

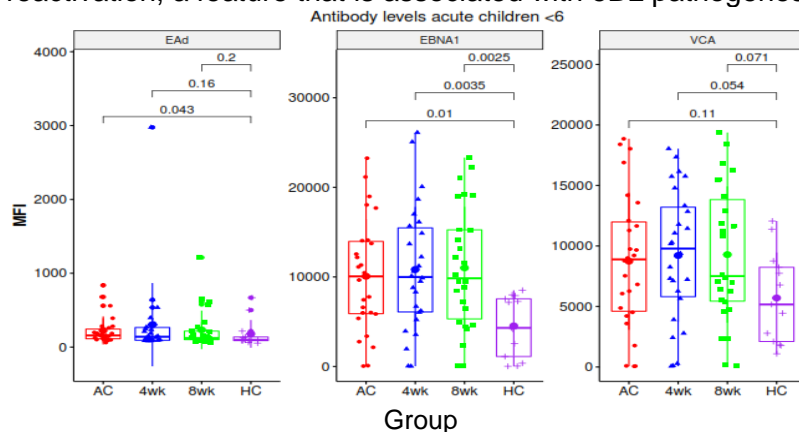
# ASSESSMENT OF THE IMMEDIATE EFFECT OF *PLASMODIUM FALCIPARUM* MALARIA INFECTION ON EPSTEIN BARR VIRUS LATENCY AND REACTIVATION USING A MULTIPLEX SEROLOGICAL APPROACH

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EBV alongside repeated *P.f* malaria infections are considered as co-etiological agents in the pathogenesis of endemic Burkitt lymphoma (eBL), one of the most prevalent pediatric cancers in sub-Saharan Africa. The mechanisms involved are however largely undefined. Children and adults living in malaria holo-endemic areas have serological evidence of increased EBV reactivation. In an early seminal study, elevated titers of IgG against VCA were shown to be prognostic risk factor for BL development. Also, elevated titers of Zta and VCA IgG are reported in BL cases compared to controls. Previous serological studies have either analyzed samples from eBL cases versus healthy controls or analyzed samples from people with divergent malaria exposure. However, to date, no study has evaluated the direct and immediate effect of acute clinical malaria on EBV serology as a signature of EBV reactivation. In this study we tested the hypothesis that acute clinical *P.f* malaria results in elevated levels of EBV specific antibodies. Using a bead-based multiplex assay, we tested antibodies against VCA, EAd and EBNA1 antigens. In the preliminary data that we are presenting here, we analyzed samples from 26 children with acute uncomplicated clinical malaria and 17 healthy controls age range 1-6 years. Children with acute malaria were followed up at 4 and 8 weeks. As expected, EAd IgG, a lytic gene, were significantly elevated in the malaria cases at enrollment as compared to the healthy controls, *P*-value **0.043**. The levels of anti-EBNA1 antibodies among HC were significantly lower compared to cases at enrollment, 4- and 8-week recovery. Similarly, though not statistically significant, healthy controls had generally low VCA IgG antibodies compared to acute malaria cases. Overall, this study confirms the synergy between EBV and *Plasmodium falciparum* malaria in eBL pathogenesis as it demonstrates the immediate effect of clinical *P.f* malaria on EBV reactivation, a feature that is associated with eBL pathogenesis



Key:

AC- Acute cases at enrollment  
4wk- 4week recovery  
8wk- 8week recovery

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