to SP. We therefore assessed whether SP-IPT has remained effective against malaria and anemia despite this trend. We conducted a cross-sectional analysis of third trimester pregnant Malawian women who were being screened for enrollment in a study investigating the effects of malaria on HIV mother-to-child transmission. As part of the screening process, venous blood was collected for HIV rapid antibody tests, malaria blood smears and hemoglobin (Hb) measurement. In addition, various data were collected, including socio-demographic information and the use of SP-IPT, iron/folate supplementation and bednets. From 2000-2004, 3821 women were screened for HIV, of whom 1155 (30.2%) were infected. 9.3% (353/3815) of women had peripheral parasitemias and 44.1% (1675/3799) had anemia (Hb less than 11g/dl). The odds of malaria parasitemia were lower for women who took one or more SP-IPT dose than those who did not, after adjusting for confounders including HIV status and parity (Adjusted Odds Ratios [AOR], 95% Confidence Interval (CI); 0.70 (0.44-1.10), 0.72 (0.46-1.13) and 0.53 (0.32-0.86) for women taking 1, 2 and 3 or more SP-IPT doses, respectively). Similarly, in multivariate analysis which adjusted for HIV status, parity, malaria, duration of iron supplementation and other confounders, the odds of anemia were lower for women who took one or more SP-IPT dose than those who did not (AOR, 95%CI; 0.77 (0.57-1.04), 0.63 (0.47-0.85) and 0.55 (0.40-0.75) for women taking 1, 2 and 3 or more SP-IPT doses, respectively). Despite the high prevalence of malaria parasites with DHFR and DHPS mutant genotypes in Malawi, SP-IPT appears to be effective against malaria and anemia. However, 3 or more SP-IPT doses may be required.

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IDENTIFYING THE CAUSAL CHANNELS OF THE MALARIA GAP: POPULATION MOBILITY AND ITS IMPACT ON GDP IN THE FORMER FEDERATED MALAY STATES

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There is an enormous gap when estimating the economic burden of malaria depending on whether macroeconomic or microeconomic methodologies are employed. Over the 16-year period from 1980-1995, macroeconomic estimates of malaria's economic burden in sub-Saharan Africa (SSA) exceed US$ 73 billion while conventional microeconomic studies yield an estimate of US $ 7 billion (expressed in 1987 US $ adjusted for purchasing power parity) {Sachs, 2002; WHO, 2001}. This order of magnitude discrepancy is due to differences in the accounting schemes between the two methodologies. The macroeconomic studies utilize cross-country regression analysis while the conventional microeconomic studies apply a cost of illness (COI) methodology with a narrowly defined set of costs for inclusion. A missing piece in explaining the "malaria gap" is an analysis of the pathways affecting economic growth which cannot be identified in cross-country regression or COI analyses. We quantified the effect of malaria on gross domestic product (GDP) and GDP per capita in the former Federated Malay States (FMS) compared to the conventional microeconomic burden from 1906 to 1920. Malaria would have restricted GDP growth in the FMS over the investigated 15-year period by 1.39% per annum and GDP per capita growth by 1.07% per annum. The main channel through which malaria could have inhibited economic growth would have been an inability to recruit and maintain a healthy work force in the high transmission areas where they were needed. The total macroeconomic burden of malaria in the FMS over our period of study was approximately US$ 2.85 billion (expressed in 2000 US$). In comparison, the microeconomic burden with integrated vector control was only US$ 110 million while the microeconomic burden without integrated vector control would have been actually even less, around US$ 12 million, due to the direct costs of integrated vector control being greater than the resulting savings in treatment and indirect costs (expressed in 2000 US$). We conclude that the conventional approach to evaluating the microeconomic burden of malaria is inappropriate due to its circular nature and failure to account for the focal aspect of malaria's burden on society. Alternatives to the COI methodology are urgently needed to inform policy makers of the means, if any, through which malaria exhibits an externality to income in SSA economies today.

(ACMCIP Abstract)

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ANALYSIS OF SINGLE NUCLEOTIDE POLYMORPHISMS AT THE *PLASMODIUM VIVAX* APICAL MEMBRANE ANTIGEN 1 (*PVAMA1*) LOCUS AMONG SRI LANKAN ISOLATES

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The assessment of genetic structure among parasite populations has significant relevance for both the control and epidemiology of malaria; in particular, examining diversity among antigenic loci is crucial for effective local vaccine development. Here, we characterized the distribution of SNPs in both the coding and non-coding regions of the eminent vaccine candidate, *Plasmodium vivax* AMA-1, in Sri Lanka. An extensive analysis of this type has yet to be conducted from field isolates in this region. Furthermore, this is the first time polymorphisms across the entire coding sequence of *PvAMA-1*, as well as its upstream region, have been investigated. Blood samples were collected from patients presenting at clinics in both non-endemic (Colombo) and endemic settings in Sri Lanka (Kataragama and Anuradhapura). 30 single clone infections were selected following RFLP analysis at the *MSP-3α* locus. SNPs, the majority of which were non-synonymous, were found amongst all three ectodomains of *PvAMA-1*, but were notably absent in the pro-domain and transmembrane/carboxy-terminal tail regions of the gene. Polymorphisms across ectodomains I and II specifically, show significant deviance from neutrality (McDonald-Kreitman test; $p<0.05$), consistent with previous findings that this antigen is likely under host immune selection. Each polymorphic site uncovered here was also conserved among *vivax* isolates from various localities in Africa and Asia. When these residues were mapped onto the recently published *PvAMA1* crystal structure (Pizarro, 2005), the majority clustered on the outer surface of the protein; two electrostatically interesting polymorphisms were also localized to a putative receptor-binding site of the protein. Finally, the SNP profile of the 5' upstream region of *PvAMA-1* was characterized following sequence analysis of a ~700bp segment upstream of its start codon in five isolates. Only two SNPs, one single-site deletion, and two insertion-deletion repeats were evident, representing a polymorphism frequency that is significantly lower than that found in the coding region of *PvAMA-1*.