

# Prevalence and density of malaria parasitaemia amongst HIV Individuals in Warri, Nigeria

Johnson Daniel Jemikalajah<sup>1</sup>, Clement Oliseloke Anie<sup>2</sup>, Felix Oghenemaro Enwa<sup>2</sup>

1. Department of Microbiology, Faculty of Science, Delta State University Abraka, Delta State, Nigeria.
2. Department of Pharmaceutical Microbiology, Faculty of Pharmacy, Delta State University, Abraka, Delta State, Nigeria.

## Abstract:

**Background:** Malaria parasite has been observed to be a common infection in Human Immunodeficiency virus (HIV), an increase malaria infection in adults.

**Objective:** This experimental study is sets to determine the prevalence and density of malaria parasitaemia in Warri community, South-Southern Nigeria.

**Methods and Results:** A total of 600 participants were screened for Human immunodeficiency virus and malaria parasite using WHO systems two and Geimsa staining technique for thick and thin blood films and absolute parasite counts done respectively. The prevalence rate of 38% and 39% were obtained for malaria parasite infection among HIVSP and HIV/ span>SN respectively. The difference in malaria parasite infection was not statistically significant ( $P>0.05$ ) between HIVSP and HIVSN. However, the mean parasite density in HIVSP was significant ( $P<0.05$ ) when compared with HIVSN. The mean parasite densities of  $2384 \pm 747$  and  $1883 \pm 645$  were recorded for HIVSP and HIVSN respectively. The mean parasite densities of  $2385 \pm 782$  and  $2383 \pm 717$  observed for males and females respectively showed no statistical significant difference ( $P<0.05$ ).

**Conclusion:** This study has shown a high prevalence of malaria parasite among the HIV infected subjects.

**Keywords:** Density, Malaria, HIV, prevalence, Nigeria.

**DOI:** <https://dx.doi.org/10.4314/ahs.v21i2.16>

**Cite as:** Jemikalajah JD, Anie CO, Enwa FO. Prevalence and density of malaria parasitaemia amongst HIV Individuals in Warri, Nigeria. *Afri Health Sci.* 2021;21(2). 614-618. <https://dx.doi.org/10.4314/ahs.v21i2.16>

## Introduction

Malaria is a parasitic disease that is transmitted from one individual to another through the bite of the female anopheles mosquito<sup>1</sup>. Those living in the endemic and poorest regions are vulnerable to contracting malaria<sup>2</sup>. It is the commonest disease of the tropical and subtropical regions of the world and accounts for more than 300 million infected persons and a minimum of 1 million deaths yearly<sup>3</sup>. It is an endemic disease in Nigeria and constitutes about 60% of the illness consultations in the country and about 10% in Warri, south south<sup>4</sup> Nigeria. Malaria accounts for 25-30% of infant and childhood mortality<sup>5</sup>. In Warri, south-south of Nigeria, the malaria parasite rate is 57.9%<sup>6</sup>. Human immunodeficiency virus (HIV) infection has been a major cause of illness and death among children, teens and young adults' worldwide<sup>7</sup>. It accounts for most premature

mortality in sub-Saharan Africa and the entire world<sup>8</sup>

In respect to geographical overlap between malaria and HIV/AIDS, a reasonable number of coinfections occur<sup>9</sup>. Infection with HIV-1 causes progressive cellular immunosuppression, and any resulting impairment in immune response to malaria might be associated with failure to prevent infection<sup>10</sup>. Some components of human immune response to *P. falciparum* are modified by HIV and further increase the potential reservoir in the patient thereby increasing the number of chemokine receptor positive macrophages. This response stimulates HIV-1 replication through the production of cytokines by activated lymphocytes<sup>11</sup>. During the process, the number of chemokine receptor positive macrophages was increased as result of elevated reservoir for HIV in the placenta<sup>12</sup>.

A study in Guinea Bissau found no difference in HIV-2 progression as measured by viral load between adults with parasitaemia and those without<sup>13</sup>. Whereas, in Malawi HIV plasma viral load were found to be significantly higher in patient with malaria than in those

## Corresponding author:

Johnson Daniel Jemikalajah,

Phone:+2347032222372/

E-mail: Jemikalajahjohnson2007@yahoo.com

without<sup>14</sup>. This is slightly difficult to interpret because there was no base line viral load measurement available before the episodes of malaria. HIV infection and malaria are disturbing because they co-exist in African countries<sup>15,16</sup> but studies vary in their outcomes in the interactions between the infections<sup>17,18</sup>. This may be why HIV and malaria still remain the most essential global health challenges of the developing countries including Nigeria where it has been reported to cause more than 4 million deaths yearly with HIV infection increasing the danger and severity of malaria infection and its burdens<sup>19</sup>. This suggests that malaria can cause faster progression of HIV disease whereas HIV in turn facilitates the rate of malaria transmission which causes strong immune cell activation and up-regulation of pro-inflammatory cytokines production which create ideal microenvironment for the spread of HIV among CD4 cells, thus rapid HIV replication<sup>20</sup>. This provides the best evidence of an effect of malaria of HIV in non pregnant adults.

Both HIV and malaria are probable factors in the modification of each other's development, disease severity, and disease progression rate<sup>21</sup>. This is an indication that malaria is still a compound factor in HIV infection. Thus, this study was aimed at determining the prevalence and density of Malaria parasitaemia among HIV infected patients to provide information to improve the management of HIV and malaria in

### **Materials and methods**

This study was conducted amongst inhabitants of Warri, in Central Hospital, Nigeria for a period of 2016 to 2018. Prospective comparative study was carried at Central Hospital, Nigeria. A total of 600 subjects were recruited into the study.

### **Ethical and sample collection**

Samples were collected from participants based on the approved ethical procedures of Central Hospital, Warri research and ethics committee with No. CHW/HA/45/2018.

### **Sample analysis**

Blood samples were collected from the subjects into EDTA containers and transported to the laboratory for analysis.

### **Human immunodeficiency virus screening**

This was carried out using two enzymes linked immunosorbent assay rapid screening kits based on WHO systems two<sup>22</sup>. Determine and immunocomb II rapid screening kits of Abbott Laboratories, Japan and Organics, France were used. Test was carried out according to manufacturer's instruction.

### **Malaria parasite screening**

This was determined by Geimsa staining technique of thick and thin blood films<sup>16</sup>. Malaria parasite count was done using WHO standard method of absolute parasite count (counting parasites number/microlitre of blood)<sup>23</sup>.

### **Data analysis**

Data obtained were analyzed using statistical package for social sciences (IBM SPSS version 25.0 which includes descriptive statistics, mean, standard deviation for observed variables. The association between variables was tested using Chi-square and t-test to compare infectivity between seropositive and seronegative subjects. All test were two tails with statistical significance set at 5% (0.05) confidence level.

### **Results**

Out of the 600 subjects examined, 300 were HIV seropositive (HIVSP), comprising of 143 males and 157 females while 300 were HIV-seronegative (HIVSN).. The prevalence rate of 38% and 39% were obtained for malaria parasite infection amongst HIVSP and HIVSN subjects respectively. The malaria parasite infection was not statistically significant ( $P > 0.05$ ) among HIVSP and HIVSN (Table:1).

**Table 1:** Prevalence of malaria parasite in the study populations.

Subjects	No. examined	No.(%) infected	No.(%) not infected	% prevalence	X <sup>2</sup> cal.	P-value
HIV-seropositive subjects	300	228(76)	72(24)	38		
					0.34	3.843
HIV-seronegative	300	243(78)	66(22)	39		
Total	600	462(77)	138(23)	77		

Table: 2 shows that the mean parasite densities (MPD) of HIVSP subjects was significantly high ( $P < 0.05$ ) when compared with HIVSN subjects. Meanwhile, the mean parasite densities of  $2384 \pm 747$  and  $1883 \pm 645$  were recorded for HIVSP and HIVSN subjects respectively.

It was also observed that there was no significant difference ( $P < 0.05$ ) in the prevalence of malaria infection among HIVSP males and females. A mean parasite densities of  $2385 \pm 782$  and  $2383 \pm 717$  were obtained for males and females respectively in this study (Table:3).

**Table 2:** Mean parasite density of HIV seropositive and HIV seronegative subjects

Subjects	No. examined	No. (%) infected	Mean parasite density ( $X \pm S.D$ )	t cal	P-value
HIV seropositive subjects	300	228(76)	2384±747		
Hiv seronegative subjects	300	234(78)	1883±645		
				8.8	1.96
Total	600	462(77)	2130±740		

**Table 3:** Sex related distribution of malaria parasitaemia amongst HIV seropositive subjects

Sex	No. examined	No. (%) infected	No. (%) not infected	X <sup>2</sup> cal.	P-value
Males	143	109(76.2)	34(28.8)		
Female	157	119(75.8)	38(24.2)		
				0.07	3.841
Total	300	228(76)	72(24)		

## Discussion

This study has demonstrated that malaria parasite infection was not significantly different among HIV seropositive and HIV seronegative ( $p = 3.843$ ) This confirms the earlier report<sup>14</sup> that there was no significant difference between HIV infection in adults with parasitaemia or

without to malaria parasite which might be associated with failure to prevent infection as earlier reported<sup>9</sup>.

The overall prevalence rate of 77% observed for malaria parasite infection in this study is far higher than the prevalence rate of 57.9% reported<sup>6</sup> in part of Cross River State. This difference may be due to the timing

of the study and seasonal variation. The high mean parasite density observed in HIVSP subjects was significantly higher than in HIV and agrees with the earlier report<sup>23</sup> that HIV infection is associated with an increased frequency of malaria parasitaemia.

Meanwhile, the high prevalence rate of malaria parasite infection among HIVSP subjects in this study was far higher than the 7.3% earlier reported<sup>24</sup> in parts of Delta State. This agrees with the fact that malaria is an endemic disease in Nigeria and constitutes about 60% of illness consultation as earlier reported<sup>4</sup>. However, this study was carried out partly during the wet season when the vector of malaria parasite (female anopheles mosquito) has a lot of breeding sites.

### Conclusion

This study has shown that there was no significant difference among malaria parasite and HIVSP subjects in the studied area. Therefore, HIV infected patients should be encouraged to avoid malaria infection by ensuring that mosquitoes are adequately prevented by sleeping under insecticide treated net (ITN) as recommended by World Health Organization (WHO) so as to reduce the morbidity and mortality associated with malaria and HIV infections.

### Acknowledgement

We appreciate the entire staff of Central Hospital Warri for all their support.

### Funding

None.

### Conflict of interest

None.

### References

1. World Health Organization. United Nations Decade to Roll Back malaria: monitoring and Evaluation Geneva. Available from <http://www.who.int/inffs/en/informationsheet11.pdf>.1-23. 2002.
2. World Health Organisation and United Nations Children's Fund. Africa malaria Report Available from <http://www.rbm.who.int/amd/2003/amr/2003/amr-toc.htm>.1-12. 2003.
3. Roll Back Malaria. What is malaria? Available from <http://www.rbm.int/>. 2003, 1-30
4. Federal Ministry of Health (FMOH). National Malaria and vector control division. 1998, 7-15.
5. Netmark. Insecticide treated materials in Nigeria. *Regional Africa Programme Briefing Book*. 2000, 1-10.

6. Alaribe AA. Amodiaquine in the therapy of chloroquine resistant *Plasmodium falciparum* infection in parts of South Eastern Nigeria. A thesis submitted as parts of the requirement for the degree of master of science of the university of Calabar, Calabar. 1990, 33-39.
7. Masci J. HIV/AIDS update. Available from <http://kidshealth.org/parent/infections/std/hiv.html>. UN-AIDS/WHO 2003: "AIDS epidemic update". 1-40.
8. Wabwire-Margen F, Shift CJ, Viahov D, Kline R, Serwadda D, Sewankombo NK, Mugerwa, RD and Quinn TC. Immunological effects of HIV-1 infection on the humoral response to malaria in an African population. *American Journal Tropical Medicine of Hygiene*. 1998, 4(15):504-511
9. World Health Organisation. Malaria in HIV/AIDS patients. <https://www.who.int>. 2017.
10. Migot F, Quedrago JB, Diallo J, Zampan H, Dubois B, Scott-Finigan T, Sanou PT and Deloron P. Selected *P. falciparum* specific immune responses are maintained in AIDS adults in Burkina-Faso. *Parasite Immunology*. 1996, 18(17):333-339.
11. Tkachuk AN, Moormann AM, Poore JA, Rochford RA, Chensue SW, Mwapasa V and Meshnick SR.. Malaria enhances expression of CC Chemokine receptor 5 or placenta macrophages. *Journal of Infectious Diseases*. 2001, 183(6):967-972.
12. Ariyoshi K, Berry N, Wilkins A, Ricard D, Aaby P, Naucier A, Ngom PT, Jobe O, Jaffer S, Dias, Tedder RS and Whittle H. A community based study of human immunodeficiency virus type-2 provirus load in rural village in West Africa. 1996, *Journal Infectious Diseases*. Vol.173(1):245-248.
13. Hoffman IF, Jere CS, Taylor TE, Munthali P, Dyer JJ, Fiscus SA, Chakraborty H, Taha TE, Cohen MS and Molyneux ME. The effect of *Plasmodium falciparum* on HIV-1RNA blood plasma concentrations. *Acquired Immune Deficiency Syndrome*; 1999, 13(4): 487-494.
14. Whitworth J. Malaria and HIV. HIV insite knowledge base chapter March. Medical research council programmes on AIDS, Uganda virus Research institute. *A Project on the UCSF Centre for HIV information* 2002, 23-50.
15. Abu-Raddad IJ, Patnaik P, Kublin JG. Dual infection with HIV and malaria fuels the spread of both diseases in sub-saharan Africa. *Science*. 2006,314:1603-1126
16. Nkuo-Akenji T, Tevoufouet EE, Nzang F, Ngufor N.High prevalence of HIV and Malaria co-infection in urban Douala, Cameroon. Africa. *Journal of Aids Research*.2008, 7: 229-35.
17. Saracino A, Nacarapa FA, da Costa Massinga FA, Prevalence and clinical features of HIV and malaria

- co-infection in hospitalized adults in Beira, Mozambique. *Malaria Journal*. 2012, p.241 10.1186/1475-2875.
18. Flateau C, Le Loup G, Pialoux G. Consequences of HIV infection on malaria and therapeutic implications: A systematic review. *The Lancet Infectious Diseases*. 2011, pp. 541-556.
19. Alemu A, Shiferaw Y, Addis Z, Mathewos B. Effect of malaria on HIV AIDS transmission and progression. *Parasitology vectors*. 2013, 6: 1810-1186.
20. Felicia EJ, Tinuade IO, Surajudeen AA, Henry AM. Effect of HIV and malaria parasites co-infection on immune- haematological profiles among patients attending anti- retroviral treatment (ART) clinic in infectious Disease Hospital in Kano, Nigeria. *PLoS One*. 2017, 12(13): 10. 1371.
22. Kassler WJ, Alwano-Edyegu MG, Marum E. Rapid HIV testing with same day result: a field trial in Uganda. *International Journal of Sexually Transmitted Diseases-Acquired Immune Deficiency Syndrome*. 1998, 9:134-138.
23. Cheesbrough M. District laboratory practice in tropical countries. Low price edition, Cambridge University Press. Part 2, 2000, 253-264.
24. More JM, Ayisi J, Nahlen BL, Misore A, Lal AA and Udhaykumar V. Immunity to placenta malaria. 11. Placenta antigen-specific cytokine responses are impaired in human immunodeficiency virus-infected women. *Journal of Infectious Diseases*. 2000, 182(3):960-964.