 ±1.30[a]	3.37	0.03
HDL	1.40 ±0.91	1.77 ± 0.85	1.67 ± 0.54	1.97 ± 0.79	2.24	0.06
LDL	1.68 ±0.90	5.96 ± 0.66 [a]	3.35 ±0.98[a]	6.60±1.79[a,]	10.42	0.02

Table 5: Collection time effect on lipid profile concentrations in EDTA container sampled blood

Parameters mmol/L	Control n=100	Fasting n=100	Starvation n=100	Random n=100	F-value	P-value
Cholesterol (mmol/L)	3.84 ±0.01	4.48±1.12	4.63±1.06	3.91±0.87	2.03	0.13
Triglyceride (mmol/L)	0.89±0.01	0.74±0.35	0.77±0.35	0.92±0.63	3.70	0.21
HDL (mmol/L)	2.10±0.08	2.26±1.01	1.96±1.03	2.01±2.01	3.00	0.24
LDL (mmol/L)	1.34±0.11	1.88±0.19	2.32±0.96	1.48±0.66	3.31	0.24

Table 6: Collection time effect on lipid profile concentrations in fluoride oxalate container sampled blood

Parameters mmol/L	Control n=100	Fasting n=100	Starvation n=100	Random n=100	F-value	P-value
Cholesterol	4.70 ± 0.18	4.97 ± 1.67	5.88 ± 1.45	5.91 ±1.88	0.99	0.41
Triglyceride	0.88 ± 0.01	1.43 ± 0.92	0.98 ± 0.53	0.98 ± 0.47	0.62	0.61
HDL	1.54 ± 0.02	1.32 ± 0.67	1.23 ± 0.88	1.33 ± 0.78	0.67	0.33
LDL	2.76 ± 0.92	3.00 ±0.95	4.23 ±1.78	4.13 ±0.85	1.40	0.79

Table 7: Effect of routine containers on mean concentrations of lipid profile concentrations of the control group

Parameters mmol/L	Plain	Lithium	EDTA	Fluoride Oxalate	F-value	P-value
TC	2.78±0.00	3.40±0.01	3.84±0.01	4.70±0.18	0.89	0.46
TG	0.80±0.02	0.70±0.01	0.89±0.01	0.88±0.01	0.33	0.34
HDL	1.52±0.03	1.40±0.91	2.10±0.08	1.54±0.02	0.57	0.55
LDL	0.9± 0.05	1.68 ±0.90	1.34±0.11	2.76±0.92[a]	1.30	0.04

Table 8: Effect of routine containers on mean concentrations of lipid profile concentrations of the fasting group

Parameters mmol/L	Plain	Lithium	EDTA	Fluoride Oxalate	F-value	P-value
TC	3.78±0.90	8.33±1.40[a]	4.48±1.12[b]	4.97±1.67[b]	0.35	0.03
TG	0.91±0.33	1.31±0.82	0.74±0.35	1.43±0.92	2.73	0.64
HDL	1.62±0.33	1.77± 0.85	2.26±1.01	1.32±0.67	1.66	0.54
LDL	1.75±0.03	5.96 ±0.66 [a]	1.88±0.19[b]	3.00±0.95[a,b,c]	1.30	0.03

Table 9: Effect of routine containers on mean concentrations of lipid profile concentrations of the starvation group

Parameters mmol/L	Plain	Lithium	EDTA	Fluoride Oxalate	F-value	P-value
TC	3.72±0.56	5.51 ± 1.66[a]	4.63±1.06	5.88 ± 1.45a	0.11	0.05
TG	0.93±0.24	1.07 ± 0.31	0.77±0.35	0.98 ± 0.53	5.03	0.11
HDL	1.43±0.66	1.67 ± 0.54	1.96±1.03	1.23 ± 0.88	2.34	0.76
LDL	1.87± 0.23	3.35 ±0.98[a]	2.32±0.96	4.23 ±1.7[a,b]	2.22	0.04

Table 10: Effect of routine containers on mean concentrations of lipid profile concentrations of the random group

Parameters mmol/L	Plain	Lithium	EDTA	Fluoride Oxalate	F-value	P-value
TC	3.49±0.82	9.30±1.17[a]	3.91±0.87[b]	5.91±1.88[a,b,c]	0.98	0.02
TG	1.22± 0.74	1.59 ± 1.30	0.92±0.63[b]	0.98 ± 0.47[b]	9.01	0.04
HDL	1.73±0.69	1.97 ± 0.79	2.01±0.08	1.33 ± 0.78	5.98	0.56
LDL	0.78±0.09	6.60 ± 1.79[a]	1.48±0.66[b]	4.13 ±0.85[a,b]	4.56	0.03

Table 11: Multiple comparison of the diet types on lipid profile parameters

Parameters mmol/L	Control	Carbohydrates	Protein	Lipid	F- Value	P -Value
Cholesterol	3.84 ±0.01	4.48 ± 1.12	4.63 ± 1.01	3.91 ± 0.87	2.03	0.13
Triglyceride	0.35 ±0.01	0.74 ± 0.35[a]	0.77 ± 0.35[a]	0.92 ± 0.63[a,b,c]	3.70	0.02
High Density Lipoprotein	1.17 ±0.23	1.10 ± 0.66	1.77 ± 0.57	1.18 ± 0.31	3.66	0.34
Low Density Lipoprotein	2.54 ±0.93	1.87 ± 0.67	2.53 ± 0.69	2.10 ± 0.83	4.44	0.63

Table 1 shows the age range, frequency, and the calculated percentage of demographics with age 18-25 showing a higher frequency and higher percentage, unlike age 26-35, which shows a lower frequency and percentage. Table 2 shows the gender status with varying frequency and percentage. The female gender has a higher frequency and percentage than the male. Table 3 shows a significant decrease ($P<0.05$) in the TC concentration of the control sample when compared to the other groups in plain containers. However, LDL concentrations increased significantly ($P < 0.05$) in the fasting and starvation groups compared to the control and random samples. Table 4 shows a significant decrease ($P<0.05$) in the concentrations of TC, TG, and LDL in the control samples when compared to the other groups using lithium heparin containers. However, the concentrations of the lipid profile parameters tend to be highly elevated. Table 5 results show a statistically nonsignificant difference ($P>0.05$) of all the studied parameters when compared within the various groups in EDTA containers. Table 6 shows no significant difference ($P >0.05$) of the studied parameters when compared among the various groups in fluoride oxalate containers. Table 7 shows a significant increase in the concentration of LDL in controls in fluoride containers when compared to other routine containers used in sample collection. Table 8 shows an increase in TC concentration of the fasting sample in lithium heparin containers when compared to other studied containers. In a similar vein, the concentration of LDL in lithium heparin and fluoride oxalate increased significantly ($P>0.05$) when compared to that of plain and EDTA containers. Table 9 shows a significant increase ($P <0.05$) in the concentration of TC of the starvation group in lithium heparin when compared to plain; however, it was not significant ($P>0.05$) compared to EDTA and fluoride oxalate. Whereas, LDL increases in lithium heparin and fluoride oxalate in the starvation group when compared to plain and EDTA. Table 10 shows a significant increase ($P<0.05$) in the concentrations of TC and LDL in lithium heparin and fluoride oxalate when compared to plain and EDTA containers in randoms. Table 11 shows a significant increase ($p<0.05$) in the concentration of triacylglycerol in the lipid diet group, when compared to other groups.

DISCUSSION

The demographic analysis reveals a predominance of individuals aged 18-25, comprising 89% of the sample, compared to just 11% in the 26-35 age group (Table 1). Regarding gender distribution, females represented 58% of the sample, while males constituted 42% (Table 2). The demographic presentations are balanced and suitable for empirical decisions and other scientific consumption.

The study revealed a significant decrease in the concentrations of the control lipid profile compared to other timing groups using some of the various routine sample containers (Table 3-6). This could be attributed to the known predetermined lower concentrations of the control sera. In a similar vein, LDL concentrations increased significantly in the fasting and starvation groups compared to the control and random samples using plain containers (Table 3). However, the concentrations of the lipid profile parameters tend to be highly elevated in lithium heparin, irrespective of the timing categorization (Table 4).

Furthermore, LDL concentrations increased significantly in the fasting and starvation groups compared to the control and random samples. This points to the significant effect of timing on lipid profile estimation using plain containers. The decrease observed in the controls compared to the other groups is a result of the predetermined value of the control sera, which was lower in concentration. However, the increased LDL concentration in the fasting and starvation states compared to the random state could be a result of the initiation of lipolysis. Lipolysis usually takes place upon the cessation of the intake of a meal, which is attributable to fasting and starvation states. This implies that random samples are a true reflection of the LDL concentration, as fasting or starvation could lead to pseudo-elevation of LDL. This finding is in line with that of Sävendahl and Underwood [17] and Steen *et al.* [18]. A study conducted by Sathiyakumar *et al.* [19] suggested that random sampling is preferred for LDL estimation using the fixed Friedewald equation [20].

The preference of random or nonfasting lipid profiles to the traditional 8 hours or 12 hours fasting as posited in this study is in line with the endorsements, guidelines and statements in Denmark [21], the United Kingdom [22], Europe [6-7], Canada [23-24], Brazil [9] and the United States [25-27].

On the contrary, the findings of this study is contrary to that of Vittal and Abhijith, [8] that showed no statistically significant difference between total cholesterol, HDL cholesterol and LDL cholesterol in the fasting and postprandial state indicating that it should not make any difference whether the blood sample is collected in the fasting state or non-fasting state for these three parameters. A statistically significant difference was noted in fasting and postprandial levels of triglycerides and VLDL cholesterol, indicating that fasting and non-fasting samples cannot be used interchangeably for these parameters. Eberly *et al.* [28] in their study published in 2003, stated that fasting and non-fasting triglycerides are similarly predictive of non-fatal and fatal coronary artery disease (CAD). In contrast, Kamrul-Hasan [29] advanced that a random lipid profile correlates significantly with a fasting lipid profile, with little difference.

Moreover, the study also investigated the impacts of the studied routine sample containers on the accuracy and precision of lipid profile parameters (Tables 7-10) using control sera and other timed samples as case studies. The findings revealed a significant increase in the concentration of LDL in the controls of the fluoride oxalate containers when compared to other routine containers used in sample collection (Table 7). Oxalate prevents blood clotting by binding to calcium ions. In contrast, fluoride interferes with the activities of the specific proteolytic enzymes crucial in the coagulation pathway and enolase, which is far downstream in the glycolytic pathway [30]. Furthermore, fluoride has been implicated in lipid peroxidation [31]. The roles of some of these anticoagulants on the cell membrane, dominated by lipids, could contribute to the biochemical distortions observed. These synergic roles could be the basis of the significant increase in LDL concentration in the fluoride oxalate tubes. This finding contradicted that of Le and Le [32], which posited a decrease in total cholesterol, triglycerides, HDL-C, and LDL-C concentrations in the sodium fluoride-potassium oxalate tube when compared to the plan tube, though with a different anticoagulant composition.

Furthermore, fasting samples revealed an increase in TC concentration in lithium heparin containers compared to other studied containers. In a similar vein, the concentration of LDL in lithium heparin and fluoride oxalate increased significantly when compared to that of plain and EDTA containers (Table 8). The results of this study contradicted that of Anto et al. [33], though ours was compared against plain containers, which indicated a significantly reduced mean level of TC, LDL-c, and TG when heparin, K_2EDTA, and sodium citrate anticoagulant tubes were used compared to the gel separator tube. In a similar vein, the study concurred with the above author on HDL concentration measurement. The mechanism linking heparin-induced increases in TC and LDL levels in vitro is not well understood. However, heparin has been found to bind to LDL under the physiological conditions, probably by Van der Waals interactions and hydrogen bonding. Furthermore, the presence of heparin inhibits LDL binding to the intact LDL receptor, which might have consequences on the cholesterol metabolism in vivo [34].

In addition, there was a significant increase in the concentration of TC in the starvation group treated with lithium heparin compared to plain, whereas LDL increased in the starvation group treated with lithium heparin and fluoride oxalate compared to plain and EDTA (Table 9). The findings further posited an increase in the concentrations of TC and LDL in lithium heparin and fluoride oxalate when compared to plain and EDTA containers in randoms (Table 10). The incremental tendencies of TC and LDL in lithium heparin in fasting, starvation, and random have further validated the stance above.

The above findings make it crystal clear that anticoagulants such as lithium heparin, and fluoride oxalate have clinical adverse effects on the accuracy and precision of some lipid profile parameters. However, on the other hand, EDTA and plain containers stand as the most suitable for lipid profile sample collection with insignificant anticoagulant and biochemical alterations and effects. Based on the suitability grading, it could be inferred that EDTA is the most suitable, followed by plain container, and then lithium heparin and fluoride oxalate (EDTA>Plain Container>Fluoride Oxalate>Lithium Heparin).

Furthermore, the findings on the effect of diet types on lipid profile revealed a significant increase in triacylglycerol concentration in the lipid diet group when compared to other groups, whereas other parameters were stable (Table 11). The plausible reason could be attributed to the breakdown of dietary lipids, thereby increasing the endogenous concentration of triacylglycerol, as agreed by other authors [35-36].

CONCLUSION

We have shown in this study that random sampling is most suitable for the estimation of lipid profile as to the traditional fasting sample. On blood containers, our findings have shown that the K_2EDTA tube is the most suitable for the estimation of lipid profile, followed by plain container. Furthermore, the findings on the effect of diet types on lipid profile revealed a significant increase in triacylglycerol concentration in the lipid diet group when compared to other groups, whereas other parameters were stable. Further studies are needed to reaffirm these findings using a larger cohort of a healthy population.

Declarations

Ethics Approval and Consent to Participate

The ethical approval was granted by the Directorate of Research and Quality Assurance (DR&QA) of the Federal University Otuoke following the laid-down requirements and processes. The participants were duly informed of the procedures of the study, and individual consents were obtained before the commencement.

Consent for Publication

The authors agreed without any iota of coercion to using this platform for the publication of their article.

Availability of Data and Material

The data that support the findings of this study are available from the corresponding author, ESA, upon reasonable request.

Competing Interest: The authors declare no conflicts of interest.

Funding: Nil

Authors' Contributions

Eni-Yimini S. Agoro contributed to the conception of the research idea, data collection, interpretation, paper drafting, and revision. Harlims O. Ofomola contributed to the paper drafting and revision. Jane U. Chinedu-Madu contributed to data collection and sample analysis. All authors approved the final manuscript before publication and agree to be accountable for all aspects of the work.

Acknowledgements

Our sincere gratitude to our 2023/2024 final students of the Department of Biochemistry, Federal University Otuoke, and the staff of the Eni-yimini Laboratories (eL) LTD, Yenezue Gene-Epie, Yenagoa, Bayelsa State

REFERENCES

1. Asokapandian S, Sreelakshmi S, Rajamanickam G. (2021). Lipids and oils: an overview. *Food biopolymers:* In book: Food biopolymers: Structural, functional and nutraceutical properties. 2021; 389-411.
2. Hu J, Zhang Z, Shen WJ, Azhar S. Cellular cholesterol delivery, intracellular processing and utilization for biosynthesis of steroid hormones. Nutr Metab (Lond). 2010, 1;7:47. doi: 10.1186/1743-7075-7-47. PMID: 20515451; PMCID: PMC2890697.
3. Wang HH, Garruti G, Liu M, Portincasa P, Wang DQ. (2017). Cholesterol and lipoprotein metabolism and atherosclerosis: Recent advances in reverse cholesterol transport. Annals of Hepatology. 2017; 16(1)27-42.
4. Alavi N, Khan SH, Saadia A, Naeem T. Challenges in Preanalytical Phase of Laboratory Medicine: Rate of Blood Sample Nonconformity in a Tertiary Care Hospital. EJIFCC. 2020 Mar 20;31(1):21-27. PMID: 32256286; PMCID: PMC7109505.
5. Lorenzo Lozano MC, Cosmen Sanchez A, Belinchón Torres PM, Prieto Menchero S, Pineda - Tenor D. Is fasting necessary for lipid profile determinations? Some considerations from the perspective of the clinical laboratory. Clin Chem Lab Med. 2017;55(9):187-8.
6. Nordestgaard BG, Langsted A, Mora S, Kolovou G, Baum H, Bruckert E, Watts GF, Sypniewska G, Wiklund O, Borén J, Chapman MJ, Cobbaert C, Descamps OS, von Eckardstein A, Kamstrup PR, Pulkki K, Kronenberg F, Remaley AT, Rifai N, Ros E, Langlois M; European Atherosclerosis Society (EAS) and the European Federation of Clinical Chemistry and Laboratory Medicine (EFLM) joint consensus initiative. 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. Eur Heart J. 2016;1:37(25):1944-58.
7. Catapano AL, Graham I, De Backer G, Wiklund O, Chapman MJ, Drexel H, Hoes AW, Jennings CS, Landmesser U, Pedersen TR, Reiner Ž, Riccardi G, Taskinen MR, Tokgozoglu L, Verschuren WMM, Vlachopoulos C, Wood DA, Zamorano JL, Cooney MT; ESC Scientific Document Group. 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias. Eur Heart J. 2016; 14:37(39):2999-3058.
8. Vittal BG and Abhijith D, Fasting and non-fasting lipid profile: A comparative study. JABR, 2021;

5(1): 06-08 . DOI: https://doi.org/10.33545/26174693.2021.v5.i1a.56

9. Scartezini M, Ferreira CEDS, Izar MCO, Bertoluci M, Vencio S, Campana GA, Sumita NM, Barcelos LF, Faludi AA, Santos RD, Malachias MVB, Aquino JL, Galoro CAO, Sabino C, Gurgel MHC, Turatti LAA, Hohl A, Martinez TLDR. Positioning about the Flexibility of Fasting for Lipid Profiling. Arq Bras Cardiol. 2017;108(3):195-197. doi: 10.5935/abc.20170039. PMID: 28443960; PMCID: PMC5389867.
10. Agoro ES, Alabere A. Concentrations of Selected Metals and Polycyclic Aromatic Hydrocarbons in Fresh and Fried Bayelsa Suya (Oryctes rhinoceros) Sold in Bayelsa State, Nigeria. Asian Sci. Bull. 2025; 3 (1): 32-38.
11. Agoro ES, Madu FU. Forensic Significance of Dried Seminal and Vaginal Fluid, Biochemical Parameters in the Corroboration of the Occurrence of Rape. Int J Health Res Medico Leg Prae. 2024; 10(1):17-23.
12. Agoro ES, Adias TC, Ikimi CG. Use of Anopheles Mosquito Bloodmeal's Serology and Haemoglobin Genotypes in Crime Scene Forensics.Asian J. Biol. Sci. 2024;17 (4): 574-581.
13. Agoro ES, Ikimi CG. Translocation capacity of some heavy metals in aquatic food chain of crude oil impacted community of Imiringi in Bayelsa state, Nigeria. Toxicology Research and Application. 2022; 6: 1–8, DOI: 10.1177/23978473221146222
14. Agoro ES, Johnson JT. (2022). Translocation Concept of Heavy Metals in Plantain Consumption Chain of Crude Oil Impacted Community in Bayelsa State Nigeria. Journal of Industrial Pollution Control. 2022; DOI: 10.4172/0970-2083.001
15. Adias TC, Ajugwo AO, Erhabor T, Nyenke CU. Effect of Pumpkin Extract (Telfairia occidentalis) on Routine Haematological Parameters in Acetone-Induced Oxidative Stress Albino Rats." American Journal of Food Science and Technology. 2013; 67-69.
16. Araoye MO. Sample Size Determination. Research Methodology with Statistics for Health and Social Sciences. 1st Edition, Nathadex Publishers, Ilorin. 2004; 115-120.
17. Sävendahl L, Underwood LE. Fasting increases serum total cholesterol, LDL cholesterol and apolipoprotein B in healthy, nonobese humans. J Nutr. 1999 ;129(11):2005-8. doi: 10.1093/jn/129.11.2005. PMID: 10539776.
18. Steen DL, Umez-Eronini AA, Guo J, Khan N, Cannon CP. The effect of fasting status on lipids, lipoproteins, and inflammatory biomarkers assessed after hospitalization for an acute coronary syndrome: Insights from PROVE IT-TIMI 22. Clin Cardiol. 2018;41(1):68-73. doi: 10.1002/clc.22851. Epub 2017 Dec 28. PMID: 29283450; PMCID: PMC6489711.
19. SathiyakumarV, Park J,Golozar A, Lazo M, Quispe R, Guallar E, Blumenthal RS, Jones SR, Martin SS. Fasting Versus Nonfasting and Low-Density Lipoprotein Cholesterol Accuracy. CIRCULATIONAHA. 2017; 137:1.
20. 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.
21. Nordestgaard BG, Hilsted L, Stender S. Plasma lipids in non-fasting patients and signal values of laboratory results. Ugeskr Laeger. 2009; 171:1093
22. NICE Guidance. Cardiovascular Disease: Risk Assessment and Reduction, Including Lipid Modification. NICE. Updated 2016 Available at: https://www.nice.org.uk/guidance/cg181/resources, Accessed 7th Aug 2017
23. Anderson TJ, Grégoire J, Pearson GJ, Barry AR, Couture P, Dawes M, Francis GA, Genest J Jr, Grover S, Gupta M, Hegele RA, Lau DC, Leiter LA, Lonn E, Mancini GB, McPherson R, Ngui D, Poirier P, Sievenpiper JL, Stone JA, Thanassoulis G, Ward R. 2016 Canadian Cardiovascular Society Guidelines for the Management of Dyslipidemia for the Prevention of Cardiovascular Disease in the Adult. Can J Cardiol. 2016;32(11):1263-1282.
24. Leung AA, Nerenberg K, Daskalopoulou SS, McBrien K, Zarnke KB, Dasgupta K, Cloutier L, Gelfer M, Lamarre-Cliche M, Milot A, Bolli P, Tremblay G, McLean D, Tobe SW, Ruzicka M, Burns KD, Vallée M, Prasad GV, Lebel M, Feldman RD, Selby P, Pipe A, Schiffrin EL, McFarlane PA, Oh P, Hegele RA, Khara M, Wilson TW, Penner SB, Burgess E, Herman RJ, Bacon SL, Rabkin SW, Gilbert RE, Campbell TS, Grover S, Honos G, Lindsay P, Hill MD, Coutts SB, Gubitz G, Campbell NR, Moe GW, Howlett JG, Boulanger JM, Prebtani A, Larochelle P, Leiter LA, Jones C, Ogilvie RI, Woo V, Kaczorowski J, Trudeau L, Petrella RJ, Hiremath S, Drouin D, Lavoie KL, Hamet P, Fodor G, Grégoire JC, Lewanczuk R, Dresser GK, Sharma M, Reid D, Lear SA, Moullec G, Gupta M, Magee LA, Logan AG, Harris KC, Dionne J, Fournier A, Benoit G, Feber J, Poirier L, Padwal RS, Rabi DM; CHEP Guidelines Task Force. Hypertension Canada's 2016 Canadian Hypertension Education Program Guidelines for Blood Pressure Measurement, Diagnosis, Assessment of Risk, Prevention, and Treatment of Hypertension. Can J Cardiol. 2016 ;32(5):569-88.
25. Miller M, Stone NJ, Ballantyne C, Bittner V, Criqui MH, Ginsberg HN, Goldberg AC, Howard WJ, Jacobson MS, Kris-Etherton PM, Lennie TA, Levi M, Mazzone T, Pennathur S; American Heart Association Clinical Lipidology, Thrombosis, and Prevention Committee of the Council on Nutrition, Physical Activity, and Metabolism; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular Nursing; Council on the Kidney in Cardiovascular Disease. Triglycerides and cardiovascular disease: a scientific statement

from the American Heart Association. Circulation. 2011, 24;123(20):2292-333.
26. O'Malley PG, Arnold MJ, Kelley C, Spacek L, Buelt A, Natarajan S, Donahue MP, Vagichev E, Ballard-Hernandez J, Logan A, Thomas L, Ritter J, Neubauer BE, Downs JR. Management of Dyslipidemia for Cardiovascular Disease Risk Reduction: Synopsis of the 2020 Updated U.S. Department of Veterans Affairs and U.S. Department of Defense Clinical Practice Guideline. Ann Intern Med. 2020, 17;173(10):822-829.
27. Jellinger PS, Handelsman Y, Rosenblit PD, Bloomgarden ZT, Fonseca VA, Garber AJ, Grunberger G, Guerin CK, Bell DSH, Mechanick JI, Pessah-Pollack R, Wyne K, Smith D, Brinton EA, Fazio S, Davidson M. American Association Of Clinical Endocrinologists And American College Of Endocrinology Guidelines For Management Of Dyslipidemia And Prevention Of Cardiovascular Disease. Endocr Pract. 2017;23(2):1-87.
28. Eberly LE, Stamler J, Neaton JD; Multiple Risk Factor Intervention Trial Research Group. Relation of triglyceride levels, fasting and nonfasting, to fatal and nonfatal coronary heart disease. Arch Intern Med. 2003, 12;163(9):1077-83.
29. Kamrul-Hasan ABM, Talukder SK, Kabir MA, Mustari M, Un Nabi MM, Gaffar AJ, Hossain MF, Alam MS, Islam MR, Hannan MA, Zarin N, Paul AK, Akter F, Ahammed A, Kabir ML, Rahman MM, Asaduzzaman M, Saifuddin M, Chanda PK, Rafi MA, Hasan MJ, Selim S. Comparison of fasting and random lipid profiles among subjects with type 2 diabetes mellitus: an outpatient-based cross-sectional study in Bangladesh. Diabetol Metab Syndr. 2023, 26;15(1):139. doi: 10.1186/s13098-023-01120-y. PMID: 37365577; PMCID: PMC10291787.
30. Gupta S, Kaur H. Inhibition of Glycolysis for Glucose Estimation in Plasma: Recent Guidelines and their Implications. Indian J Clin Biochem. 2014;29(2):262-4.
31. Wang AG, Xia T, Chu QL, Zhang M, Liu F, Chen XM, Yang KD. Effects of fluoride on lipid peroxidation, DNA damage and apoptosis in human embryo hepatocytes. Biomed Environ Sci. 2004 ;17(2):217-22.
32. Le XT, Le NTM. Effects of Sodium Fluoride – Potassium Oxalate on Blood Lipid Profile Results and Lipid Stability when using this Anticoagulant. Med Pharm Res 2023;7(2):59-63.
33. Anto, O.E., Owiredu, W.K.B., Obirikorang, C., Acheampong, E., Amankwaa, B., Afranie, B.O., Donkor, S., Segbefia, S.P., Osei, A.A. Influence of K2EDTA, sodium citrate and lithium heparin anticoagulants on lipid profile in plasma samples as measured by an automated chemical analyzer. Biomed Res Clin Prac, 2017, 2(5): 1-4.
34. Kriško, A., Piantanida, I., Kveder, M., Pifat, G., Lee, A., Greilberger, J., Kipmen-Korgun, D., Jürgens. G. The effect of heparin on structural and functional properties of low-density lipoproteins. Biophysical Chemistry,2006, 119:3,234-239.Agarwala,A., Petersen,K.S., Jafari,F.,Kris-Etherton,P.M.
35. Dietary management of dyslipidemia and the impact of dietary patterns on lipid disorders,Progress in Cardiovascular Diseases,2022,75:49-58.
36. Bays, H.E., Toth,P.P., Kris-Etherton, P.M., Abate, N., Aronne,L.J., Brown, W.V., Gonzalez-Campoy, J.M., Jones, S.R., Kumar, R., Forge, R.L., Samuel, V.T. Obesity, adiposity, and dyslipidemia: A consensus statement from the National Lipid Association, Journal of Clinical Lipidology, 2013,7(4):304-383.