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Mutation in Cd36gene protects from malaria

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Erythrocytes infected with *Plasmodium falciparum* adhere to host CD36 receptors expressed on endothelial cells, platelets and leucocytes. This process is thought to benefit parasite survival in malaria. In the 12 May Lancet, Arnab Pain and colleagues from the Institute of Molecular Medicine in Oxford, UK report a mutation in the *Cd36* gene that is associated with protection from severe malaria.

Pain *et al.* sequenced DNA from two African-Americans who did not express CD36 on their platelets and identified a point mutation from T to G in the Cd36 gene at nucleotide 188 in exon 10. Looking at the influence of this mutation on the outcome of severe malarial infection in 693 African children, they found that heterozygosity (for the Cd36 gene) is associated with protection from severe disease (*Lancet*, 2001, **357**; 1502-1503).

The mutation may influence the expression of CD36 on the endothelium and reduce the sequestration of malaria-infected erythrocytes. But altered function of CD36 probably exists as a balanced polymorphism in malaria-endemic areas and might be associated with susceptibility to other diseases.

References

1. Pain A, Urban BC, Kai O, Casals-Pascual C, Shafi J, Marsh K, Roberts DJ: A non-sense mutation in *Cd36*gene is associated with protection from severe malaria. *Lancet* 2001, 357:1502-1503., [http://www.thelancet.com]

2. The Institute of Molecular Medicine, [http://www.imm.ox.ac.uk/]

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