Approximately 75,000 or 0.42% from Sub-Sahara Africa were resettled in the United States. Peer reviewed medical literature and evidence based research for the past two decades described the exposure of refugees to the detrimental effects of pathogenic parasites from countries of origin or while in refugee camps. The aim of the Study is to contribute to a reduction of health disparities that may challenge the effective implementation of interventions and strategies to reduce or eliminate asymptomatic and reactivated diseases. Misdiagnosis of endemic diseases in Sub-Saharan refugees arriving to settle in the United States was also included. We examined peer reviewed medical literature and evidence based research from 2002-2015 using: National Vital Statistics System (NVSS), MEDLINE, PubMed, Cochrane Library, and Science Direct. Data on prevalence of asymptomatic or reactivated pathogens as well as clinical misdiagnosis amongst refugees entering the United States to settle was included in the search. Five medical conditions were selected: Latent TB, Malaria, Schistosomiasis, Strongyloidiasis and Oral health. The criteria applied were: Eosinophil count, regional diagnosis disease, geographical analysis, mapping, screening and surveillance. Although the population of Sub-Saharan refugees entering the United States is small (0.42% or approximately 75,000) findings of the Study indicate that hospital departments are concerned with the economic burden of treating refugees and immigrants. Hospitals may lack health care professionals with sufficient training and skills to treat refugees with tropical or endemic disease. Medical schools must include differential diagnosis in their syllabi that train students to: diagnose, treat and manage the health of the growing foreign born population in the United States that will become patients before or after resettlement. This requires skills and knowledge of diseases endemic to country of origin and risk to the foreign born resettling in the United States. Public Health policy and surveillance does not always include preventative health initiatives or programs that consider the health disparities of refugees born in Sub-Sahara Africa.

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RELAPSES VERSUS REINFECTIONS: ASSESSING THE PARASITOLOGICAL AND CLINICAL IMPLICATIONS USING *PLASMODIUM CYNOMOLGI* AS A MODEL FOR *P. VIVAX*

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Plasmodium vivax causes significant morbidity and mortality worldwide and remains a major obstacle to global malaria eradication. One of the obstacles this parasite presents is its liver-stage reservoir comprised of hypnozoites that are capable of reactivating and causing relapses. Relapses are thought to contribute significantly to the prevalence and transmission of *P. vivax*, but it is unclear if either relapses or reinfections are more responsible for clinical vivax malaria cases. To assess the contribution of relapses as well as homologous and heterologous re-infections to clinical vivax malaria, a series of experiments using the rhesus macaque - cynomolgi malaria model were conducted. Clinical and parasitological data were collected daily for 100 days during the initial infection and for 45 – 60 days during the re-infections. Relapses did not induce significant clinical alterations, and when minor changes were observed, they resolved without the need for clinical intervention. Homologous reinfections resulted in considerably lower parasite burden and minimal alterations, if any, in clinical parameters, similar to relapses. Interestingly, infection with a heterologous strain of *P. cynomolgi* did result in significant changes in clinical parameters, although there may have been some clinical protection conferred by the initial infections given the evidence of self-controlled acute parasitemia upon heterologous challenge. Collectively, the data from these experiments suggest that relapses caused by *P. vivax* parasites that are genetically similar to parasites in primary infections and homologous re-infections likely do not contribute significantly to clinical vivax malaria cases. Contrastingly, infections with genetically dissimilar strains of *P. vivax* can have pathological consequences, although severity may be less than with the initial infection. Overall, these studies demonstrate that there is much to learn about the clinical consequences of relapses and re-infections and also highlight that the dynamics of *P. vivax* infections are complicated.

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PLASMODIUM FALCIPARUM FIELD ISOLATES TRIGGER APOPTOSIS PREFERENTIALLY IN HUMAN BRAIN ENDOTHELIAL CELLS COMPARED TO PULMONARY ENDOTHELIAL CELLS

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Plasmodium falciparum infection can progress unpredictably to severe forms including respiratory distress and cerebral malaria. The mechanisms underlying the variable natural course of malaria remain elusive. Here we used cocultured brain and pulmonary endothelial cells challenged with *P. falciparum* field isolates taken directly from malaria patients and scrutinized their capacity of inducing endothelial apoptosis via cytoadherence or not. A total of 27 *falciparum falciparum* isolates were collected from patients with uncomplicated malaria (n=25) or severe malaria (n=2). About half the isolates (n=17) were able to bind brain endothelial cells (12 isolates, 44%) or lung endothelial cells (17 isolates, 63%) or both (12 isolates, 44%). Sixteen (59%) of the 27 isolates were apoptogenic for brain and/or lung endothelial cells. The apoptosis stimulus could be cytoadherence, direct cell-cell contact without cytoadherence, or diffusible soluble factors. While some of the apoptogenic isolates used two stimuli (direct contact with or without cytoadherence, plus soluble factors) to induce apoptosis, others used only one. Among the 16 apoptogenic isolates, eight specifically targeted brain endothelial cells, one lung endothelial cells, and seven both. These results suggest that *falciparum falciparum* field isolates killing brain endothelial cells are more prevalent than those killing pulmonary endothelial cells and may provide new insights into host-parasite interactions.

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CHARACTERIZATION OF ANTIBODIES AGAINST *PLASMODIUM FALCIPARUM* INVASION PROTEIN PFMSP10

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The aim of this study is to evaluate monoclonal antibodies (mAb) generated against pfMSP10 (Merozoite surface protein 10) and their functional role to inhibit the invasion of Peruvian *Plasmodium falciparum* isolates using the Growth inhibition Assay (GIA) *in vitro* as well as the potential use of pfMSP10 in malaria diagnostics. Seven mAb (Gen Script and Abmart) and a polyclonal antisera were evaluated by Western Blot (WB) against the recombinant MSP10 full protein (rMSP10). Synchronized and purified schizonts from *P. falciparum* 3D7 and their concentrated supernatant were obtained by ultrafiltration. Detection of pfMSP10 protein was also evaluated in synchronized ring stage of *P. falciparum* cultures from 1 to 12 hours post-invasion. IFA assays were also carried out. In addition a quantitative direct sandwich ELISA for rMSP10 was developed using all the possible combination of the eight antibodies in evaluation. From all the eight evaluated antibodies, only one mAb (anti pfMSP10-1)