Multigene families of *P. falciparum* are central to severe malaria in human.

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P. falciparum has been known as the “the strongest force for evolutionary selection in the recent history of the human genome”. Most of the severity associated with *P. falciparum* is attributed to the fact that it can either sequester in the microvasculature or bind to uninfected erythrocytes, the phenomena known as cytoadherence and rosetting, respectively. In my talk, I would discuss about two members of the multigene families, PfEMP1 and RIFIN, which are responsible for sequestration of the parasites in the microvasculature and have tremendous impact on the genetic burden in endemic areas of Africa. We have identified a new molecular pathway through which PfEMP1 gets transported onto the infected RBC surface to mediate malaria pathogenesis in placental malaria parasites and this conserved pathway could be a potential target to reduce severity. To further understand and explain genetic pressure in endemic areas due to malaria pathogenesis and genetic drift of population towards blood group O, we discovered the role of RIFINs (not PfEMP1) in mediating increased rosetting of parasites. Thus, RIFINs play a major role in ABO blood group distribution.