Glucose-6-phosphate dehydrogenase deficiency and Southeast Asian ovalocytosis in asymptomatic Plasmodium carriers in Sumba island, Indonesia

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Abstract

Glucose-6-phosphate dehydrogenase (G6PD) deficiency and Southeast Asian ovalocytosis (SAO) caused by a 27-bp deletion in the band 3 gene (Band3Delta 27) are well-documented genetic traits resistant to malarial diseases; however, relationships between these traits and asymptomatic malaria infection hitherto had not been investigated. Filter-blotted blood samples were collected from a total of 210 healthy individuals, 100 males and 110 females, aged 6-17 years, in Sumba island, Indonesia, to survey for the presence of Plasmodium parasites, G6PD activity and the Band3Delta 27 mutation. Presence of P. falciparum and/or P. vivax was identified in 25 subjects (11.9%). In all, 24 subjects (11.4%) showed Band3Delta 27 heterozygously. In males and females, eight and nine subjects, respectively, showed G6PD deficiency. There was no significant difference in the prevalence of asymptomatic malaria infection between individuals with or without these traits ($P > 0.05$). No alterations in the prevalence of asymptomatic malaria infection suggest that parasite invasion into erythrocytes is unlikely to be a target phase in which the two polymorphisms demonstrate possible protective effects against malaria.

Keywords: G6PD deficiency, Ovalocytosis, 27-bp deletion in the band 3 gene, Asymptomatic malaria, Indonesia

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