EXPRESSION OF CD36 ON ERYTHROCYTES AND ITS INVOLVEMENT AS A ROSETTING RECEPTOR FOR PLASMODIUM FALCIPARUM-INFECTED ERYTHROCYTES

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Plasmodium falciparum-infected erythrocytes (PRBC) can adhere to uninfected erythrocytes (RBC) to form rosettes. PRBC can also adhere to endothelial cells (EC) and to the EC surface antigen CD36. These adherence phenomena have previously been considered quite different. We show that monoclonal (Mab) antibodies that react specifically with CD36 antigen, reverse rosetting of both culture-adapted parasites and natural isolates. Three Mabs that reverse rosetting by greater than 50% at less than 1µg/ml (OKM5, OKM8, 8A6) also block adherence of PRBC to Ec Or C32 melanoma cells. Two other anti-CD36 Mabs (lBl, lD3) failed to reverse rosetting and also to block PRBC adherence to EC. Rosettes were disrupted and the RBC and PRBC pretreated separately with antibodies before mixing to allow rosette formation. Only pretreatment of RBC had an effect. Pretreatment of Mab8A6 blocked rosette reformation while MablBl pretreatment did not. Rosetting was also reversed by purified human platelet CD36. FACS analysis with the anti-CD36 Mabs consistently detected a low level of CD36 expression on the majority of normal RBC. This levels of expression is sufficient to mediate adherence of RBC to anti-CD36 Mabs coated on plastic. In view of the wide distribution of CD36 receptors among wild isolates of P.falciparum, CD36 on RBC may commonly act as a host receptor for rosetting.