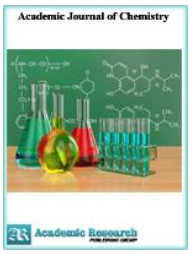

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# Evaluation of Apolipoprotein Profile and Cd4+ T Cell Count in Adult HIV Seropositives in Nauth Nnewi, South Eastern, Nigeria

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**Abstract:** Aim: To determine the Apolipoprotein profile and $CD4^+$ T cell count in adult HIV seropositive participants in Nnamdi Azikiwe University Teaching Hospital Nnewi, South Eastern Nigeria. Methods: Blood samples collected from the 300 randomly recruited participants were used for HIV screening, $CD4^+$ T cell count, Apolipoprotein A-1, A-11, B, C-11, C-111 and Apo E. Standard Laboratory methods were used for the analysis. Results: The results showed that the mean serum Apo A-11, Apo B, Apo C-11 and Apo E levels were significantly higher in symptomatic HIV participants not on antiretroviral therapy (ART) compared with asymptomatic HIV participants at $P< 0.05$, in each case. But the mean serum Apo A-1, Apo C-111 and $CD4^+$ T cell levels were significantly lower in symptomatic HIV participants not on ART compared with asymptomatic HIV participants at $P< 0.05$, in each case. Also, the mean serum Apo A-11, Apo B and Apo E levels were significantly higher in symptomatic HIV participants not on ART compared with HIV seronegative control subjects at $P<0.05$ respectively. However, the mean serum Apo A-1, Apo C-111 and $CD4^+$ T cell levels were significantly lower in symptomatic HIV participants not on ART compared with asymptomatic HIV participants at $P<0.05$ in each case. Again, the mean serum Apo A-11, Apo B, Apo E were significantly higher in asymptomatic HIV participants compared with HIV seronegative control subjects at $P<0.05$ in each case. But the mean serum Apo A-1, Apo C-11 and $CD4^+$ T cell levels were significantly lower in asymptomatic HIV participants compared with HIV seronegative control subjects at $P<0.05$ in each case.

**Keywords:** HIV; Apolipoproteins; $CD4^+$ T cell; participants.

## 1. Introduction

Human Immunodeficiency virus (HIV) is a member of the genus lentivirus, the virus that contains only RNA. HIV does not replicate outside of living host cells, but it enters into host cells, replicates readily in body type of white blood cell called CD4 cell, thereby releasing HIV virons from infected cells, depleting the CD4 cells [1, 2]. The depleted CD4 cells weaken the immune system thereby developing into Acquired Immunodeficiency syndrome (AIDS) [3]. The T cells also known as CD4 cells are essential to the immune response and without them the body cannot fight infections or kill cancerous cells [4].

There are two types of HIV; HIV-1 and HIV-2. Both types may be transmitted by sexual contact, from mother to children; through blood and the (AIDS) produced by them is clinically indistinguishable [5]. However, it seems that HIV-1 is less easily transmitted and the period between initial infection and illness is longer in the case of HIV-2 [6]. Worldwide, the predominant virus is HIV-1 while HIV-2 is more in West Africa [7].

AIDS has been declared a pandemic disease by World Health Organisation, affecting 35million people globally, 2/3 of them from sub-Saharan Africa. 95% of the death is from young youth [8]. Globally South Africa has the largest population with HIV, followed by Asian, Nigeria and India [9].

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HIV infection has been reported to affects the adipocyte function, causing fat redistribution [10] and lipoatrophy [11, 12]. Ezeugwunne, *et al.* [13] observed lower levels of lipid profile in HIV subjects not on ART.

Apolipoproteins are proteins that bind to lipids to form lipoproteins, whose main function is to transport lipids. Apolipoproteins are important in maintaining the structural integrity and solubility of proteins [14]. They also serve as cofactors for enzymes and ligands for cell-surface receptors [15].

In South Eastern region of Nigeria, no study to my knowledge has been done to evaluate the effect of HIV/AIDS on apolipoproteins of people living with the disease. Hence, identification of these biomarkers in them will generate data and information that may be useful for better treatment, management and follow-up of individuals.

# 2. Materials and Methods

## 2.1. Subjects

The study was conducted in Nnamdi Azikiwe University (NAUTH), Nnewi in Anambra state, South East Nigeria. Based on 3.1 % prevalence rate of HIV in Nigeria [16] and using the formular of Naing, *et al.* [17] for sample size calculation, a total of 300 (M=170, F= 130) subjects aged between 17 and 58 (38 ±9) years were recruited by convenient sampling technique from patients that attended HIV clinic, NAUTH Nnewi, South Eastern Nigeria from 2012 to 2014 for this study. The study design was case study. Using the World Health Organization [18], staging for HIV as a guide, the participants were grouped, comprising of 100 symptomatic HIV subjects not on ART (M=64, F=36), 100 asymptomatic HIV subjects (M=57, F=43) and 100 HIV seronegative control subjects (M=49, F=51). Ethical approval was sort and obtained from the NAUTH ethics committee and informed consent was obtained from the participants.

## 2.2. Sample Collection

6 ml of fasting blood samples were collected from all the participants in this study. 2ml of blood samples were collected into EDTA sample tubes for HIV screening and CD4+ T cell count. The remaining 4 ml of blood sample were collected into plain tube and allowed to clot, centrifuged, the serum separated and analyzed for Apo A-1, Apo A-11, Apo B, Apo C-11, Apo C-111 and Apo E levels.

## 2.3. Quality control measures

Quality control sera were run along test in each batch of analysis these were compared with the reference values of the control sera. Standard deviation and coefficient of variation were calculated on them.

## 2.4 Methods of Assaying

### 2.4.1. Determination of Antibodies to HIV-1 and HIV-2 in Human Plasma

**Procedure**

Two different methods were used, namely, Abbott determine TM HIV -1 and HIV-2 kit, which is an in-vitro visually read immunoassay (Abbott Japan Co.Ltd.Tokyo, Japan) and HIV-1 and 2 STAT-PAK Assay kit, which is an Immunochromatographic test for the quantitative detection of antibodies to HIV-1 and HIV-2 in Human plasma (CHEMBIO Diagnostic system, Inc, New York, USA). For the Abbott determine TM HIV -1 and HIV-2 kit, the procedure described by the manufacturer was used for the analysis. Briefly, 50 µl of participant serum samples separated from the corresponding whole blood samples in EDTA were applied to the appropriately labeled sample pad. After 15 minutes but not more than 60 minutes of sample application, the result was read. This method has inherent quality control that validates the results. For the Immunochromatographic method for HIV -1 and HIV-2, the procedure described by the manufacturer was used for the analysis. In brief, 5 ml of participant's plasma was dispensed into the sample well in the appropriately labeled sample pad. Three drops of the buffer supplied by the manufacturer was added into the appropriately labeled sample pad. The results of the test were read at 10 minutes after the addition of the running buffer. This method has inherent quality control and validates the results.

### 2.4.2. Determination of $CD4^+$T cells counts by CyFlows SL-Green

**Procedure**

200 ml EDTA whole blood was collected into PARTEC test tubes (Rohren tube). Then 20 µl of $CD4^+$ T antibody was added into the tube. The contents was mixed and incubated in the dark for15 minutes at room temperature. 800 ml of CD4 buffer was added into the mixture and mixed gently. Then the Partec tube was plugged on the Cyflow counter and the $CD4^+$ T cells were displayed as peaks and interpreted as figures.

### 2.4.3. Quantitative determination of Apolipoprotein A-I in human sera

**Principle**

Turbidimetric test was used for the measurement of apolipoprotein A-I. Anti- Apo A-I antibodies were mixed with samples containing Apo A-I, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo A-I concentration of the patient's sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

## Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 $^{O}$C. 800 µ/l of Reagent R1 (Tris buffer, 20 mmol/l. PEG, pH 8.3, sodium azide 0.95 g/l) was dispensed into a cuvette and 7 µ/l of calibrator was added respectively, mixed and the absorbance ($A_1$) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as $A_1$ sample. Immediately, 200 µ/l of Reagent R2 (Anti-human apolipoprotein A-1 goat- polyclonal antibody, tris buffer, 50 mmol/l, pH 7.5, sodium azide 0.95 g/l) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 2 minutes as $A_2$ for calibrator and sample respectively.

## Calculations

$$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrator}} \times \text{Calibrator concentration} = \text{mg/dl Apo A-I}$$

## 2.4.4. Quantitative determination of Apolipoprotein A-11 in human Sera

## Principle

Turbidimetric test was used for the measurement of apolipoprotein A-11. Anti- Apo A-11 antibodies were mixed with samples containing Apo A-11, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo A-11 concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

## Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain). The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 $^{O}$C. 300 µ/l of Reagent R1 (2-amino-2-hydroxymethyl-1, 3-propanediol buffer, 100 mmol/l, pH 8.5, macrogol) was dispensed into a cuvette and 5 µ/l of calibrator was added respectively, mixed and the absorbance ($A_1$) of calibrator was read at 600 nm. The sample was treated the same way as the calibrator and its absorbance reading as $A_1$ sample. Immediately, 100 µ/l of Reagent R2 (Anti-human apolipoprotein A-11 goat- polyclonal antibody) was dispensed into each of the same cuvette, mixed and was read again at 600 nm after 5 minutes as $A_2$ for calibrator and sample respectively.

## Calculations

$$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrator}} \times \text{Calibrator concentration} = \text{mg/dl Apo A-II}$$

## 2.4.5. Quantitative determination of Apolipoprotein B in human sera

## Principle

Turbidimetric test was used for the measurement of Apolipoprotein B. Anti- Apo B antibodies were mixed with samples containing Apo B, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo B concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

## Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited, Spain).The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 $^{O}$C. 800 µ/l of Reagent R1 (Tris buffer, 20 mmol/L, PEG, pH 8.3, sodium azide, 0.95 g/L) was dispensed into a cuvette and 7 µ/l of calibrator was added respectively, mixed and the absorbance ($A_1$) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as $A_1$ sample. Immediately, 200 µ/l of Reagent R2 (Anti-human apolipoprotein B goat- polyclonal antibody, Tris buffer, 50 mmol/L, pH 7.5, sodium azide, 0.95 g/l ) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 2 minutes as $A_2$ for calibrator and sample respectively.

## Calculations

$$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrato}} \times \text{Calibrator concentration} = \text{mg/dl Apo B}$$

## 2.4.6. Quantitative determination of ApolipoproteinC-11 in human Sera

## Principle

Turbidimetric test was used for the measurement of apolipoprotein C-11. Anti- Apo C-11antibodies were mixed with samples containing Apo C-11, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo C-11 concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

## Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited,Spain).The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 $^{O}$C. 750 µ/l of Reagent R1 (Tris buffer, 100 mmol/L, PEG 4000, pH 8.5, sodium azide, 0.95 g/L) was dispensed into a cuvette and 25 µ/l of calibrator was added respectively, mixed and the absorbance ($A_1$) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as $A_1$ sample. Immediately, 250 µ/L of Reagent R2 (Anti-human apolipoprotein C-11 goat- polyclonal antibody, Tris buffer, 100 mmol/L, pH 7.2, sodium azide, 0.95 g/L) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 5 minutes as $A_2$ for calibrator and sample respectively.

**Calculations**

$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrator}}$ x Calibrator concentration = mg/dl Apo C-11

## 2.4.7. Quantitative determination of ApolipoproteinC-111 in Human Sera

## Principle

Turbidimetric test was used for the measurement of apolipoprotein C-111. Anti- Apo C-111 antibodies were mixed with samples containing Apo C-111, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo C-111 concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

## Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited,Spain).The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 $^{O}$C. 750 µ/lLof Reagent R1 (Tris buffer, 100 mmol/L, PEG 4000, pH 8.5, sodium azide, 0.95 g/L) was dispensed into a cuvette and 20 µ/L of calibrator was added respectively, mixed and the absorbance ($A_1$) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as $A_1$ sample. Immediately, 250 µ/l of Reagent R2 (Anti-human apolipoprotein C-111 goat- polyclonal antibody, Tris buffer, 100 mmol/L, pH 7.2, sodium azide, 0.95 g/L ) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 5 minutes as $A_2$ for calibrator and sample respectively.

## Calculations

$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrator}}$ x Calibrator concentration = mg/dl Apo C-111

## 2.4.8. Quantitative determination of Apolipoprotein E in human sera

## Principle

Turbidimetric test was used for the measurement of apolipoprotein E. Anti- Apo E antibodies were mixed with samples containing Apo E, form insoluble complexes. These complexes cause an absorbance change, dependent upon the Apo E concentration of the patient sample, which was quantitated by dividing the absorbance of sample by that of the calibrator and multiply by the concentration of the calibrator.

## Procedure

The procedure was as described by the manufacturer (Spinreact laboratories limited,Spain).The spectrophotometer was zeroed with distilled water. The reacting mixture was carried out at 37 $^{O}$C. 750 µ/L of Reagent R1 (Tris buffer, 100 mmol/L, PEG 4000, pH 8.5, sodium azide, 0.95 g/l) as dispensed into a cuvette and 25 µ/L of calibrator was added respectively, mixed and the absorbance ($A_1$) of calibrator was read at 340 nm. The sample was treated the same way as the calibrator and its absorbance reading as $A_1$ sample. Immediately, 250 µ/l of Reagent R2 (Anti-human apolipoprotein E goat- polyclonal antibody, Tris buffer, 100 mmol/L, pH 7.2, sodium azide, 0.95 g/l) was dispensed into each of the same cuvette, mixed and was read again at 340 nm after 5 minutes as $A_2$ for calibrator and sample respectively.

## Calculations

$\frac{(A_2\text{-}A_1)\ \text{sample}}{(A_2\text{-}A_1)\ \text{calibrator}}$ x Calibrator concentration = mg/dl Apo E

## 2.5. Data analysis

The result of the analysis was statistically analyzed. Students't-test and analysis of variance (ANOVA) were used to compare means. The analyses were performed with the use of Statistical *Package for Social Sciences* (SPSS) statistical software package, version 16.0. P <0.05 is considered statistically significant.

# 3. Results

The result of the analysis of variance showed that the mean serum apolipoprotein A-1, Apo A-11, Apo B, Apo C-11, Apo C-111, Apo E (g/l) levels and CD4+ T cell counts were significant different amongst the groups at $P < 0.05$ ($F = 291.30$; 173.89; 83.64; 7.14; 20.76, 87.65 and 216.22) respectively.

Between group comparison showed that the mean serum Apo A-11, Apo B, Apo C-11 and Apo E levels were significantly higher in symptomatic HIV infected subjects not on ART compared with asymptomatic HIV infected subjects at $p<0.05$ respectively. However, the mean serum Apo A-1**,** Apo C-111levels and CD4+ T cell counts were significantly lower in symptomatic HIV infected subjects not on ART compared with asymptomatic HIV infected subjects at $P>0.05$ respectively.

Similarly, the mean serum Apo A-11, Apo B and Apo E levels were significantly higher in symptomatic HIV infected subjects not on ART compared with HIV seronagative control subjects at $p<0.05$ respectively. But, the mean serum Apo A-1, Apo C-111 levels and CD4+ T cell counts were significantly lower in symptomatic HIV infected subjects not on ART compared with HIV seronegative control subjects at $p<0.05$ respectively. However, the mean serum Apo C-11 was the same at $P>0.05$.

Again, the mean serum Apo A-11, Apo B and Apo E levels were significantly higher in asymptomatic HIV infected subjects compared with HIV seronegative control subjects ($p<0.05$, in each case). But, the mean serum Apo A-1 and Apo C-11 levels and CD4+ T cell counts were significantly lower in asymptomatic HIV infected subjects compared with HIV seronegative control subjects ($p<0.05$, in each case). However, the mean serum Apo C-111 was the same at $P>0.05$.

**Table-1.** Comparison of mean ± SD serum levels of Apolipoproteinprofile in symptomatic HIV infected subjects not on ART (A), in asymptomatic HIV subjects (B) and control group (C).

| Group | CD4 (/Ml) | Apo- A-1(g/L) | Apo A-1 1(g/L) | Apo-B(g/L) | Apo C-11(g/L) | Apo C-111 (g/L) | Apo E (g/L) |
|---|---|---|---|---|---|---|---|
| A(n=100) | 374.78 ± 121.59 | 0.35 ± 0.20 | 0.55 ± 0.34 | 2.84 ± 0.89 | 0.05 ± 0.02 | 0.01 ± 0.01 | 0.24 ± 0.11 |
| B(n=100) | 437.20 ±129.75 | 0.60 ± 0.23 | 0.44 ± 0.13 | 1.50 ± 0.02 | 0.04 ± 0.01 | 0.03 ± 0.01 | 0.11 ± 0.07 |
| C(n=100) | 940.64 ± 148.85 | 1.26 ± 0.06 | 0.24 ± 0.09 | 0.68 ± 0.29 | 0.05 ± 0.02 | 0.03 ± 0.02 | 0.05 ± 0.01 |
| F(p)-value | 216.22 (.000) | 474.03 (.000) | 52.13 (.000) | 128.50 (.000) | 9.80 (.000) | 27.70 (.000) | 131.12 (.000) |
| A v B | <0.05 | <0.05 | <0.05 | <0.05 | <0.05 | <0.05 | <0.05 |
| A v C | <0.05 | <0.05 | <0.05 | <0.05 | >0.05 | <0.05 | <0.05 |
| B v C | <0.05 | <0.05 | <0.05 | <0.05 | <0.05 | >0.05 | <0.05 |

Key: F (p) value = mean ± SD of parameter compared among groups A, B, C and D (using ANOVA).
A V B p value = mean ± SD of parameter compared between group A and B (using t-test).
A V C p value = mean ± SD of parameter compared between group A and C (using t-test).
B V C p value = mean ± SD of parameter compared between group B and C (using t-test).

# 4. Discussion

The present study showed that the serum concentrations of Apo A-1, Apo A-2, Apo B, Apo C-2, Apo C-3 and Apo E were significantly different in HIV positive individuals. The concentration of Apo A-1 was significantly lowered in HIV positive individuals. The reduction was more marked in symptomatic HIV subjects not on ART. The connotation of this finding is that the reduction of serum concentration of Apo A-1 may compromise the structural composition of high density lipoprotein (HDL), since it is the major apolipoprotein in HDL [15, 19]. Also, the activity of lecithin cholesterol acyl transferase (LCAT) may be affected. LCAT functions in the removal of excess cholesterol from HDL and transported it to the liver for excretion. Again, for LCAT to function properly, it needed Apo A-1 as a cofactor [20]. Apo A1 is often a biomarker for cardiovascular diseases [21, 22]. It also plays a role in protection of Alzheimer's disease and with Apo E have been found to interact to modify triglyceride levels in coronary heart disease in patients [23].

The study also showed that the serum level of Apo A-11 was significantly increased in symptomatic HIV individuals not on ART. The implication of this finding is that it may cause the deposition of abnormal proportion of Apo A-11 in HDL, thereby affecting the normal functioning of HDL in removing "bad cholesterol" from the system. This finding may implies that HIV infection plays a role in affecting the normal structural composition of HDL which may lead to the abnormal lipid metabolism in HIV individuals. Therefore, the significant raise in serum Apo A-11 status of HIV infected participants may be a determinant in ascertaining factors that predisposes severity of the disease in HIV.

Also, the serum level of Apo B was significantly higher in symptomatic HIV individuals not on ART. The implication of this finding is that an excess of serum Apo B has been found to be a better predictor of cardiovascular disease [24, 25]. An excess of Apo B particles is a main trigger in the atherogenic process [26]. But a significant

lower level of Apo C-111 and a higher level of Apo C-11were observed in symptomatic HIV participants not on ART. Reports have it that Apo C-111 inhibits lipolysis of TG-rich lipoproteins [27, 28]. Elevated level of serum Apo C111 has been linked with coronary heart diseases [29]. Serum levels of Apo –C1 and Apo C111 are reduced in patients with stomach cancer and may have a role in the formulation of a diagnostic score for stomach cancer patients [30].

In this study there was an exaggerated higher level in serum Apo E in symptomatic HIV participants not on ART. Also, elevated Apo E was found in HIV positive individuals [31]. Apo E is an essential apolipoprotein for the normal catabolism of TG-rich lipoprotein constituents [32]. The increased level of Apo E may explained the wasting disease found in the individuals and may be due to the increased activity of TG-rich lipoprotein catabolism in their body which may be as a result of the effect of the HIV infection on them. There is evidence that Apo E protects against atherogenesis [33].

Apo E has been reported to influence psoriasis risk-an autoimmune disease with chronic recurring reddish patches on the skin. Psoriasis causes abnormal lipid metabolism [34, 35]. Studies suggested that an increased Apo E level was found to increased hepatic synthesis of VLDL and decreased clearance of triglycerides with lipid abnormalities [36].

The CD4 cell was significantly reduced in HIV positives. The depleted values of CD4 observed in symptomatic HIV positives may be link to rate of CD4+ T cell depletion with HIV disease progression [37]. The CD4 cell value was markedly reduced in HIV subjects not on antiretroviral therapy. Ezeugwunne, *et al.* [38] observed that HIV and malaria co-infection were capable of lowering the value of CD4 counts in HIV individuals.

## 5. Conclusion

HIV infection was found to cause abnormal apolipoproteinemia with respect to Apo A-1, Apo-A-11, Apo B, ApoE and reduced value of CD4 cells.

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## Conflict of Interest

There is no conflict of interest whatever with anyone or group of persons. The studied was sponsored by Tertiary Educaion trust Fund (TETFUND), Nigeria

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