

SEROTYPING OF DUFFY BLOOD GROUP IN SEVERAL THAI ETHNIC GROUPS

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Abstract. Duffy blood groups were serologically investigated in 434 individuals from Black Lahu (N = 54), Shan (N = 62), Lisu (N = 74), Red Karen (N = 112), White Karen (N = 102) and Manni (N = 30) in Thailand. High frequency of Fy^a (0.917-1.0) which is comparable with other Mongoloid populations was observed. The presence of weak-Fy^a antigen was detected in eight individuals of northern ethnic groups.

INTRODUCTION

Duffy blood group was discovered in the middle of the 20th century (Cutbush *et al*, 1950) and an erythrocyte chemokine receptor has been identified as Duffy blood group antigen recently (Horuk *et al*, 1993). The responsible gene for Duffy blood group has been assigned on q22-q23 of the HSA1 (Mathew *et al*, 1994).

Major serotypes of Duffy blood group are Fy (a⁺b⁻), Fy (a⁺b⁺), Fy (a⁻b⁺) and Fy (a⁻b⁻) and several minor variants have been known (Beattie, 1988). Serotyping of Duffy blood group has been carried out extensively in a large number of human populations in the world (reviewed in Roychoudhury and Nei, 1988). It is of our great interest that Duffy antigen serves not only as the chemokine receptor but as a receptor for *Plasmodium vivax* invasion into erythrocytes (Miller *et al*, 1975, 1976). Red blood cells with Fy (a⁻b⁻) phenotype escape from *Plasmodium vivax* infection and, in fact, no *vivax* malaria infections have been identified in Africans with Fy (a⁻b⁻) phenotype (Miller *et al*, 1976). Therefore, it is hypothesized that the phenotype of Fy (a⁻b⁻) is the result of genetic adaptation to *vivax* malaria infection (Smith, 1993).

Southeast Asia is one of the highly endemic

areas of malaria infection with *Plasmodium falciparum* and *Plasmodium vivax*, however, the presence or absence of Fy (a⁻b⁻) phenotype in this region has been left open. Clarifying Duffy phenotypic status in this region is essential to generalize the hypothesis mentioned above. Unfortunately, data of Duffy blood typing are unavailable among the various ethnic groups in malaria endemic Southeast Asia (Roychoudhury and Nei, 1988).

In Thailand, several ethnic minorities are distributed throughout the country. Those who inhabit a part of so-called Golden Triangle are purported to have migrated from Southern China, Myanmar or Lao PDR and are called Hill tribes in Thailand (Anderson, 1993). On the other hand, there are a few nomadic hunter and gatherer groups, called the Manni, in the southernmost Thailand. The climate is tropical/sub-tropical and malaria infections are quite common. In line with population genetic studies of these populations, we carried out a sero-screening of Duffy blood group and report our findings here.

MATERIALS AND METHODS

Samples

A total of 434 blood specimens were collected from 54 Black Lahu, 62 Shan, 74 Lisu, 112 Red Karen and 102 White Karen in Mae Hong Son Province, and 30 Manni in Trang Province in Thailand (Fig 1) after oral informed consent. Several ethnic minorities live in Mae Hong Son Province.

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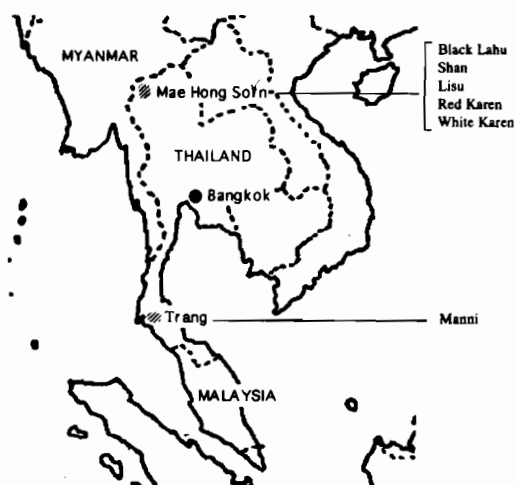


Fig 1—Locality of Thai ethnic groups tested for Duffy blood groups.

They have been classified into three linguistic groups: Sino-Tibetan, Austro-Thai and Austro-Asiatic (Voegelin and Voegelin, 1977; Anderson, 1993). We investigated five of these tribes which belong to the first linguistic group (Voegelin and Voegelin, 1977; Anderson, 1993). On the Malay Peninsula, indigenous populations belonging to Seman/Senoi, known as “Sakai” or “Ngo” in Thailand. They call themselves Manni and are traditional nomadic hunters and gatherers.

Serotyping

Anti-Fy^a and -Fy^b antisera and anti-human globulin containing anti-IgG (Ortho Diagnostic System, NJ, USA) were used. The indirect Coombs test was employed in this screening.

RESULT AND DISCUSSION

Table 1 shows the result of serotyping of Duffy blood group systems in six ethnic groups in Thailand. Fy^a frequency was as high as 0.9 in all the groups. This is fully expected since all the groups tested in this study belong to the Mongoloid ethnic stocks in which Fy^a frequency is predominant (Roychoudhury and Nei, 1988). The frequency of Fy^a varied by group and ranged between 0.917 and 0.995 among northern Thai ethnic groups in Mae Hong Son Province. These frequencies were comparable with those of Hui (0.927), Dong (0.965), Zhuang (0.981) and Tibetans (0.913) in China (Roychoudhury and Nei, 1988; Yuan *et al*, 1984), but differed from that of Thais in Bangkok (0.8875) (Chandanayingyong *et al*, 1979). This difference in frequency is probably due to the ethnic background of their origins. The absence of Fy^b in the Manni in southern Thailand might be explained by a bottle neck effect or a high consanguinity among them. Their population size is extremely small and some

Table 1
Duffy blood group studied in six ethnic groups in Thailand.

Population	Locality ¹	No. tested	Serotype of Duffy ²				Gene frequency	
			a ⁺ b ⁻	a ⁺ b ⁺	a ⁻ b ⁺	a ⁻ b ⁻	Fy ^a	Fy ^b /Fy
Black Lahu	NT	54	49	1	0	4	0.917	0.083
Shan	NT	62	56	3	1	2	0.927	0.073
Lisu	NT	74	68	3	1	2	0.939	0.061
Red Karen ³	NT	112	101	8	3	0	0.9375	0.0625
White Karen ⁴	NT	102	101	1	0	0	0.995	0.005
Manni ⁵	ST	30	30	0	0	0	1.0	0.0

¹NT: northern Thailand (Mae Hong Son Province); ST: southern Thailand (Trang Province).

²Erythrocytes without apparent agglutination but with weak association against anti-Fy^a and -Fy^b antisera were typed as a⁻b⁻. Serotypes of a⁺b⁻ and a⁺b⁺ reflect erythrocytes weakly reacted with anti-Fy^a.

³from two unrelated villages

⁴from three unrelated villages

⁵from two bands

other genetic markers showing monomorphism indicated little genetic heterogeneity (data not shown).

It is well documented that the Fy (a⁻b⁻) phenotype shows resistance to *Plasmodium vivax* infections (Miller *et al*, 1976; Barnwell *et al*, 1989; Chitnis and Miller, 1994). In Africans and Afro-Americans, the Fy (a⁻b⁻) phenotype (FyFy genotype) is common and Fy^a frequency is very low (Sanger *et al*, 1955). The Fy allele in Africans is an Fy^b mutant with transcriptional defect (Tournamille *et al*, 1995). Thus far, no Fy (a⁻b⁻) phenotypes have been confirmed in Mongoloids (Kar *et al*, 1991). We thus emphasize here the presence of erythrocytes which reacted weakly with anti-Fy^a antiserum, (weak-Fy^a) in eight individuals from different ethnic groups in Mae Hong Son Province. A structural alteration in Fy^a antigen or a decrease in the number of Fy^a antigens on the erythrocyte or both account for the presence of weak-Fy^a. Whichever cause is responsible for the weak-Fy^a, it is readily conceivable that the presence of weak-Fy^a provides *vivax* malaria parasites with less opportunity to infect erythrocytes as if weak-Fy^a mimicked the Fy (a⁻b⁻) in Africa. As Fy^a is predominant in frequency among Mongoloid ethnic groups, mutations with selective advantage, such as Fy in Fy^b, might occur in Fy^a in Mongoloids. In this study we serotyped samples according to the recommended criteria, however, the presence of a substantial number of weak-Fy^a and Fy (a⁻b⁻) in northern Thai ethnic groups prompted us to do further characterization of this phenotype by sero- and molecular-testing. Fortunately we obtained several DNA samples from serotyped individuals and preliminary DNA typing was carried out. One individual serotyped as Fy (a⁻b⁻) phenotype was then genotyped as Fy^a positive (unpublished data).

Both serological and molecular Duffy blood group typings are essential in Southeast Asia to clarify (1) the presence or absence of Fy (a⁻b⁻), (2) the heterogeneity of Fy (a⁻b⁻), (3) the presence of weak-Fy^a, and (4) the discrepancy between sero- and molecular-typing. Collectively, screening of Duffy blood groups against records of malaria infection in other ethnic populations of Southeast Asia is desirable to elucidate the role of Duffy antigens in the genetic adaptation to malaria infection.

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