The role of von Willebrand Factor (VWF) in malaria pathogenesis

Fact Sheet

Project Information

VWFMAL
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EU contribution
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Coordinated by
LIVERPOOL SCHOOL OF TROPICAL MEDICINE
United Kingdom

Objective

The pathogenicity of Plasmodium falciparum is thought to relate to the unique ability of infected erythrocytes to adhere to and subsequently activate the vascular endothelium. The primary process of cytoadherence has been studied in detail and a large number of receptors have been identified as being able to mediate binding of infected erythrocytes (IE). In addition the main ligand on the IE surface for adhesion has been identified as a variant surface protein, PfEMP1, such that antigenically variant parasites have different repertoires of host receptor usage. Host endothelia have differential receptor expression, for example cerebral endothelium has little or no CD36 which could influence parasite sequestration, limiting it to IE able to bind to other receptors.

Recently however sequestration of IE to brain endothelium by CD36-binding variants
has been demonstrated in paediatric cerebral malaria through bridging by platelets. Our recent work has shown that levels of von Willebrand Factor (vWF) are elevated in malaria, indicating specific endothelial activation and providing a mechanism for platelet accumulation on endothelium.

The aims of this study are;

i) To confirm the increased expression of vWF in malaria.

ii) To elucidate its potential role in the platelet bridging adhesion mechanism, including vWF multimerisation and proteolytic turnover by ADAMTS13.

iii) To examine whether direct adhesion of IE to vWF takes place and, if so, its significance in paediatric disease.

Programme(s)

Topic(s)

Call for proposal

FP6-2002-MOBILITY-5

Funding Scheme

EIF - Marie Curie actions-Intra-European Fellowships

Coordinator

LIVERPOOL SCHOOL OF TROPICAL MEDICINE

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