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Association of Lipid Profile with Selenium Status of People Living with HIV in Botswana

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Abstract: Selenium is an essential trace element that plays a vital role in antioxidant defense and immune function especially in people living with HIV (PLWH). This population is vulnerable to micronutrient imbalances, due to chronic inflammation, increased oxidative stress, and altered nutrient absorption. The study aimed to investigate the relationship between serum selenium levels and lipid profiles (total cholesterol, triglycerides, HDL-C, LDL-C), age, and gender in people living with HIV (PLWH). A cross-sectional study of 108 individuals at Princess Marina Hospital. Serum selenium was measured using ELISA, and lipid profiles/demographic data were retrieved from medical records and LIMS. Data analysis utilized R Studio with normality testing, Pearson correlation, and multivariate linear regression adjusted for age and sex. From the results, serum selenium concentration averaged 119.48 ng/ml and showed no significant association with total cholesterol, LDL-C, HDL-C, triglycerides, or gender ($p > 0.05$). After adjusting for covariates, an increase in age was significantly associated with higher triglyceride levels. Serum selenium is likely not a primary determinant of lipid metabolism in this population. The findings underscore the need for longitudinal studies to better understand nutritional and metabolic challenges, which can improve clinical care and targeted nutritional interventions for PLWH.

Keywords: *Selenium, Trace Element, Antioxidant Defense, Immune Function, HIV, Micronutrient Imbalances, Oxidative Stress, Serum Selenium Concentration, Lipid Profiles, Total Cholesterol, Triglycerides, HDL-C, LDL-C, Cross-Sectional Study, Multivariate Linear Regression.*

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I. INTRODUCTION

According to the (Shrivastav, et al., 2025) at least 42.3 million individuals have lost their lives to Human Immune deficiency Virus and AIDS and approximately 39.9 million were living with HIV and AIDS. In 2022, (Simela, et al., 2025) found out that 39 million (33.1- 45.7 million) individuals were living with HIV. A total 67% of these infected individuals are from Sub-Saharan Africa (Worku, et al., 2020; Moyo, et al., 2023). This is the highest cases of HIV and AIDS by region in the world. As part of Sub-Saharan Africa, Botswana is among the countries with the highest disease prevalence: having the third highest number of cases globally. The first HIV case was reported in 1985 in Selebi Phikwe (Song, et al., 2023) and ever since then, HIV and AIDS infections have been recorded in both children and adult citizens (Mupundu, 2017). The highest death cases were reported in the early 2000, before introduction of the antiretroviral drugs (Farahani, et al., 2014). To date, Botswana has a high HIV prevalence of 19.7%, following Eswatini and Lesotho, making it the third highest in the world (Ofori, 2024).

HIV affects the body in many ways. It results in gradual weakening of the immune system and oxidative stress when left untreated. This is because HIV targets the CD4+ T helper cells: the white blood cells responsible for coordinating immune responses (Bekker, et al., 2023). Over time, as the cells are destroyed the body becomes more vulnerable to opportunistic infections and other complications. Without proper management HIV advances to a more dire stage: AIDS, where immune function is severely compromised resulting in life threatening infections and malignancies such as lymphoma (Masenga, et al., 2023). This is because the virus targets and destroys CD4+ T helper cells which play an important function in coordinating immune responses, and over time this leads to immune dysfunction making it difficult for the body to fight of opportunistic infections as CD4 count declines (Isaguliants, et al., 2021).

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HIV infection triggers chronic inflammation and persistent immune activation which then generates high levels of reactive oxidant stress (Tingxia, et al., 2021). When reactive oxygen species (ROS) build up more quickly than cellular antioxidants can neutralize them, oxidative stress results. Excess ROS generation in HIV infection is driven by both host responses and viral mechanisms. For instance, HIV 1 proteins like trans-activator of transcription (TAT) and glycoprotein (GP120) cause redox imbalance by activating host enzymes like NADPH oxidases and compromising antioxidant mechanisms. Thus, impaired antioxidant defenses and increased oxidative stress are common in infected people, which aids in HIV pathogenesis and related problems (Harshithkumar, et al., 2024; Chen, 2024). According to research by (Schank, et al., 2023) CD4+ T cells from ART-controlled PLWH had noticeably greater amounts of mitochondrial and cellular ROS than those from healthy people. Mitochondrial dysfunction and decreased production of antioxidant defense proteins such as apurinic/apyrimidinic endonuclease 1 (APE1) and superoxide dismutase 1 (SOD1) were linked to this rise in ROS. According to (Gusev & Zhuravleva, 2022) by definition, an essential reaction to infection or tissue damage is inflammation. Even with ART, systemic inflammation and persistent immunological activation continue in HIV-positive individuals. According to studies by (Gusev & Zhuravleva, 2022), "chronic immune activation and inflammation, which are predictors of disease progression," are characteristics of HIV infection. In fact, higher viral set points, a higher risk of non-AIDS, and a faster loss of CD4+ T cells are all associated with enhanced inflammatory markers (such C-reactive protein, IL 6, or sCD14). In other words, poorer clinical outcomes are predicted by a high inflammatory burden in HIV It is characterized by several elements, such as a condition in which the body's antioxidant defenses are overpowered by too many reactive oxygen species, which lowers the amount of antioxidant micronutrients. Protease inhibitors, (such as ritonavir, lopinavir and nelfinavir) one of the ART medications included in the multidrug classes advised for HIV regimens in Botswana, are specially utilized in ART. It is a common third agent linked to elevated oxidative stress, which contributes to diseases like diabetes and dyslipidemia, and, finally, disruptions of the antioxidant defense system, including glutathione and selenium (Martemucci, et al., 2023).

In as much as ART has significantly transformed HIV from a fetal disease into a manageable chronic condition by suppressing viral replication and reducing the rate of immune deterioration it is also associated with metabolic complications such as disruptions in lipid metabolism which increases the risk of cardiovascular disease (Sapuła, et al., 2022). For instance, research by (Lagathu, et al., 2019) states that altered fat redistribution has been related to first generational nucleosides-reverse-transcriptase-inhibitors (NRTIs) (stavudine zidovudine) and some protease inhibitors (PIs) which resulted in weight gain. Another study by (da Cunha , et al., 2015) also emphasizes on this finding that “Apart from the substantial benefits that result from the use of various HAART regimens, laboratory and clinical experience has shown that HAART can induce severe and considerable adverse effects related to metabolic complications of lipid metabolism, characterized by signs of lipodystrophy, insulin resistance, central adiposity, dyslipidemia, increased risk of cardiovascular disease and even an increased risk of atherosclerosis” This translates into higher morbidity and mortality rates in PLWH (Abarca, et al., 2025). Another study by (Ji, 2023) suggests that protease inhibitors increase triglyceride and low-density lipoprotein cholesterol by causing mitochondrial toxicity affecting adipocyte function and promoting hepatic lipogenesis (Ji, 2023). According to (Jacobi, et al., 2016) hepatic lipogenesis is a condition in which patients may experience peripheral fat loss but have higher central fat accumulation.

It is worth noting that not all the drug regimen for HIV are associated with this complication, however, it is associated with the interaction of ritonavir and pregnase x receptor. Ritonavir is found in the commonly used regimen in Botswana as a booster for darunavir (DRV/r) in antiretroviral therapy regimens (Quercia, et al., 2024). This therefore raises the concern of the long-term management of HIV. This is because the ART regimen used in Botswana include protease inhibitors and a combination of (such as ritonavir, lopinavir and nelfinavir) which induce mitochondrial dysfunction, oxidative stress and alteration in lipid transport and metabolism resulting in dyslipidemia (Schank, et al., 2021; Manda, et al., 2011). This highlights a broader impact of HIV and its management other than immune suppression only rather, it highlights that although preventing HIV replication and restoring immunological function are the main objectives of ARVs, prolonged use of these medications may have physiological side effects that go beyond immune system recovery. These include consequences including lipid abnormalities, mitochondrial malfunction, oxidative stress, and metabolic issues, which are increasingly being recognized in the literature (Jiménez, et al., 2022). According to these clinical trials, PLWH particularly those receiving antiretroviral therapy (ART) have greater levels of oxidative stress indicators than people without HIV. Reduced antioxidant capacity is linked to this oxidative imbalance, indicating that oxidative stress is a result of both HIV infection and its therapy (Antonella, et al., 2009).

Given the role of oxidative stress in HIV progression and metabolic complication, antioxidant defense mechanism becomes crucial in disease management. For instance, microelement selenium: A vital vitamin, crucial for cardiovascular health; both excess and deficiency are associated with poor cardiac results. The serious cardiac consequences of insufficient selenium intake are best illustrated by Keshan disease, an endemic cardiomyopathy that was initially discovered in selenium-deficient areas of China (Gutiérrez, et al., 2022). Widespread selenium supplementation programs have greatly decreased the incidence of Keshan disease as a result of epidemiological research showing a substantial correlation between low environmental selenium levels and the disease's prevalence (Chen, 2012). Mechanistically, a lack of selenium reduces the activity of selenium-containing proteins including thioredoxin reductase (TrxR1) and glutathione peroxidase (GPx1), which are essential for reducing oxidative stress in cardiac tissue. Keshan disease patients' myocardium have

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been found to exhibit elevated levels of oxidative damage indicators, such as 8-hydroxy-2'-deoxyguanosine (8-OH-dG), highlighting the part oxidative stress plays in the disease's pathophysiology (Pei , et al., 2013). On the other hand, excessive selenium consumption has been linked to elevated risks of hypertension and other cardiovascular conditions, indicating a U-shaped relationship between selenium status and cardiovascular health, even though normal levels are protective (Joseph, 2013). Because both excess and lack of selenium can make people more susceptible to cardiovascular problems, it is imperative to maintain adequate levels.

A key component of selenoprotein such as glutathione peroxidase helps to neutralize reactive oxidative stress and reduce oxidative damage (Tinggi, 2008). It also plays a crucial role in mitochondrial function by maintaining mitochondrial integrity through reducing mitochondrial reactive oxidant stress production (Mehta, et al., 2012). A most recent study by (Joy, et al., 2024) estimated the risk of selenium deficiency across various African countries and it found that the Southern African region has an estimate of 26% risk of selenium deficiency among its population. According to (Saka, et al., 2023) selenium deficiency can contribute to oxidative stress thereby increasing oxidation of the lipids causing lipid abnormalities in PLWH. Increased oxidation of lipids leads to acceleration of atherosclerosis which is a hallmark of endothelial dysfunction (Gianazza, et al., 2021).

Endothelial dysfunction is the first step to cardiovascular diseases. Selenium levels have been associated with increased risk of cardiovascular disease (Gianazza, et al., 2021) therefore failure to address selenium deficiency issue can lead to an increased burden of cardiovascular disease (Sharma, 2023) which is already a rising concern among HIV-positive individuals (Ntsekhe & Baker, 2023).

Despite evidence linking selenium deficiency to disease progression and immune dysfunction in PLWH, there is limited research on the direct relationship between selenium levels and lipid metabolism, particularly in Botswana. However a study by (Yousefi Rad , et al., 2019) suggests that while effects of vitamins, minerals and other dietary compounds were beneficial on lipid profile it also concluded that selenium supplementation could also decrease significantly serum levels of Total cholesterols and triglycerides Another meta-analysis study by (Kelishadi , et al., 2022) shows that 9 clinical trials for selenium supplementation was able to reduce significantly serum levels of total cholesterol, triglycerides and VLDL. It is worth noting that some old research papers identify other elements such as copper and zinc as micro minerals associated with abnormal lipid levels. It reports a positive correlation between serum copper concentrations and total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG) (Clifford, et al., 2003) while current studies focus on ART-induced dyslipidemia but do not explore the potential role of micronutrient deficiencies in lipid metabolic changes (Suleman, et al., 2024; Prakash, et al., 2024). Understanding this relationship could inform clinical interventions to optimize HIV management beyond viral suppression. (Obeagu, et al., 2024) Suggest that selenium deficiency is common in people living with HIV and may contribute to faster disease progression, impaired immune function and increased risk of metabolic disturbances (Obeagu, et al., 2024). However, (Pitney, et al., 2009) claims that in their study selenium was only effective at reducing risk of prostate cancer as compared to cardiovascular disease, HIV and AIDS. Another study by (Shreenath, et al., 2023) emphasizes the role of selenium against oxidative stress, selenium dependent thyroid hormone conversion, immune system modulation and impact on HIV-progression. They also found out that selenium deficiency is associated with an increased risk of various health conditions, including cardiovascular disease" (Shreenath, et al., 2023). However, there are contradictory studies which suggest that high selenium levels have been associated with increased risk of developing hypertension, a significant cardiovascular condition (Klaudia , et al., 2024; Di , et al., 2022) This finding highlights the complexity of selenium's role in cardiovascular health and the need for further research to clarify the relationship between selenium levels and cardiovascular diseases.

Hence, this study focuses on selenium status as the independent variable and lipid profile markers such as total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides as the dependent variables. Given that ART regimens, particularly protease inhibitors, are associated with metabolic disturbances (Hatleberg, et al., 2021), additional factors such as ART duration, regimen type, dietary selenium intake, age, and BMI will be considered to control for potential confounders. Although ART has significantly improved the quality of life for PLWH it also causes metabolic side effects especially dyslipidemia (Sapuła, et al., 2022). While protease is strongly linked to these effects, it is still not clear how the duration of ART use affects selenium levels and lipid metabolism. This raises questions such as whether prolonged exposure contributes to oxidative stress that gradually depletes selenium. Additionally, different ART regimens, such as PIs and integrase strand transfer inhibitors (INSTIs), impact lipid metabolism in distinct ways, yet little is known about their influence on selenium status or whether selenium plays a protective role against these metabolic disruptions. Another important factor to consider is age. Aging naturally affects lipid metabolism and antioxidant defenses (Chung, 2021), but for PLHIV on ART, it's unclear whether selenium depletion accelerates with age, potentially compounding metabolic risks. Older individuals living with HIV may be more vulnerable to oxidative stress, making it crucial to understand whether selenium levels decline more significantly in this group. Addressing these gaps will provide a clearer picture of how selenium interacts with ART-related metabolic changes and could inform better strategies for managing long-term health in PLHIV. This highlights the need for a broader perspective in HIV care that integrates both antiviral and metabolic considerations.

HIV has been extensively examined; however, studies exploring the behavior of the lipids in relation to varying selenium concentrations remain underexplored, particularly

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in Botswana. Evidence shows that there is no information available on selenium deficiency and dyslipidemia in HIV infected individuals and further studies need to be done to clarify the role of selenium deficiencies during the progression of HIV infection, as well as the preventive and therapeutic role of supplementation in monitoring the clinical evolution of PLHIV (Rodrigues, et al., 2025). This lack of information makes it difficult to navigate the possible risks that HIV infection poses infected individuals, specifically in relation to the constitution of microelement selenium in their bodies (Rodrigues, et al., 2025). With less information available on this association, development of increased oxidative damage, immune dysfunction, and disturbances in lipid metabolism, which have been the key findings of researches carried outside the country, may remain unaccounted for in clinical settings (Obeagu, et al., 2024). In addition to this, some of the ART regimens used in Botswana function by inhibiting proteases (Mosepele , et al., 2017). These regimens are found to play a role in mitochondrial dysfunction which through a cascade of events, results in dyslipidemia, a major risk factor for cardiovascular disease (Mehta, et al., 2012). However, there is no information available on the how these regimens influence immunological responses in the local people living with HIV in Botswana. Given these gaps, this study seeks to investigate the relationship between selenium status and lipid profile parameters in people living with HIV. Specifically, by comparing lipid profile markers such as total cholesterol, triglycerides, LDL, and HDL cholesterol between individuals with different selenium levels. The null hypothesis (H_0) states that there is no significant difference in lipid profile parameters between individuals with different selenium status levels, meaning that selenium does not have a significant effect on lipid metabolism in people living with HIV. While the alternative hypothesis (H_1) proposes that there is a significant difference in lipid profile parameters between these groups, suggesting that selenium levels may influence lipid metabolism in this population. By addressing this gap locally relevant data will be made available on the potential implications of selenium status on lipid metabolism in HIV-infected individuals, which may contribute to better clinical management strategies and nutritional interventions.

➢ *General Objectives*

To explore the relationship between selenium levels and lipid profile markers in people living with HIV (PLWH) in Botswana. The goal is to determine whether selenium deficiency plays a role in lipid abnormalities and the potential risk of cardiovascular disease in this population.

➢ *Specific Objectives*

- To measure selenium levels in PLWH undergoing antiretroviral therapy (ART) in Botswana.
- To analyze the relationship between selenium levels and lipid profile markers in PLWH.
- To account for possible influencing factors such as age and gender.

➢ *Research Hypotheses*

- Null hypothesis: There is no significant relationship between serum selenium concentration and lipid profiles in PLWH.
- Alterative hypothesis: There is a significant relationship between serum selenium concentration lipid profiles in PLWH.

II. MATERIALS AND METHODS

➢ *Study Design*

This study employed a cross-sectional analytical design to investigate the association between selenium levels and lipid profile markers in people living with HIV (PLWH) undergoing antiretroviral therapy (ART) in Botswana. A cross-sectional study was found advantageous because it allowed for the assessment of selenium levels and lipid profile simultaneously without intervention. This ensured that less time was spent between collecting data while reducing the costs as well. Furthermore, it enabled the identification of associations between selenium levels and lipid profiles without requiring long-term follow-up (Holy, et al., 2018). However, one key limitation was the inability to establish causality, as the exposure and outcome were measured at the same time this limitation was overcome by using the duration of enrolment in ART as an instrumental variable. Only patients who had been enrolled on ART for more than 6 months were considered. This information was obtained using the laboratory information management system (LIMS). Additionally, cross-sectional studies are susceptible to biases such as selection bias and information bias. These limitations were addressed by standardized data collection process. This limitation was reduced by using inclusion and exclusion criteria.

➢ *Study Site*

The study was conducted at Princess Marina Hospital where serum samples are routinely collected for HIV monitoring. The selection of these sites ensured accessibility to a well-characterized patient population and high-quality laboratory infrastructure. Laboratory processing of blood samples was conducted in the University of Botswana, Faculty of Health sciences laboratories.

➢ *Sample Population*

The reference population for this study included all PLWH receiving ART in Botswana. The source population consisted of PLWH attending Princess Marina Hospital for HIV monitoring, where plasma and whole blood samples for HIV monitoring and serum for lipid profile are routinely collected. Due to the sample type availability in this facility, serum from the yellow tube was used for selenium measurements. These samples are justified by their stability and established reliability in biochemical analysis (Ducros, et al., 2000). While selenium levels were measured as part of this study, lipid profile data was obtained from the laboratory's pre-existing test results, as lipid profile testing is routinely performed at the facility.

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Previous studies have been carrying out this test from a range of 67-365 samples (Augusto, et al., 1991; Ranjana, et al., 2010) hence a total of 108 samples was tested for in this study. Random sampling was used to ensure generalizability and reliability of findings. A total of 108 samples collected from the 5th to the 20th of November 2025 were used for the study. This allowed for collection of all samples regardless of age and gender. This provided a generalized representation of where metabolic rates are more likely to change as one ages, as younger individuals generally have lower risk of cardiovascular disease (Anxin, et al., 2021).

➢ *Sample Size Determination*

The sample size was determined using statistical methods appropriate for cross-sectional studies. The formula for estimating sample size in prevalence studies. A comprehensive analysis of dietary mineral supply across African region estimated a 26% risk of selenium deficiency in the Southern African region, of which Botswana is geographically located within. Hence a 26% prevalence rate will be used in determining the sample size.

A 9% margin of error will be used for this study to balance accuracy with limited resources. Under normal circumstances a 5% margin of error would be used, however it would significantly increase the sample size thus also increasing the cost for carrying out the test. On the other hand, 8% margin of error will be used because it falls within an acceptable range for micronutrient deficiency studies while maintaining a reasonable statistical power of 80% (Bhardwai, et al., 2024) .

$$n = \frac{Z^2 \times p \times (1 - P)}{d^2}$$

Where;

n = required sample size,
Z = standard normal variate (1.96 for 95% confidence level),
P = estimated prevalence for selenium deficiency (assumed to be 26% for Southern African region),
d = margin of error (set at 8%) due to limited resources and funding for a bigger sample size.

By using these values, the calculated sample size is approximately 90 patient samples. However, this study decided to utilise a higher sample size so as to enhance the statistical power, hence a sample size of 108 patients will be used instead as it still falls within the range for sample size used in different studies as mentioned under study sample section.

➢ *Inclusion Criteria*

Blood samples of individuals living with HIV (PLWH) who have been on ART for more than 6 months and had a CD4 count of more than 200 were selected for the study. The patients' samples were only used for the study if they had already pre-existing lipid profile results not older than 3 months from the date of selenium measurement. The requirement for at least six months on ART ensured that selenium and lipid profile levels were not influenced by early ART-induced metabolic fluctuations. The first six months of ART initiation involve significant metabolic changes as the body adjusts to the medication. After this period, patients typically achieve stable viral suppression and metabolic homeostasis, allowing for a more accurate assessment of long-term ART effects.

➢ *Exclusion Criteria*

Patients with known conditions that may interfere with lipid or selenium levels, such as chronic inflammatory diseases such as Tuberculosis, liver disease, and chronic kidney diseases were excluded from the study as these conditions may cause exaggerated lipid accumulation. Patients with a significantly low CD4 count of less than 200 cells/µL were also excluded because these patients may represent non-adherence or treatment failure (Mota, et al., 2024). This data was obtained from the laboratory information system.

➢ *Potential Sampling Bias*

Since collection of samples occurred at Princess Marina Hospital, individuals who do not regularly attend this laboratory were excluded. However, it is worth noting that this laboratory gets samples from multiple branches as well as referred samples from other health facilities. This means that participants from multiple geographically diverse clinics were accounted for. All the necessary information was collected using LIMS (Laboratory Information Management System) and laboratory protocols were used to mitigate information bias.

➢ *Data Collection Procedures*

Demographic and clinical data was collected through medical record reviews on LIMS. Data will include age and gender.

➢ *Ethical Considerations*

Ethical approval was obtained from the Botswana Ministry of Health Ethics Committee and UB-IRB for collection of samples at the specified facility. The study complied with Botswana's Data Protection Act of 2018 in order to secure patient identification while gaining access to their current lipid profile information. Before analysis, all personal data was anonymized using coding, meaning that no information that might be used to directly or indirectly identify actual patient was included. Additionally, the study received permission from the appropriate ethics committee, guaranteeing that the handling of private health information conforms to legal and ethical norms. By putting these safeguards in place, the study respected the values of responsibility, confidentiality, and integrity when managing patient data. Samples collected included those that are HIV positive and with their lipid profile results. Lipid profile results were required because they were expensive to measure.

➢ *Laboratory Investigations*

- *Acquisition of Blood Samples*

Blood samples will be obtained from Princess Marina Hospital in serum separator tubes vacutainers on a weekly basis. They were carried in a cooler box suitable to transport

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blood samples while maintaining proper protective wear such as wearing of gloves and lab coat all the time when working in the lab. Medical reports of suitable participants were retrieved from the hospitals LIMS and necessary information was transferred into an excel spreadsheet, leaving out their personal details.

- *Preparation and Separation of Blood Serum*

A total volume of 10 mL of the blood samples from each patient was centrifuged using Centrifuge 5415 R at 1000g for 15 minutes a 2-4°C (Clark, et al., 2003). The supernatant serum obtained was aliquoted into cryovials and stored in a mechanical freezer at -20°C or 80°C until the samples were ready for analysis (International Society for Biological and Environmental Repositories, 2005). This method of preparing blood samples for analysis has been stated by (Malvy, et al., 1994; Clark, et al., 2003). All samples were analyzed for selenium concentration at the University of Botswana school of Allied Health Sciences

- *Determination of Selenium Concentration*

The samples used for the quantitative analytical determination for selenoprotein P in human serum were analyzed with the help of the selenOtest ELISA kit. This test was conducted following proper instructions to ensure correctness in carrying out the incubation periods, volumes, and wash steps involved in this analytical procedure.

Samples of serum were stored and frozen until a later time. The samples were stored at temperatures of less than −60°C. all samples of the serum were stored in tightly sealed cryovial tubes, and no evaporation or change of concentration of the components was possible because of these containers. Before analyzing, the samples that were stored frozen had to be completely thawed to room temperature and were gently mixed.

All reagents and samples were equilibrated at room temperature before starting the assays. In preparing the calibrator stock solution, 1000 µl of reconstitution buffer was added to the calibrator stock vial. After allowing it to stand at room temperature for at least five minutes, it was vortex-mixed briefly for 2-3 seconds and centrifuged. The reconstituted calibrator stock solution was stable at room temperature for at least 24 hours.

The working solution of the detection antibody was obtained by adding 100 µl of the detection antibody concentrate to 12 ml of the detection antibody dilution buffer. The bottle was swirled gently four to five times. The diluted solution of the detection antibodies is stored at room temperature. This should not be used after eight hours.

The 1-fold wash buffer was made by diluting two lots of 50ml volumes of the 10-fold concentrate of the washing buffer with lab-scale water for a final volume of 1000ml. The contents were well mixed prior to use. The precipitates that were present in the concentrate were left to dissolve at room temperature or during the process of dissolution and did not have any effect on the assay process. The buffer was stable at room temperature and could be used for up to seven working days.

Calibration standards and control preparations were made by using the calibrator stock solution and sample dilution buffer according to volumes listed in the quality report. 1.5ml reagent vials, labelled were used for preparation. The sample dilution buffer was first added, followed by a specified amount of calibrator stock solution using a fresh pipette tip each time. These preparations were vortexed for 2-3 seconds and then centrifuged briefly. Prepared calibrations and controls remained stable for at least 24hrs at room temperature and one was assigned for each assay plate run to eliminate variability associated with plates.

A dilution plate was employed to reduce variability. A minimum of 150 µl of each calibrator and control sample, and Control B, were pipetted into columns 1-3 on the dilution plate based on the pipetting scheme predefined. Subsequently, sample dilution buffer was added to the wells assigned for the addition of the serum samples in columns 4-12. The dilution factor employed varied by lot and followed guidelines provided by the manufacturer's quality report however for this kit the same dilution factor was used since the kits were from the same lot number.

Samples were directly pipetted into their respective wells, filled with sample dilution buffer. The samples were aspirated and dispensed once for accurate pipetting, and the duplicate dilution samples were done independently for each sample. The reagents in the dilution plate were mixed by pipetting up and down for three cycles using an 8-channel pipette. 100 µl of the mixed reagents were transferred to the corresponding wells of the Assay plate for each calibrator, control, and diluted sample by changing the tips among transfers to prevent contamination.

The plate was incubated for a period of 45 minutes at a room temperature of 25°C. it was then washed four times with a volume of 250 µl of 1-fold wash buffer using an automatic wash machine.

Following the final incubation step for the assay, 100 µL of TMB Substrate solution (component SU) was added to each well with the use of a multichannel pipette. The assay plate was next incubated at room temperatures (approximately 25°C) for 30 minutes, where it was protected from any direct light with the use of aluminum foil to prevent premature oxidation of the substrate. To stop the enzymatic reaction, 100 µL of stop solution (component ST) was added to the wells and briefly mixed with the use of a microtitre plate shaker. Yellow dye developed in the process was quantified for its absorbance 15 minutes after the addition of the stop solution. Quantification was done by the use of a microtitre plate photometer with an additional detection wavelength at 450 nm. The data for results was evaluated using mean values from the duplicate measurements of the calibrators, controls, and samples from the sera. The blank control (Control Sample B) was used as a reference and not subtracted from the measurements made for the other samples. Concentrations of the Selenoprotein P were calculated from

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the measurement results by use of the 4- or 5-parameter logistics log function by use of rigorously designed statistical software package which is excel and they were recorded for statistical analysis.

➢ *Statistical Analyses*

Descriptive statistics including mean, standard deviation and range, were calculated for selenium levels and lipid profile variables to summarize the central tendency and variability of the data. Data analysis was performed using R studio (version 4.5.2). The Shapiro–Wilk test was performed to assess the normality of the distributions of continuous variables. Spearman correlation was performed to evaluate he correlation between serum selenium concentration and lipid profile parameters (HDL-C, HDL-C, total cholesterol and triglycerides). Multivariate linear regression models were applied to determine the relationship between serum selenium concentration and lipid profiles adjusting for potential cofounders including sex and age. A p-value of <0.05 was considered statistically significant.

III. RESULTS

➢ *Socio-Demographic Characteristics of Sample Population*

Table 1 Socio-Demographic Characteristics of Sample Population

Characteristics	Category	Frequency (n)	Percentage (%)	Total (%)
Sex	Male	30	28	
	Female	78	72	100
Age	18-39	8	7	
	40-59	82	76	
	60+	18	17	100

In table 1 above, 72% of the participants from the cohort were females. Only 30 males (27%) were part of the study. A larger proportion of the participants were aged between 40 and 49 (76%) while only 8 individuals (7%) were aged between 18 and 39 years.

➢ *Serum Selenium Concentrations and Lipid Profile Characteristics*

Table 2 The Confidence Intervals and Descriptive Statistics of Serum Selenium Concentration and Lipid Profile Markers

In ng/L or mmolL	Lower CI	Max_x	Min_x	Mean_x	Sd	se	Upper CI
Serum selenium	110.78	360.77	54.63	119.48	45.58	4.39	128.17
LDL-C	2.25	4.20	0.63	2.37	0.65	0.06	2.49
HDL-C	7.60	2.90	0.40	1.44	0.96	0.09	2.68
Triglycerides	1.33	4.60	0.40	1.48	0.65	0.07	1.63
Total Cholesterol	4.35	7.60	2.50	4.53	0.96	0.09	4.71

The table above shows the confidence intervals and descriptive statistics of serum selenium concentration and lipid profile markers. The standard error (se) of the lipid profiles ranged between 0.06 and 0.96, with serum selenium having the highest se of 4.39. Besides serum selenium, total cholesterol had the highest mean concentration of 4.53 mmol/L. HDL-C had the lowest concentration.

➢ *Histograms of Lipid Profiles and Serum Seleniun Concentration*

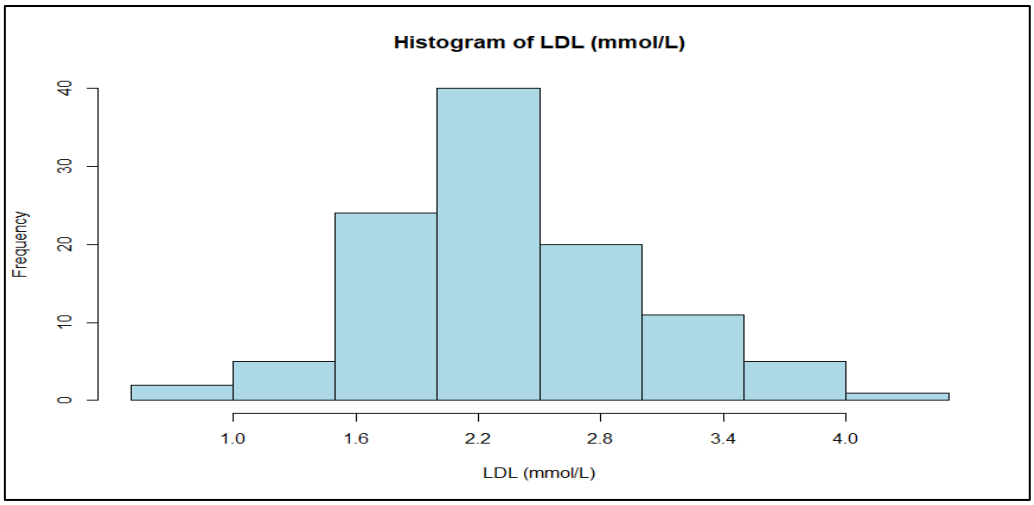


Fig 1 A Histogram of the Frequnecy of LDL (mmol/L) in 108 Sample Participants

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LDL had a mean of 2.37 mmol/L with less than 5% of the sample population having LDL-C levels of less than 1 mmol/L.

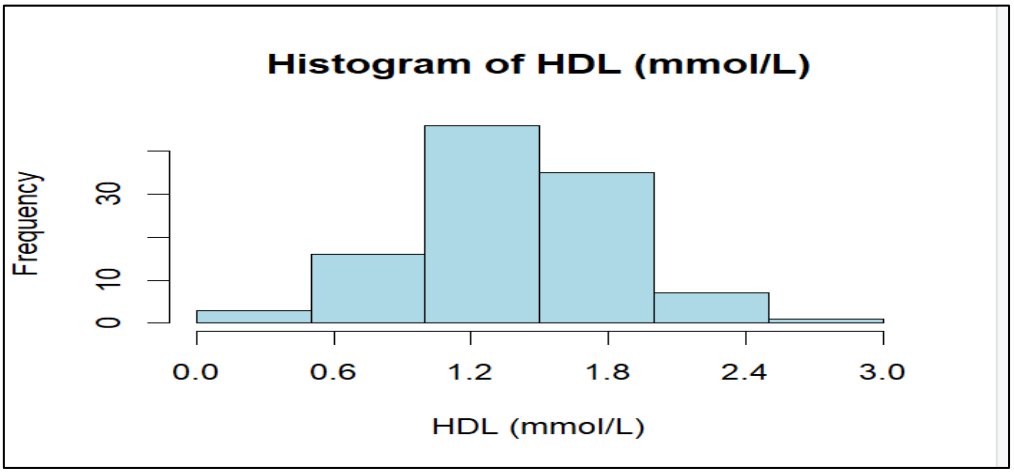


Fig 2 A Histogram of the Frequnecy of HDL (mmol/L) in 108 Sample Participants

HDL-C levels had a mean of 1.44 mmol/L. More than 30 individuals had concentration ranging between 1.0 mmol/L to 2.0 mmol/L.

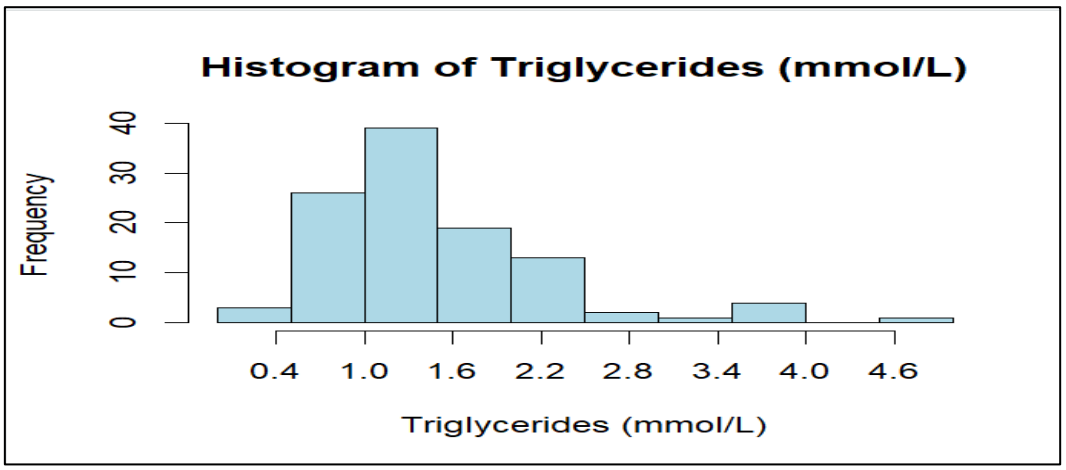


Fig 3 A Histogram of the Frequnecy of Triglycerides (mmol/L) in 108 Sample Participants

A large numner of samples had trigycerides levesl ranging between 1.0 mmol/L to 1.5 mmol/L. Less than 5% of indviduals had triglcerids levles of 4.6 mmol/L.

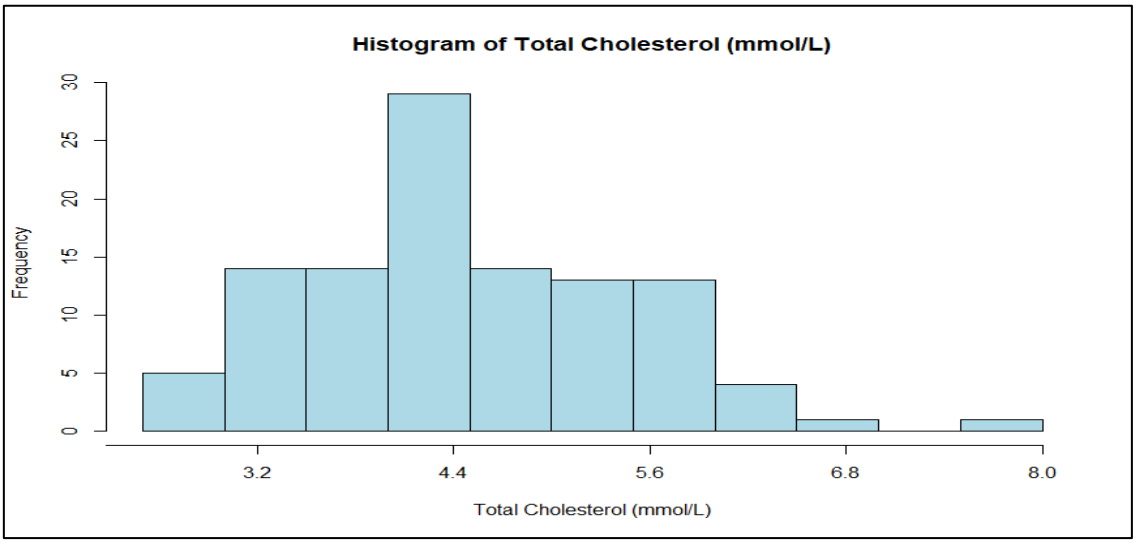


Fig 4 A Histogram of the Frequnecy of Total Cholesterol (mmol/L) in 108 Sample Participants

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Equal number of participants (n=15) had total cholesterol levels between 3.0 mmol/L and 4.0 mmol/L The highest numebr of individuals (n=30) had triglyceride levels ranging between 3.6 and 4.1 mmol/L.

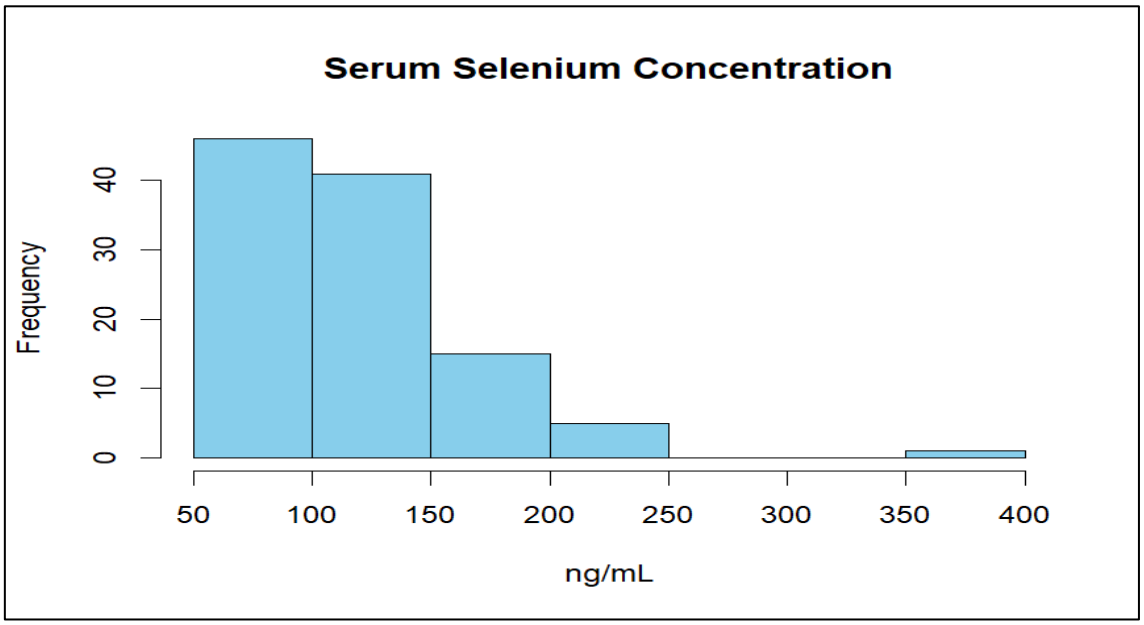


Fig 5 A Histogram of the Frequnecy of Serum Selenium Concentration (ng/mL) in 108 Sample Participants

More than 40 individuals (22%) had serum selenium concentrations ranging between 50 ng/ml and 100 ng/ml. Only 1 individual had extremely high serum selenium concentration of 360.77. No individuals had serum selenium concentration ranging between 250 and 350 ng/l. This is biologically plausible.

➢ *Correlation Analysis*

Table 3 Spearman's Rank Correlation Between Serum Selenium Concentration and Lipid Profile Parameter

Variable 1	Variable 2	Spearmans correlation	P-value	interpretation
Serum Selenium (ng/L)	Total Cholesterol	0.133	0.169	Weak, not significant
	HDL	-0.057	0.556	Very weak, not significant
	LDL	0.143	0.140	Weak, not significant
	Triglycerides	0.132	0.173	Weak, not significant

In table 2 above, Spearman's correlation between serum selenium and lipid profiles shows weak, non-significant associations for all parameters ($p > 0.05$).

➢ *Scatter Plots of Correlation Between Serum Selenium Band Lipid Profiles.*

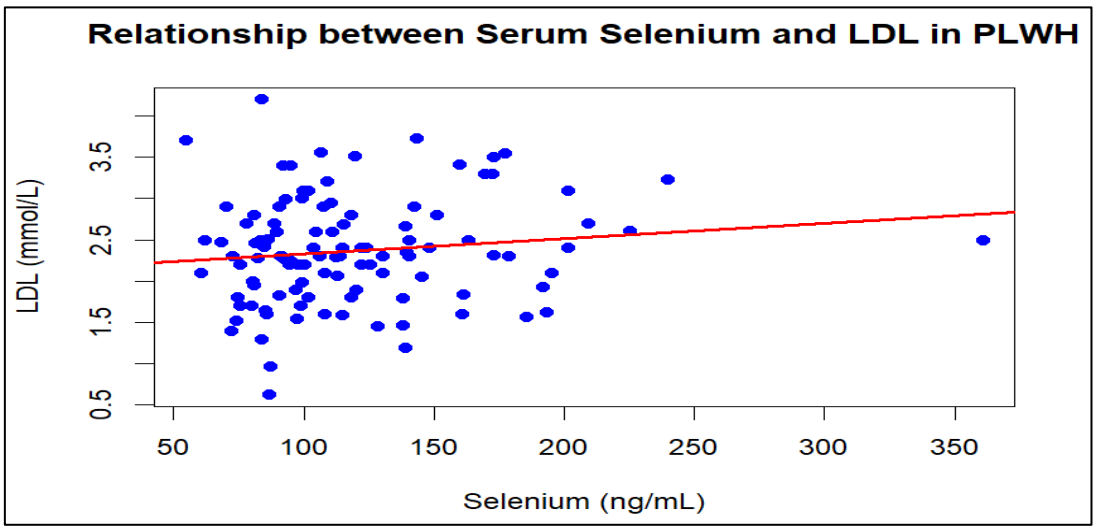


Fig 6 A Scatter Plot of the Correlation Between Serum Selenium Concentration (ng/mL) and LDL (mmol/L)

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This scatter plot shows the relationship between serum selenium concentration and LDL-C in PLWH. The plot demonstrates a weak, non-significant positive association between serum selenium levels and LDL-C.

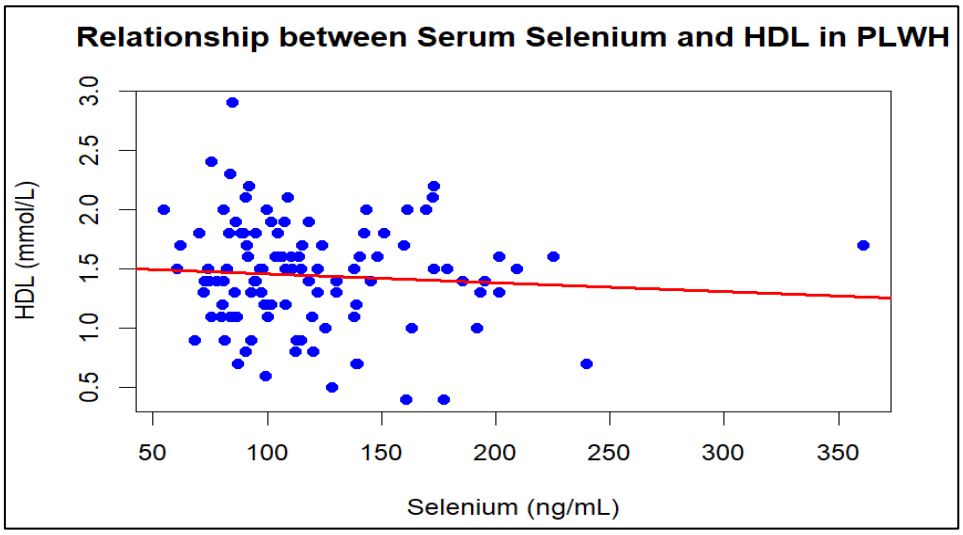


Fig 7 A Scatter Plot of the Correlation Between Serum Selenium Concentration (ng/mL) and HDL (mmol/L)

This scatter plot shows the relationship between serum selenium concentration and HDL-C in PLWH. The scatter plot demonstrates a weak, non-significant negative association between serum selenium and HDL-C.

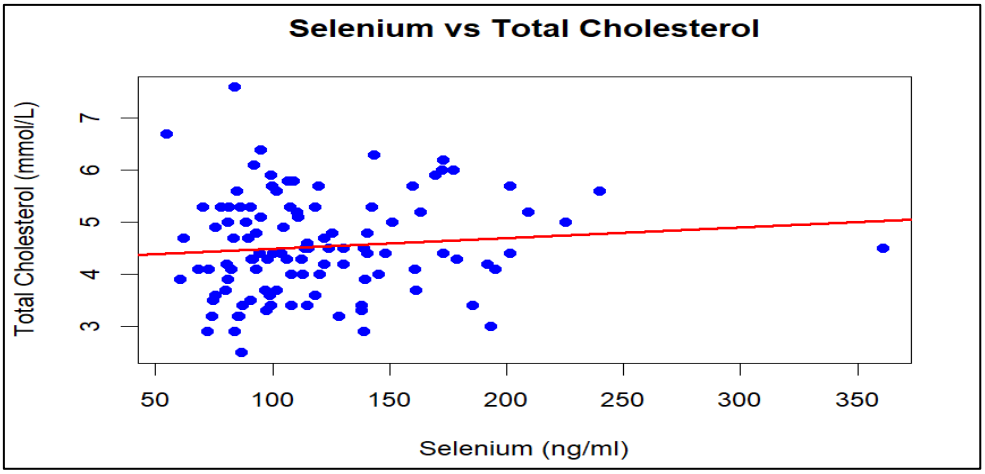


Fig 7 A Scatter Plot of the Correlation Between Serum Selenium Concentration (ng/mL) and Total Cholesterol (mmol/L)

This scatter plot shows the relationship between serum selenium concentration and total cholesterol in PLWH. The scatter plot demonstrates a weak, non-significant positive association between serum selenium and total cholesterol levels.

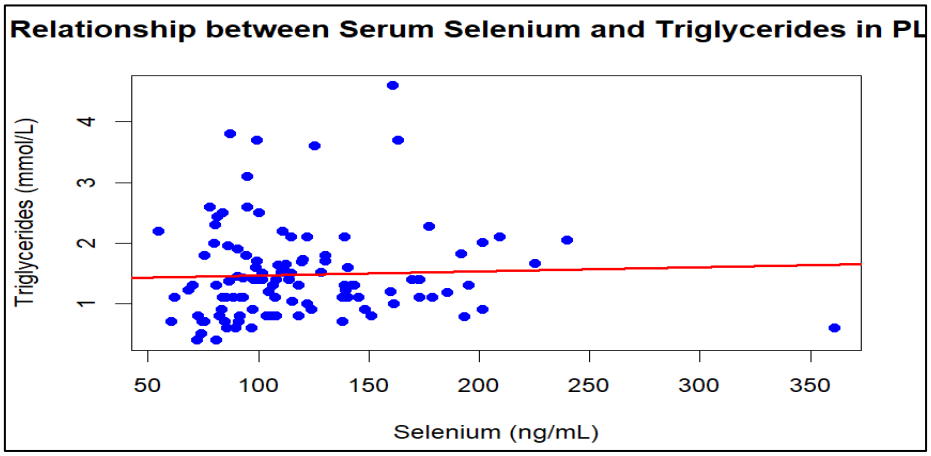


Fig 8 A Scatter Plot of the Correlation Between Serum Selenium Concentration (ng/mL) and Triglycerides (mmol/L)

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This scatter plot shows the relationship between serum selenium concentration and Triglycerides in PLWH. The scatter plot demonstrates a weak, non-significant positive association between serum selenium and Triglycerides.

- *Multivariable Linear Regression Adjusted for Age and Gender*.

Table 4 Multivariable Linear Regression of Lipid Profile Markers on Serum Selenium Concentration Adjusted for Age and Sex.

	Predictor	**β (beta coefficient) for Selenium**	**95% CI**	**p-value**	**R^2**
Total Cholesterol	Intercept	3.553	2.43 – 4.67	<0.001	0.03
	Serum Selenium (ng/mL)	0.00178	−0.0022 – 0.0058	0.386	
	Age (years)	0.0145	−0.0048 – 0.0337	0.144	
	Gender (M)	0.0142	−0.3860– 0.4144	0.945	
LDL Cholesterol	Intercept	2.044	1.29-2.80	<0.001	-0.007
	Serum Selenium (ng/mL)	0.000042	−0.000024 – 0.000109	0.213	
	Age (years)	0.00266	−0.0103−0.0157	0.689	
	Gender (M)	-0.0708	−0.341 – 0.200	0.609	
HDL Cholesterol	Intercept	1.571	1.042 – 2.100	<0.001	−0.021
	Serum Selenium (ng/mL)	−0.0000188	−0.0000649 – 0.0000273	0.427	
	Age (years)	−0.000427	−0.00950 – 0.00865	0.927	
	Gender (M)	−0.0394	−0.228 – 0.149	0.684	
Triglycerides	Intercept	0.0394	−0.799 − 0.878	0.9267	0.120
	Serum Selenium (ng/mL)	0.0000134	−0.0000598 − 0.0000866	0.720	
	Age (years)	0.02348	0.00909 − 0.03787	0.00184	
	Gender (M)	0.4683	0.1688 − 0.7678	0.00277	

Table 4 shows multivariable linear regression results assessing the association between serum selenium and lipid profiles adjusted for age and sex. Serum selenium was not significantly associated with total cholesterol (β = 0.00178, p = 0.386), LDL-C (β = 0.000042, p = 0.213), HDL-C (β = −0.0000188, p = 0.427), or triglycerides (β = 0.0000134, p = 0.720). Increasing age (β = 0.0235, p = 0.0018) and male sex (β = 0.468, p = 0.0028) were significantly associated with higher triglyceride levels, while no significant associations were observed for other lipid markers.

IV. DISCUSSION

The current study established the serum selenium concentration in people living with HIV (n=108) and investigated the relationship between selenium and lipid profile markers such as total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. Further analysis was also done using multivariable regression to take into consideration the potential confounding factors such as age and sex. Overall, these findings show weak and statistically non-significant associations of serum selenium with all lipid parameters in this cohort, which suggests that selenium is not a major determinant of lipid metabolism in people living with HIV. Results provide further detail on the general distribution of the lipid markers studied in this population and, except for triglycerides, age and sex were found to have no significant impact on the observed associations.

The descriptive statistics showed that the mean serum selenium concentration of 119.48 ±45.58 ng/mL (95% CI; 110.78-128.17). These values fall within ranges reported in populations with adequate selenium intake (1). Selenium levels usually vary depending on regional dietary sources, soil micronutrient composition, and supplementation patterns (Hurst, et al., 2013). Adequate selenium is important for selenoprotein synthesis, including glutathione peroxidase, which is integral to antioxidant defense, immune modulation, and protection against oxidative stress (Tinggi, 2008) . Given that individuals living with HIV usually have chronic inflammation, oxidative stress, and increased demand for micronutrients, along with the possible malabsorption associated with certain ART regimens, the generally normal distribution of selenium levels seen in this study indicates that

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this population may have adequate selenium status, likely supported by diet or regional nutritional patterns.

Regarding lipid markers, the mean LDL, HDL, and triglyceride concentrations were 0.4 mmol/L, 2.5 mmol/L, and 0.63 mmol/L, respectively. Histograms of Figures 1–4 show that lipid values are approximately normally distributed, with a slight positive skew of triglycerides that is commonly seen in clinical chemistry (Christensen, et al., 1980). Compared to general populations, lipid values were relatively low. This trend is well documented among people living with HIV due to dysregulated lipid metabolism associated with chronic inflammation, immune activation, and long-term use of ART, particularly older regimens associated with lipodystrophy and metabolic disturbances (Barchielli, et al., 2022). In many cohorts of HIV-positive individuals, HDL is usually low with a mean value of 1.6 mmol/L while LDL and triglycerides tend to vary with a mean value of 2.6 mmol/L and 1.7 mmol/L respectively depending on the type of ART used and disease progression, consistent with the trend in this dataset.

Spearman's rank correlation analysis showed very weak and non-significant associations between serum selenium and all markers of the lipid profile. The respective correlation coefficients were 0.133 (p=0.169), 0.057 (p=0.556), 0.143 (p=0.140), and 0.132 (p=0.173) for total cholesterol, HDL, LDL, and triglycerides, respectively. Although the direction of association was positive for total cholesterol, LDL, and triglycerides, and slightly negative for HDL, the effect sizes were minimal and did not reach statistical significance. This suggests that selenium concentration is not meaningfully associated with the lipid levels within this population.

The weak associations can be understood in the context of the biological interplay between selenium and lipid metabolism. Selenium, through selenoenzymes such as GPx, is part of antioxidant pathways that protect cellular membranes from lipid peroxidation (Mehta, et al., 2012) . Theoretically, adequate selenium should improve lipid profiles through reduced oxidative modification of lipoproteins and modulation of inflammatory pathways. Yet, complicating factors include the fact that oxidative stress is chronically elevated in HIV infection and thus may overwhelm selenium-dependent antioxidant systems; that certain ART medications strongly influence lipid metabolism independent of micronutrient status; and that considerable interindividual variability exists in selenium absorption and selenoprotein expression. Moreover, ART regimen duration, diet quality, adiposity, and metabolic comorbidities may obscure the contribution of selenium to lipid homeostasis and explain the absence of significant associations observed in this study.

After adjustment for age and sex, serum selenium concentration was not significantly associated total cholesterol ($\beta = 0.000043$, $p = 0.386$), LDL ($\beta = 0.000042$, $p = 0.213$) and was inversely associated with HDL ($\beta = -0.0000188$, $p = 0.427$). This indicates that in the present study, the change in serum selenium concentration does not independently predict the lipid profile with adjusted age and sex. Although many studies (Di Bella *et al.* 2010; Poudel-Tandukar *et al.* 2023; Osuna-Padilla *et al.* 2024) link HIV infection with selenium deficiency and chronic inflammation, the lack of association between selenium concentration and the lipid profiles may indicate adequate baseline serum selenium in the population of Botswana. Skalny *et al* (2019) and Gashu *et al* (2019) explained that serum selenium concentrations vary in different geographical regions due to soil type of inhabitants, diet and genetics. The findings of the present study are consistent with those obtained by Osuna-Padilla *et al* (2024) who found out that serum selenium was not significantly associated with any lipid profiles even after six months of supplementation in PLWH (Osuna-Padilla *et al.*, 2024).

However, a study conducted by González-Estecha *et al* (2017) indicated that higher selenium concentration was associated with increased total LDL cholesterol and not HDL cholesterol and triglycerides in the general adult population of Spain. Their findings are like those obtained by Dave *et al* (2016) who found out that PLHV on ART had relatively higher lipid profiles compared to those who are ART-naïve. However, severe dyslipidemia that is always associated with HIV infection and treatment affected only <5% of the population in their study (Dave *et al.* 2016). Contrary to or findings where 108 participants' blood samples were used, a related analysis of 4132 adults from National Health and Nutrition Examination Survey (NHANES) reported that high serum selenium concentration were associated with high lipid profiles in a large, national representative sample. This the importance of sample population (n) in data analysis. However, it is important to note that Mazaheri-Tehrani *et al.* (2025) argued that the relationship between serum selenium concentration, most lipid profiles and confounding factors vary across studies and there is no consistency in the findings. In their review, only HDL-cholesterol showed weak correlation among adults ($r = 0.1$ [0.03:0.17]; $I^2 = 71\%$).

In contrast to the other lipid markers, age and sex were significant predictors of triglyceride levels. Although serum selenium remained non-significant ($\beta = 0.0000134$, $p = 0.720$), age showed a significant positive association with triglycerides ($\beta = 0.02348$, $p = 0.00184$), indicating that triglyceride levels increase with advancing age. The findings of this study are not consistent with those obtained by González-Estecha et al (2017) whom in their study found out that higher serum selenium was positively associated with LDL-cholesterol and not triglycerides. In this study, the HIV status of the individuals was not considered.

Male sex was also independently associated with higher triglyceride levels ($\beta = 0.4683$, $p = 0.00277$). The model explained 12.0% of the variability in triglyceride levels ($R^2 = 0.120$), indicating that sex contribute to triglyceride variation. Higher triglycerides in males may be related to sex-specific hormones such as testosterone, body fat distribution, and physical activity levels. The findings of this study are in line with those obtained by Assefa *et al* (2023) who in their study concluded that males undergoing ART treatment in Ethiopia were associated with dyslipidemia (which includes elevated triglycerides). Yu *et al*. (2023) also found out that males have

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high incidence (21%) of elevated triglycerides and covariates, not considered in the present study (body status, ART regimen characteristics and immune status) contribute to triglyceride variation in males with HIV. Contrary to these findings, Kigongo *et al* (2024) found out that female sex (aPR:1.55, 95%CI: 1.32–1.84, $p < 0.001$) was independently associated with dyslipidemia which comprised of elevated triglycerides. Similarly**,** Agnieszka Lembas et al. (2025) found out that female sex was associated with high triglyceride concentrations among other lipid abnormalities.

According to Jericho *et al.* (2005) and Odikro *et al.* (2025) increasing age heightens metabolic syndrome, proving that as individuals age, more lipid profiles like triglycerides and cholesterol are more likely to accumulate as a result of age-related metabolic impairment (Jericho *et al.* 2005; Odikro *et al.* 2025). This was also the case in the present study, an increase in age was associated with an increase in triglycerides ($\beta = 0.02348, 0.00184$). However, it is important to note that, this was independent of serum selenium concentration. The findings of the present study are like those obtained by Samuel *et al.* (2014) who found out that age-related changes in lipid metabolism influence the concentration of triglycerides in PLWH. In their study, older age was associated with lower hemoglobin and plasma albumin and HDL-C concentrations, but higher Total cholesterol, LDL-C and triglyceride concentrations. Schneider-Matyka (2025) conducted a study focusing on only females and their results were consistent with those of the present study. Their study concluded that increasing age was strongly associated with higher triglycerides, indicating that indeed an increase in age increases the metabolic impairment regardless of gender.

The model explained insignificant variability for total cholesterol, LDL and HD after adjustment of sex and age. This indicates that other factors not included in the study may play a larger role in influencing these lipid parameters. This signifies the importance of considering other factors when dealing with associations between serum selenium and lipid profiles in PLWH. For instance, CD4 count. Osuna-Padilla *et al* (2024) found out that CD4 T cells percentage increased after 6 months of selenium supplementation.

V. CONCLUSIONS

In this study, it was found out that serum selenium was not independently associated with lipid profiles among PLWH. This suggests serum selenium may not be a key determinant of lipid metabolism in the sample population. However, an increase in age was significantly associated with higher triglyceride levels. Other cofounding factors, not considered in this study possibly have an influence in serum selenium concentrations and lipid profiles. For instance, ART regimen and genetics.

RECOMMENDATIONS

The findings of this study highlight a need to carry out longitudinal studies to determine the nutritional and metabolic challenges in this population, providing evidence that may inform clinical management and nutritional interventions. More robust studies where multiple understudied cofounding factors are investigated needs to be conducted to exhaust all the possible factors that influence dyslipidemia in PLWH.

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- *Conflict of Interest*

The authors declare that they have no conflict of interest.

- *Author Contributions*

Simiso Mamba and Dr Zachariah Matshediso conceptualised and originated the study idea. Simiso Mamba conducted the statistical analysis and prepared the manuscript. Mukuka Brian and Chinakila Julius Musonda reviewed the manuscript

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