

# High viremia and Poor Immunity are Potential Surrogates of Anti-toxoplasmic Immunoglobulin G Quantification among HIV-Infected Individuals: A Cross-sectional Study in Yaoundé, Cameroon

Aude Christelle Ka'e (✉ [kae.audechristelle@gmail.com](mailto:kae.audechristelle@gmail.com))

CIRCB <https://orcid.org/0000-0002-2381-4299>

**Samuel Martin Sosso**

CIRCB

**Joseph Fokam**

CIRCB; University of Yaoundé I; School of Health Sciences, Catholic University of Central Africa, Yaounde Cameroon

**Rachel Kamgaing Simo**

CIRCB

**Sara Riwom Essama**

University of Yaounde I

**Alex Durand Nka**

CIRCB; university of Tor Vergata di roma; Evangelic University of Cameroon

**Bouba Yagai**

CIRCB; University of Tor Vergata di Roma

**Cédric Joël Ninsu Dzukou**

University of Douala, Cameroon

**Michel Carlos Tommo Tchouaket**

CIRCB; School of Health Sciences, Catholic University of Central Africa Yaounde, Cameroon

**Collins Chenwi**

CIRCB; University of Yaounde I

**Aissatou Abba**

CIRCB; School of Health Sciences, Catholic University of Central Africa, Yaounde Cameroon

**Nadine Fainguem**

CIRCB; School of Health Sciences, Catholic University of Central Africa, Yaounde Cameroon

**Marie Krystel Nnomo Zam**

CIRCB

**Junie Flore Yimga**

CIRCB

**Vittorio Colizzi**

University Tor Vergata di Roma; Evangelic University of Cameroon

**Alexis Ndjolo**

CIRCB; University of Yaounde I

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## Research article

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# Abstract

**Background:** Toxoplasmosis remains a neglected common opportunistic infection in immunocompromised individuals, who are mainly people living with HIV (PLWHIV) in whom reactivation of toxoplasmosis may occur with advanced HIV conditions in resource-limited settings (RLS). Our objective was to evaluate the correlation between the anti-toxoplasmic IgG (Tg-IgG) concentration and the immuno-virological status of PLWHIV.

**Methods :** A prospective and cross-sectional study was conducted among PLWHIV aged >18 years from February to November 2018 at the Chantal BIYA international Reference Centre. Blood samples were collected from eligible consenting PLWHIV; Tg-IgG level was assessed by quantitative ELISA, CD4-T lymphocytes counts were measured by flow cytometry and HIV-1 plasma viral load (PVL) measurement by real-time-PCR. Data were analysed using Excel and Graph Pad softwares; with  $p < 0.05$  considered statistically significant.

**Results :** A total of 100 PLWHIV were enrolled: 56% seropositive for IgG anti- *Toxoplasma gondii*, 33% seronegative and 11% indeterminate results. According to viremia, 100% (19/19) of those with  $PVL > 1000$  copies/mL were seropositive to Tg-IgG versus 52.85% (37/70) of those with  $PVL < 1000$  copies/mL (median [IQR] IgG concentration 152.78 [139.24-444.43] versus 34.44 [13.04-36.47] IU/mL, respectively);  $p < 0.0001$ . According to CD4, 100% (11/11) of those with  $T-CD4 < 200$  cells/ $\mu$ L were seropositive to Tg-IgG versus 57.69% (45/78) of those with  $T-CD4 > 200$  cells/ $\mu$ L (median IgG [IQR] 432.92 [145.06-450.47] versus 35.01 [15.01-38.01] IU/mL, respectively);  $p < 0.0001$ . Interestingly, there were moderate-positive and strong-negative correlations respectively with HIV-1 PVL ( $r = 0.54$ ;  $p < 0.0001$ ) and T-CD4 ( $r = -0.70$ ;  $p < 0.0001$ ) as compared to Tg-IgG concentration. After adjusting for age, gender, immune status and PVL in logistic regression, only poor immune status ( $T-CD4 < 200$  cells/ $\mu$ L) was independently associated to Tg-IgG seropositivity ( $p = 0.0004$ ).

**Conclusion :** In a typical RLS like Cameroon, about half of PLWHIV might be seropositive to Tg-IgG. Of relevance, decreasing immunity appears with risk of increasing IgG anti- *T gondii* concentration, which suggests a relapse of toxoplasmosis. Thus, in the context of immunodeficiency, routine quantification of Tg-IgG would alleviate the programmatic burden of this opportunistic infection in RLS with generalized HIV epidemics.

## Background

The African continent is the most affected by HIV as of the 37.9 million people living with HIV (PLWHIV) worldwide in 2018, up to 25.6 million (~ 70%) live in Africa. The Joint United Nations Program on HIV and AIDS (UNAIDS) estimates that 770,000 people worldwide have died of AIDS-related causes, including 470,000 in Sub-Saharan Africa (SSA). [1]. Events of mortality in the population of PLWHIV are largely driven by opportunistic infections in resource-limited settings (RLS) [2]. Opportunistic infections

predominating among PLWHIV are tuberculosis, cryptococcal meningitis, pneumocystis carinii pneumonia, and toxoplasmosis [3].

Toxoplasmosis is still a neglected tropical parasitic infection even 110 years after its discovery [4]. It is a cosmopolitan parasitic infection caused by an intracellular protozoan *Toxoplasma gondii* (*T gondii*) that is widespread throughout the world; with one third of the world population having been in contact with the parasite [3, 5]. Of note, ingestion of undercooked meat containing the encysted stage of the parasite (tissue cysts) or food and water contaminated with cat feces containing oocysts is the usually mode of infestation [6]. In adding, people may get infected by congenital infection, blood transfusion or organ transplantation [7].

A systematic review made in 2017 reported 13,138,600 cases of *T gondii* co-infection in HIV-infected people, with 87.1% being concentrated in SSA (11,449,500 cases, 95% CI 8,236,500 – 14,662,500) [8]. *T gondii* is able to subsist and persist in immunocompetent intermediate hosts, even lifelong (latent infection) [9]. In the vast majority of cases, toxoplasmosis is harmless, generally responsible for a common transient infection in 80% of the population [6], but can represent a significant threat through substantial neurologic damage among PLWHIV who are immunocompromised [6, 10]. It was reported that disease occurs following reactivation of latent infection because of progressive loss of cellular immunity, which can occur in the setting of advanced HIV infection, which could contribute to serious neurological or neurocognitive associated diseases [11, 12]. This suggests that, in addition to the standard monitoring of HIV-infected patients with regular measurements of HIV viral load and CD4 T lymphocytes, it might be relevant to set-up strategies for periodic testing or an approach for patient-centered testing of opportunistic infections like toxoplasmosis in RLS experiencing a high burden of HIV infection, and especially in SSA [13].

In Cameroon, some evidence have been reported on HIV/toxoplasmosis co-infection [14–17]. Of note, these previous reports showed emphasis on mortality trends [14], IgG and IgM testing [15], toxoplasmosis in hospitalised patients [16], and neurological disturbances associated with HIV/AIDS [17]. However, data on anti-toxoplasma *gondii* quantification, as well as its possible correlation with standard biomarkers of HIV infection (viral load and CD4), have not yet been ascertained. Thus, generating evidence on correlations between IgG antitoxoplasmic serology, viral load of HIV-1 and CD4 T lymphocytes count, would guide on the occurrence of reactivation of toxoplasmosis following the viral replication/control and immune status of patients in SSA countries.

Our study objective was to evaluate the correlation between the toxoplasmic IgG concentration and the immuno-virological status of PLWHIV.

## Methods

### Study design and setting

A cross-sectional and prospective study was carried out from February to November 2018 among PLWHIV in Yaoundé, monitored at the “Chantal BIYA” International Reference Center for research on HIV/AIDS prevention and management (CIRCB), in Yaounde, Cameroon. CIRCB is a government institute of the Ministry of Public Health, in charge of research and reference clinical monitoring of HIV-infected patients, with participation in external quality assurance programs for HIV and hepatitis screening/diagnosis, viral load measurements, CD4 lymphocytes count, immunological analysis, besides biochemistry and haematological analysis ([http://circb.cm/btc\\_circb/web/](http://circb.cm/btc_circb/web/)).

## **Enrolment procedure and eligibiity criteria**

A standard questionnaire was adminisitered to 114 PLWHIV, assisted by trained interviewers, for the collection of basic sociodemographic data, clinical data, history of HIV infection and exposure to antiretroviral therapy. Eligibility was then assesed and participants were enrolled based on the following criteria: (a) HIV-positive confirmed using the national testing algorithm in Cameroon, (b) documented clinical report with evidence of no ongoing antiparasitic therapy, and written informed consent. Participant who were co-infected with either Hepatitis B or C infection were then excluded.

## **Sample collection**

Peripheral blood was collected by venipuncture in two Ethylene Diamine Tetra Acetic acid (EDTA) containing tubes of 4 mL; one for the measurement of CD4 T lymphocytes through whole blood and another for analysis of HIV-1 viral load through plasma. Whole blood was also collected in one dry tube of 4 mL for serological testing.

## **Serological tests**

Serum obtained after centrifugation from whole blood in the dry tube was immediately used for carrying out the lateral flow immunochromatography assays, as per the manufacturers’ instructions ([www.alere.com/Ref7D2343SET](http://www.alere.com/Ref7D2343SET)): HIV antibody testing following the serial algorithm as per national guidelines; HBsAg for the detecting of hepatitis B (Hepatitis B surface Antigen Rapid Best Diagnosis Test Kit): and anti-HCV Ab for the detection of hepatitis C antibody (Hepatitis C Antibodies Rapid Best Diagnosis Test Kit).

## **HIV-1 viremia measurement**

Centrifugation of one EDTA tube for each patient was performed to obtain plasma, which was then separated in two 700- $\mu$ L aliquots and stored at -20 °C, then leaved at 8 °C before extraction of viral RNA and amplification for viral load by Real Time PCR on the abbott m2000RT platform as per the manufacturer’s instructions.

([www.abbottmolecular.com/products/infectious-diseases/realtime-pcr/hiv-1-assay](http://www.abbottmolecular.com/products/infectious-diseases/realtime-pcr/hiv-1-assay)).

## **CD4 T lymphocytes measurements**

Whole blood from one EDTA tube was directly used to perform CD4 T lymphocyte count using flow cytometry on Cyflow cytometer according to the manufacturer's instructions.

(CD4 easy count kit (REF: 05-8405))

## Toxoplasmic IgG serology

Serum obtained after centrifugation of blood in the dry tube was immediately conserved at -20 °C, then at ambient temperature before measurement of antitoxoplasmic IgG concentration by solid phase ELISA according to the manufacturer's instructions. Results were reported as seropositive if IgG > 35UI/mL, seronegative if IgG < 35UI/mL, and indeterminate if IgG comprised between 30–35 UI/mL.

(Toxoplasma gondii IgG ELISA-IBL international (RE57101).pdf).

## Statistical analysis

Data were recorded in an Excel spreadsheet and verified by double entry for data cleaning. Data analysis, except correlations, were done with exclusion of indeterminate results of toxoplasmic serology using the software Graph Pad prism version 6.0. The Spearman correlation coefficient was used to determine the existing relationships between the quantitative variables. A logistic regression analysis was done to evaluate the association between toxoplasmic IgG serology (excluding indeterminate cases) and sex, age, PVL and immune status (CD4). The significance threshold for statistical tests was set at 0.05.

## Results

### Results

#### *Characteristics of the study population*

Among 114 people enrolled, 100 were eligible and 14 were excluded accordingly. Of the 14 cases excluded, five had a positive hepatitis C serology and the remaining nine were infected with hepatitis B. Our final study participants consisted of 39 (39/100) men and 61 (61/100) women, giving a ratio F/M of 3/2. Participants were ranged from 19 to 78 years with a median [IQR] age of 44 [36-52] years.

#### *Anti-toxoplasmic IgG level in PLWHIV*

Out of 100 samples from study participants, 33% were seronegative for toxoplasmosis (i.e. IgG < 30 IU/mL), 11% have indeterminate results (i.e. 30 < IgG < 35 IU/mL) and the positivity rate of anti-toxoplasmic IgG was 56% (i.e. IgG > 35 IU/mL). The median [IQR] IgG of the study population was 35.09 [18.86-40.12] IU/ml and ranged from 1.04 to 580.05 IU/ml. As presented in Table 1, there was no significant association between gender and antitoxoplasmic IgG seropositivity (p=0.70); however, 41.08% (23/56) of Toxoplasmic IgG-positive individuals were men versus 58.92% (33/56) for women (median [IQR] IgG concentration of 36.00 [21.06-40.96] versus 35.07 [18.9-39.64] IU/mL, respectively).

### ***Correlation between CD4 T lymphocytes count and anti-toxoplasmic IgG level***

All (100% [11/11]) severely immunocompromised individuals (T-CD4<200 cells/ $\mu$ L) were seropositive for toxoplasmosis; the median IgG [IQR] in this subset of individuals was 432.92 [145.06-450.47] IU/mL (OR: 57.76; p<0.0001). Furthermore, 35.7% (20/56) of people seropositive to anti-toxoplasmic IgG were in advanced immunodeficiency (T-CD4 comprise between 200 and 300 cells/ $\mu$ L) with a median IgG [IQR] of 37.98 [35.92-40.32] IU/mL (OR: 84.6; p<0.0001) versus 23.2% (13/56) of people with mild immunodeficiency (T-CD4 comprise between 300 and 500 cells/ $\mu$ L) with a median IgG [IQR] of 36.78 [34.94-40.14] IU/mL (OR: 0.57; p=0.57). In addition, 21.4% (12/56) of antitoxoplasmic IgG-positive individuals were without immunodeficiency (T-CD4>500 cells/ $\mu$ L) with a median IgG [IQR] of 19.01 [10.75-34.91] IU/ml (OR: 0.07 as reference to all immunocompromised patients; p<0.0001). See table 2 for detailed results.

As shown in figure 1, we observed a strong, negative and significant correlation between CD4 T lymphocytes level and the rate of antitoxoplasmic IgG ( $r = -0.70$ ;  $P < 0.0001$ ).

### ***Correlation between plasma viral load of HIV-1 and anti-toxoplasmic IgG level***

All (100% [19/19]) patients with PVL>1000 copies/mL were seropositive to anti-toxoplasmic IgG with median IgG [IQR]: 152.78 [139.24-444.43] IU/mL (OR: 152.0; p<0.0001). Anti-toxoplasmic IgG-positive participants with low HIV replication (51-999 copies/mL) represented 16.1% (9/56) with a median IgG [IQR] of 34.44 [13.06-36.29] IU/mL (OR: 43.8; p<0.0001) versus 50% (28/56) of those having an undetectable PVL (<50 copies/mL), with a median IgG [IQR] of 32.65 [13.04-37.70] IU/mL (OR: 3.9; p<0.0001). Table 2 provides additional information on the distribution of toxoplasmic IgG serology and HIV-1 viremia among the study population. A look on figure 2 shows that, we found a moderate, positive and significant correlation between antitoxoplasmic IgG level and HIV-1 viral load ( $r = 0.58$ ;  $P < 0.0001$ ).

### ***Antiretroviral therapy (ART) status and anti-toxoplasmic IgG concentration***

However, among ART experienced individuals, 53.26% (49/92) were seropositive for toxoplasmosis with a median IgG [IQR] concentration of 36.53 [15.50-39.47] IU/mL. According to ART regimens, 52.87% (46/87) of participants receiving a first-line ART were positive to toxoplasmosis with a median [IQR] concentration of 35.05[13.55-39.18] IU/mL; 3 out of 5 (~60%) receiving a second-line ART were seropositive to toxoplasmosis with a median [IQR] concentration of 38.01[34.81-40.95] IU/mL. All (4/4) people without ART were seropositive to toxoplasmosis with a median [IQR] concentration of 170.05[138.98-301.88] IU/mL.

### ***Logistic regression multivariate analysis***

After adjusting for gender, age, PVL and immune status to toxoplasmic IgG serology, with exclusion of cases with indeterminate results, only immune status was independently associated with varying anti-toxoplasmic IgG concentration ( $p=0.0004$ ), as shown in table 3.

## **Discussion**

With the goal to improve on the prevention and management strategy of toxoplasmosis within our settings, we sought to investigate the relatness between toxoplasmic IgG serology and the immuno-virological status of PLWHIV.

Socio-demographic data showed that the sex ratio of the studied population was in favor of women (61%), similar to previous reports, as women are known to have a higher risk of HIV infection and other genital infections [18]. Our results are consistent with the studies of Kouanfack et al in 2010 and Essomba et al in 2015 carried out in Cameroon, which obtained a female predominance of 70.6% and 66.3% respectively [19, 20]. Female predominance may also be explained by easier access to female screening during antenatal care, as opposed to men who have low adherence to voluntary screening [21].

The median age [IQR] of our study population was 44 [36–52] years, owing to the increasing rate of survival with the advent of ART. This finding is similar to that obtained in Cameroon by Essomba et al. in 2015 (i.e. 43 years) [19].

Regarding analysis on the seroprevalence of anti-toxoplasmic IgG, slightly above half of participants were positive to IgG toxoplasmic serology with a median IgG [IQR] of 35.09 [18.86–40.12] IU/ml. This IgG prevalence is lower than that obtained by Faustina and collaborators in southern Ghana (77.80% among PLWHIV) [22]. Inversely, our finding is higher than those reported by Ngoben and Samie in South Africa in 2017 (38% among PLWHIV) [23]. Thus, this clearly underscores the varying burden of toxoplasmosis across different geographical settings and the need of regular surveillance by settings [24]. As the prevalence of *T gondii* depends on the geographical conditions, socio-economic status and the local population food habits [6, 25], discordant prevalence may likely be attributable to the difference in geographical situation and eating habits that would exist between Cameroon and the other SSA countries [20–25].

All people with PVL > 1000 copies/mL were positive to anti-toxoplasmic IgG. Additionally, the correlation between the anti-toxoplasmic IgG concentration and viral load was 0.54 ( $P < 0.001$ ) and indicates that the association between the two variables was moderate and positively correlated. This shows that the increase in HIV-1 plasma viral load would be parallel to the reactivation of *T gondii* and can be explain by the high viral replication, which goes with impaired immune response and re-emerging opportunistic pathogens like *T gondii*. [10].

All people experiencing severe immunodeficiency (CD4-T < 200 cells/ $\mu$ L) were seropositive to toxoplasmosis. In adding, the strong inverse correlation between the anti-toxoplasmic IgG concentration and the CD4 level ( $r = -0.70$ ) shows that reactivation of *T gondii* occurs with the decrease of CD4 level [11]. This could be explained by the fact that during HIV replication, several CD4 cells are infected, leading to lysis of these infected cells through cytotoxic lymphocytes activities, and release of bradyzoites specific to *T gondii* in carriers living with HIV [25]. During their invasion, three successive waves of proteins are secreted from parasite organelles (micronemes, dense granules, and rhoptries) into the host cell. The release of these proteins will be tailed by an increase of IgG levels through the differentiation of memory B-lymphocytes into plasmocytes. This connection between toxoplasmic serology and CD4 T lymphocytes shows that the risk of relapse of a previously latent infection increases with the reduction in the host immune function [6].

A multivariate analysis by adjusting for sex, age, PVL and immune status to toxoplasmic IgG serology, only immune response was independently associated with seropositivity to toxoplasmosis. This result justifies the major implication and the harmful effect of immunodeficiency as an independent risk-factor of toxoplasmosis recrudescence during co-infection HIV-*T gondii* [27].

## Conclusion

In this SSA country with a generalised HIV epidemics, about half of PLWHIV might be seropositive to Tg-IgG. Of relevance, decreasing immunity appears with risk of increasing IgG anti-*T gondii* concentration, which suggests a relapse of toxoplasmosis. Thus, in the context of immunodeficiency, routine quantification of Tg-IgG would alleviate the programmatic burden of this opportunistic infection in RLS with generalized HIV epidemics. Extending such surveys for patients receiving second-line ART would provide further insights.

## Limitations Of Study

The main limitation of this study was the non-performance of a follow-up IgG assay after a few weeks to monitor the evolution. Other limitations include the lack of IgM quantification and the limited sample size of ART-naïve individuals (as per the ongoing test-and treat strategy). However, our findings could greatly reflect the population of PLWHIV currently receiving ART in RLS sharing similar characteristics with Cameroon.

## Abbreviations

AIDS  
Acquired Immune Deficiency Syndrome  
CD4  
Cluster of Differentiation 4  
CD8

Cluster of Differentiation 8

CIRCB

Chantal BIYA International Reference Center for research on HIV/AIDS prevention and management

EDTA

Ethylene Diamine Tetra Acetic acid

ELISA

Enzyme Linked Immunosorbent Assay

HIV

Human Immune Virus

IgG

Immunoglobulin Gamma

IgM

Immunoglobulin M $\hat{u}$

IL-2

Interleukin 2

PLWHIV

People Living With HIV

PVL

Plasma Viral Load

RLS

Resource-Limited Settings

RT-PCR

Reverse Transcriptional Polymerase Chain Reaction

SSA

Sub-Saharan Africa

UNAIDS

The Joint United Nations Programme on HIV and AIDS

VL

Viral Load

## Declarations

### *Ethics approval and consent to participate*

All patients provided written informed consent; National Ethic Committee for Human Health Research approved the study (N $^{\circ}$ 2018/05/1033/CE/CNERSH/SP) and we obtained an administrative authorization from CIRCB where the study was conducted.

### *Consent for publication:*

Not applicable.

### ***Availability of data and materials***

The dataset is available from the corresponding author.

### ***Competing interests***

The authors declared that this study is without conflicts of interests.

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The financial and material resources such as reagents, laboratory consumables and access to the various platforms for carrying out the biological analyzes of the samples was provided by CIRCB where analysis of samples and data has been heavily involved.

### ***Author's contributions:***

ACK, CJND, SMS, AN, SR, JF, BY, RK, MCTT, and ADN conceived the study. ACK, CJND, ADN and MCTT have collected data. ACK, ADN, CJND, MCTT, AA, JFY, MKNZ have realized all biological analyses. ACK, ADN, AA have done statistical analysis. ACK, SMS, JF, CJND and MCTT have initiated the manuscript. ACK, JF, SMS, RK, SR, CJND, MCTT, NF, SRD, AA, CC, VC and AN revised the paper. All the authors have approved the final manuscript.

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### ***Author's information***

1. Chantal BIYA International Reference Center for research on HIV/AIDS prevention and management (CIRCB), Yaounde, Cameroon.
2. University of Yaounde I, Yaounde, Cameroon

3. School of Health Sciences, Catholic University of Central Africa, Yaounde, Cameroon
4. University of Douala, Douala, Cameroon
5. University of Tor Vergata, Rome, Italy
6. Evangelic University of Cameroon, Bandjoun, Cameroon

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## Tables

Due to technical limitations, Tables 1, 2 and 3 are only available as a download in the supplemental files section

## Figures

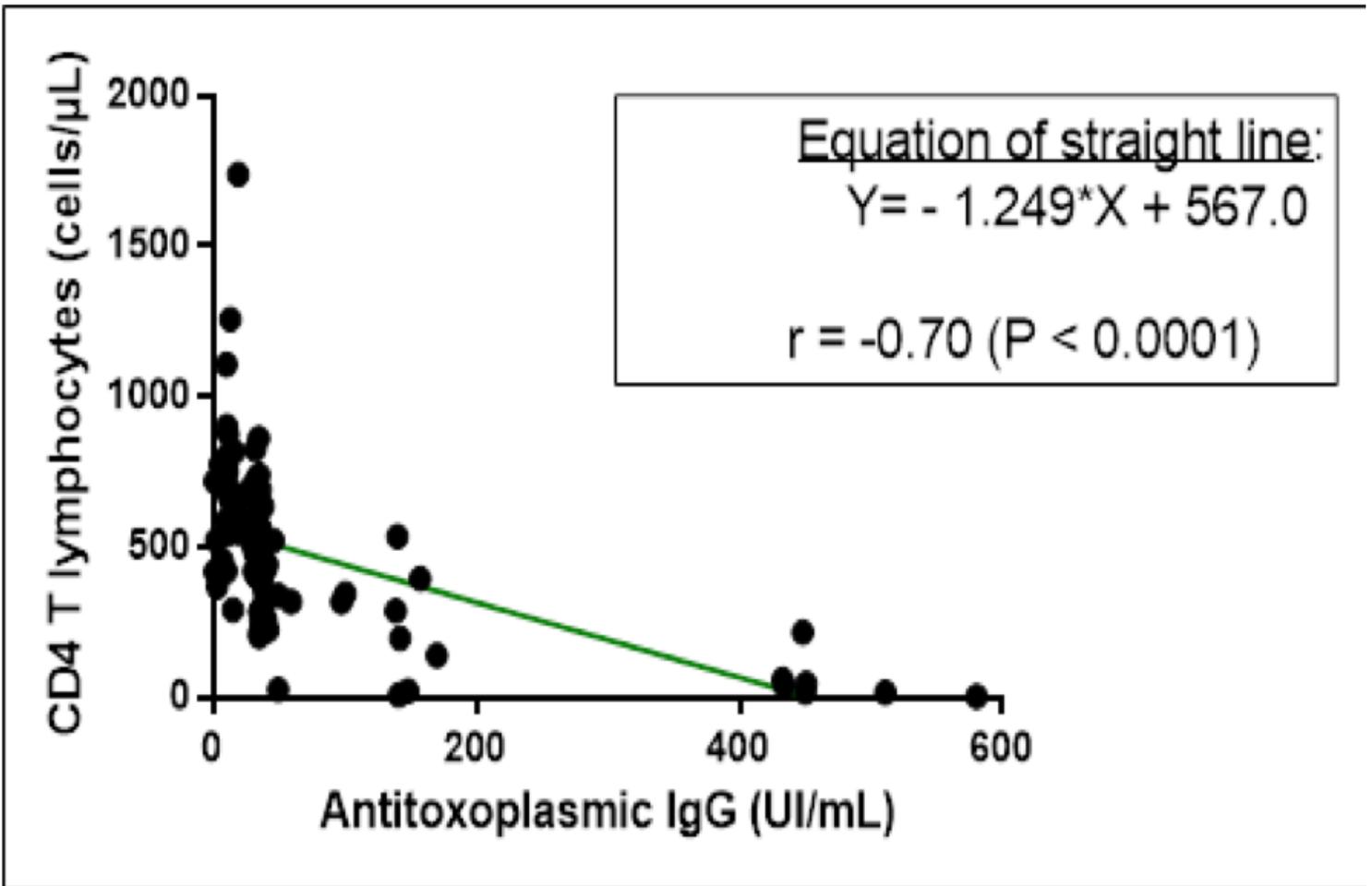


Figure 1

Correlation straight line between anti-toxoplasmic IgG and CD4 T lymphocytes. Ig: Immunoglobulin; CD: clusters of differentiation.

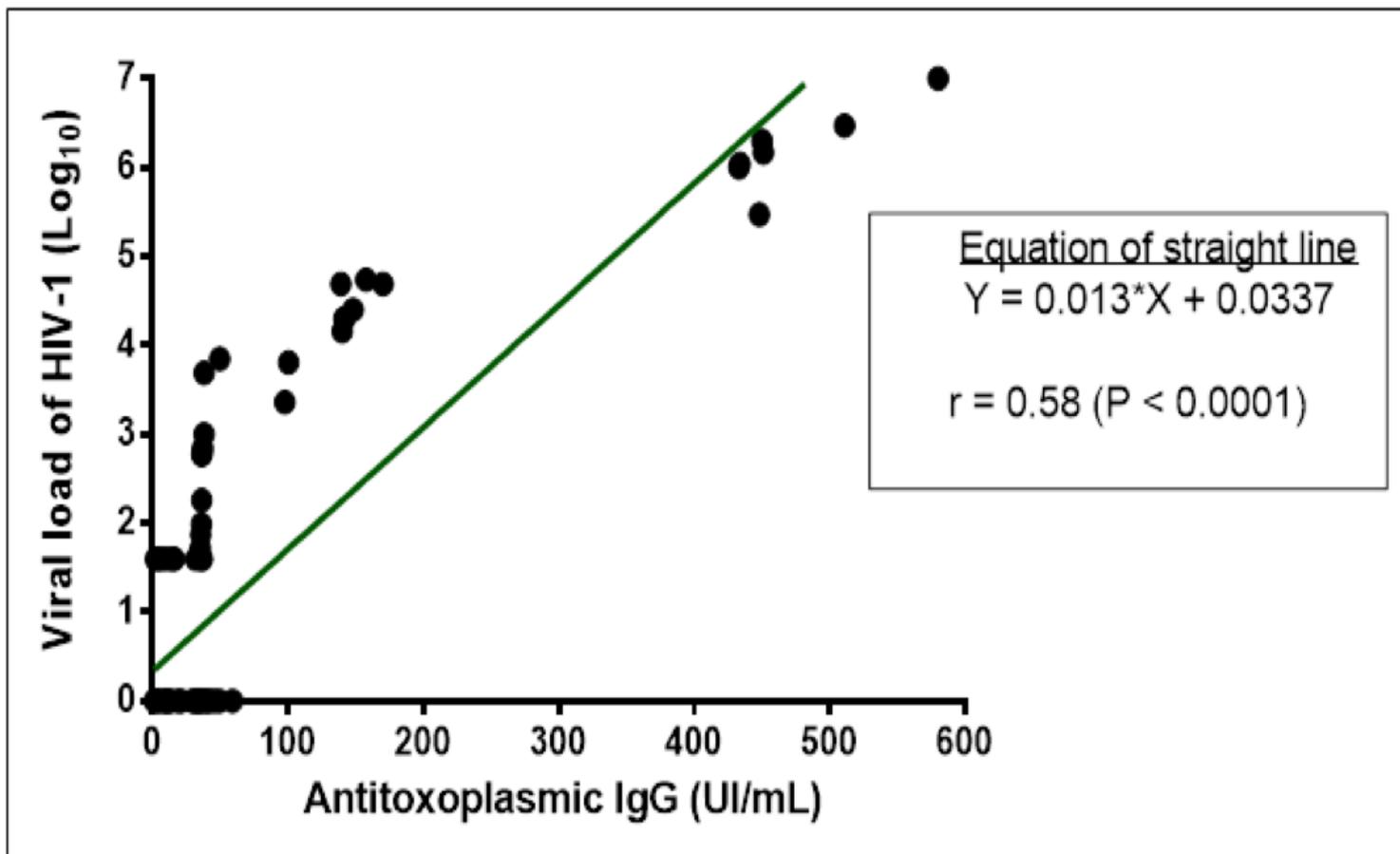


Figure 2

Correlation straight line between anti-toxoplasmic IgG level and HIV-1 viral load. HIV-1: human immunodeficiency virus type 1; Ig: Immunoglobulin.

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Table1.png](#)
- [Table2.png](#)
- [Table3.png](#)