The effect of antiretroviral naïve HIV-1 infection on the ability of autologous Natural Killer cells to produce IFNγ upon exposure to *Plasmodium falciparum*-infected Erythrocytes

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INTRODUCTION

1- Regions of intense *Plasmodium* species transmission

2- High prevalence of HIV infection (WHO, 2015)

ARV naïve HIV-1 infected people living within these regions are repeatedly exposed to malaria.

Production of IFNγ

Direct lysis of iRBC

*P. Falciparum* infected red blood cells (iRBC)

Natural Killer cell

HIV infection
Participants
- 15 ARV naïve HIV-1 infected participants and 15 Healthy controls aged between 21 to 65 years were included in this study.
- 03 malaria infected people.

Methods
1- iRBC were enrichment and culture (for 72 hours)
2- NK cells purification using Peripheral blood mononuclear cells
3- Purified NK cells were co-cultured with the iRBC for 24 hours
4- NK cells IFNy production was measured by multiparametric flowcytometry using BD FACScanto II machine and data were analyzed by flowjo 10 tristar. SPSS and graphpad prism 5 software were used for statistical analysis
RESULTS

Gating strategy

**Figure A**: Flowjo analysis for NK cells IFNγ production after co-culture with Red Blood Cells

- Data show a significant reduction (p=0.02) in IFNγ production by NK cells from antiretroviral naive HIV-1 infected people after co-culture with *plasmodium falciparum* infected RBCs.
- NK cells IFNγ production clearly indicates that this response was iRBC dependent.

**Figure B**: Production of IFNγ by NK cells before and after coculture with plasmodium infected red blood cells (iRBC) and uninfected red blood cells (uRBC).
RESULTS

**Figure C:** Impact of HIV Viral Load on Natural Killer cells IFNγ production after coculture with iRBC

IFNγ production by NK cells from untreated HIV-1 infected participants correlated inversely with the plasmatic viral load ($r = -0.9; p<0.05$).
CONCLUSION

Thus antiretroviral naïve HIV-1 infection can dampen NK cell mediated immunity to Plasmodium falciparum infection in malaria intense regions. This could in effect escalate morbidity and mortality in people chronically infected with HIV-1.