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# Variant surface antigens in cerebral malaria: distinct from others and similar to each other?

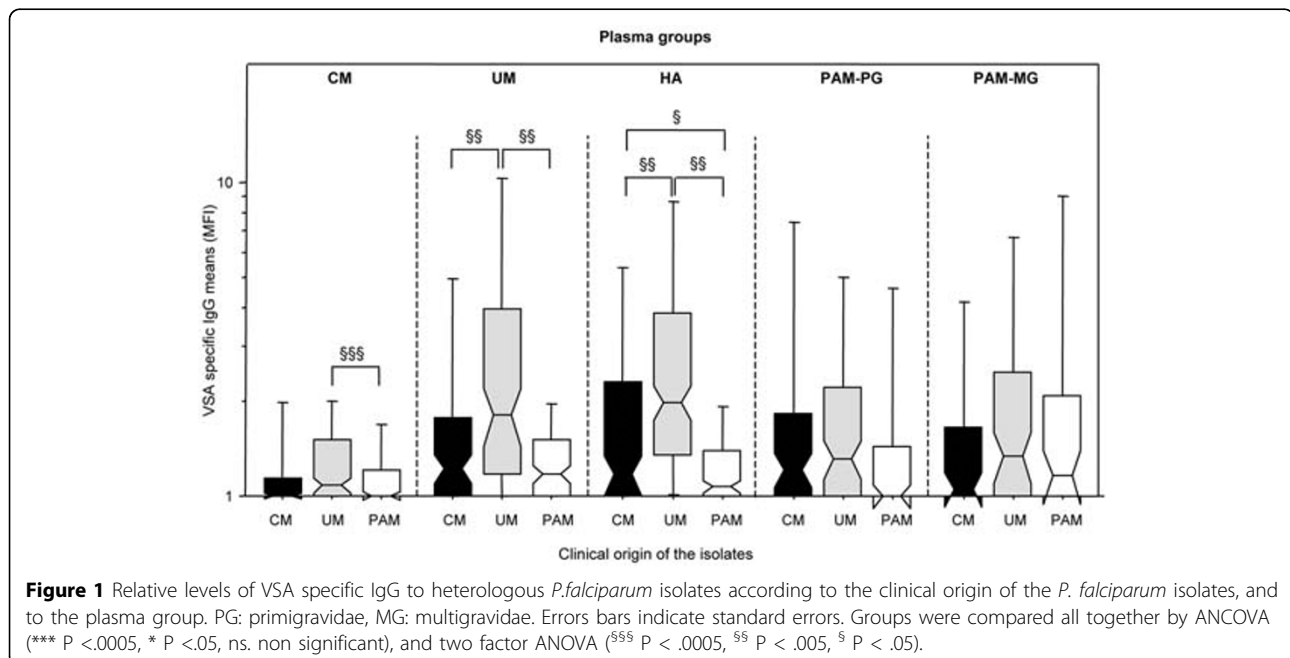
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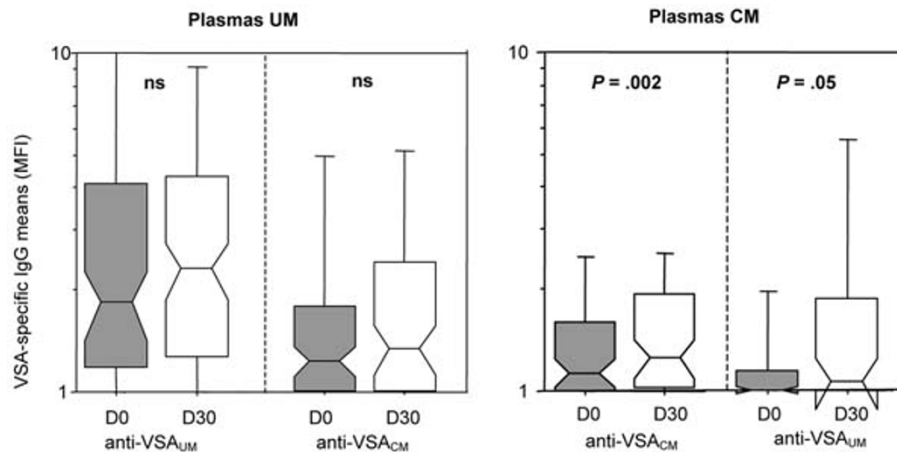
Immunological protection against *Plasmodium falciparum* blood stages is mainly antibody mediated [1,2]. Variant surface antigens (VSA) expressed on the surface of *P. falciparum*-infected red blood cells constitute a key for parasite sequestration and immune evasion [3]. In distinct malaria clinical presentations, as placental malaria, specific antibody response against VSA provides protection [4].

In the current study, we investigated in distinct clinical groups of malaria patients, the antibody response specifically directed against VSA expressed by parasites isolated

from a given clinical presentation, and particularly isolates obtained from cerebral malaria (CM) patients. Plasma and isolates were obtained from four groups of Beninese subjects: healthy adults (HA, n = 34), patients presenting uncomplicated malaria (UM, n = 62), cerebral malaria (CM, n = 41), or pregnancy-associated malaria (PAM, n = 24). Isolates were tested for their clonality by *msp1* and *msp2* genotyping. The reactivity of plasma samples from each clinical group was measured by flow cytometry against parasites isolated from individuals from each clinical group.



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**Figure 2** Acquisition of VSA-specific IgG during the month following *P. falciparum* infection in plasmas samples from UM (n = 121) and CM (n = 56) patients. UM (A, B) or CM (C, D) plasma samples were tested against *P. falciparum* isolates from UM patients (A, D) or from CM patients (B, C). Centerlines indicate medians, boxes indicate the 25th and 75th percentiles of data points, bars indicate the 10th and 90th percentiles and circles are outliers. Differences are derived from the Wilcoxon rank test for paired comparisons, ns: non significant.

The levels of clonality were similar in isolates from all clinical origins. In healthy adults and children presenting UM,  $VSA_{UM}$  antibody levels were higher than  $VSA_{CM}$  antibody levels (Figure 1). In both PAM plasma groups (primigravidae and multigravidae), antibody levels against the three types of isolates were similar. One month after infection the level of anti-VSA antibodies able to recognize heterologous  $VSA_{CM}$  variants was increased in CM patients. In UM patients, antibody levels directed against heterologous  $VSA_{UM}$  were similar during the infection and one month later (Figure 2).

The existence of shared  $VSA_{CM}$  epitopes was shown but does not necessarily involve prevalent epitopes. Prevalence is more probably due to a fine balance between transmission intensity, antibody repertoire and environmental factors.

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