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Hemoglobin E prevalence in malaria-endemic villages in Myanmar.*

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Abstract

The population of Myanmar comprises 8 major indigenous races (Bamar, Kayin, Kachin, Shan, Rakhine, Mon, Chin, and Kayah). The Bamar reside in the 7 central divisions of the country, and the others reside in the 7 peripheral states that border neighboring countries, including China, Laos, and Thailand in the east and India and Bangladesh in the west. Both malaria and HbE are endemic in Myanmar, although the actual prevalence of the latter in the different indigenous races is not yet known. Hemoglobin electrophoresis was performed in 4 malaria-endemic villages, each having a different predominating indigenous race. The overall prevalence of HbE was 11.4% (52/456 villagers), ranging from 2-6% in the Kayin-predominant villages to 13.1-24.4% in the Bamar-predominant villages. Although the overall HbE prevalence in the villages studied was not significantly different from that of the general Myanmar population, this study strongly documented the influence of racial differences on the prevalence of HbE in Myanmar. To prevent and control severe thalassemia syndromes in Myanmar, extensive prevalence studies of the country's indigenous races are suggested.

KEYWORDS: hemoglobin E, malaria, indigenous races, border areas, Myanmar

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Short Communication

Hemoglobin E Prevalence in Malaria-Endemic Villages in Myanmar

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The population of Myanmar comprises 8 major indigenous races (Bamar, Kayin, Kachin, Shan, Rakhine, Mon, Chin, and Kayah). The Bamar reside in the 7 central divisions of the country, and the others reside in the 7 peripheral states that border neighboring countries, including China, Laos, and Thailand in the east and India and Bangladesh in the west. Both malaria and HbE are endemic in Myanmar, although the actual prevalence of the latter in the different indigenous races is not yet known. Hemoglobin electrophoresis was performed in 4 malaria-endemic villages, each having a different predominating indigenous race. The overall prevalence of HbE was 11.4% (52/456 villagers), ranging from 2-6% in the Kayin-predominant villages to 13.1-24.4% in the Bamar-predominant villages. Although the overall HbE prevalence in the villages studied was not significantly different from that of the general Myanmar population, this study strongly documented the influence of racial differences on the prevalence of HbE in Myanmar. To prevent and control severe thalassemia syndromes in Myanmar, extensive prevalence studies of the country's indigenous races are suggested.

Key words: hemoglobin E, malaria, indigenous races, border areas, Myanmar

About 55 million Southeast Asians carry the α thalassemia gene or the hemoglobin E (HbE) gene [1]. Those and other hemoglobin abnormalities are common in malarious areas around the world. Myanmar is no exception, as both malaria and hemoglobinopathies are endemic there. Recent studies highlight not only the innate resistance to malaria conferred by hemoglobinopathies, but also their clinical importance: (i) their effects and influences on the clinical course of the disease [2, 3], (ii) choice of antimalarial drugs [4], and (iii)

their effects on reproductive health and behavior in malaria-endemic areas [5, 6].

While conducting a longitudinal malaria survey, we took the opportunity to study racial differences in the prevalence of hemoglobin E in malaria-endemic villages populated predominantly by either Bamar or Kayin, 2 of the 8 major indigenous races of Myanmar.

Materials and Methods

Study design. A cross-sectional analytical field-based study was conducted. The study was linked to 2 collaborative projects between the Department of Medical Research (Lower Myanmar) and: (i) the World Health

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Organization (WHO); and (ii) the Total Myanmar Exploration Project (TMEP) for longitudinal malaria surveys.

Subjects/patients. A total of 456 unrelated residents of 5 villages were studied.

WHO malaria project. Village A (Oo-Dao): 237 residents (109 males and 128 females, aged 1.5 to 78 years), Hle-Gu Township, Yangon division, 65 km north of Yangon. This small, forested village at the southern edge of the Bago mountain range is populated predominantly by Bamar (Fig. 1).

TMEP malaria project. Village B (Gant-Gaw-Taung): 65 residents (29 males and 36 females, aged 2 to 68 years), Da-Wae Township, Da-Wae State, Southeastern Myanmar. This coastal village is populated predominantly by Bamar. Village C (Mi-Gyaung-Laung): 67 residents (30 males and 37 females, aged 1 to 75 years), same vicinity as village B. This forested village is populated predominantly by Kayin. Village D (Thè-Chaung): 37 residents (25 males and 12 females, aged 2 to 51 years), same vicinity as B and C. This also is a coastal village, and it is also predominantly Kayin. Village E (Ein-Daya-Zar): 50 residents (17 males and 33 females, aged 2 to 65 years), in the vicinity as B, C, and D, is a deeply forested Kayin village (Fig. 2).

Da-Wae is the principal city of Ta-Nin-Thar-Yi division, in southeastern Myanmar. This division in southeastern Myanmar borders Thailand in the east and the Bay of Bengal in the west. Villages B-E are about 50 miles southeast of Da-Wae city.

Blood sampling and handling. Hemolysate was prepared from the cell portion of the whole blood sample (0.5 ml in EDTA). Blood was collected only after informed consent was obtained.

Cellulose acetate hemoglobin electrophoresis. Hemoglobin electrophoresis at pH 8.6 was performed using cellulose acetate paper to detect an HbE carrier, either heterozygote (HbEA) or homozygote (HbEE) as described previously [9].

Statistical Analyses. The student's *t*-test or the Chi-square test was used as applicable. A *P*-value of <0.05 was considered indicative of statistical significance.

Results

HbE was detected in 11.4% (52/456) of the overall study population and was distributed as shown in Table 1.

The prevalence of HbE in the Bamar-predominant populations ranged from 13.1 to 21.5%. The prevalence of HbE in the Kayin-predominant populations ranged from 2.0 to 6.0%. The difference is highly significant statistically ($P < 0.0001$).

Discussion

This study has clearly demonstrated that HbE prevalence differs significantly between the 2 major indigenous races of Myanmar. Four villages with similar malaria

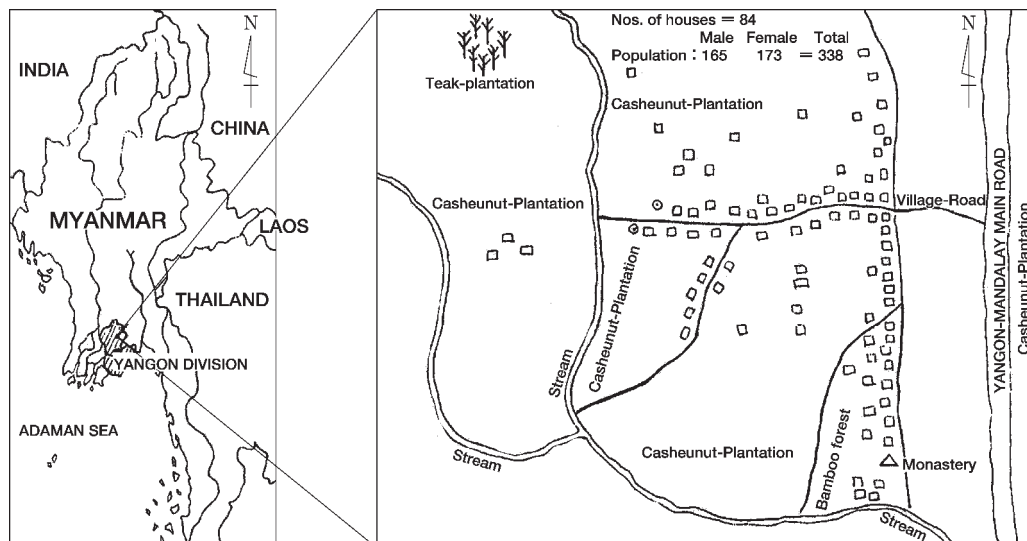


Fig. 1 Map of Oo-Dao study village (Location A).

endemism in a border area showed different rates of HbE prevalence. The striking demographic characteristic among these villages is the different indigenous races that predominate in them, either Bamar or Kayin. The prevalence of HbE is higher in the Bamar populations than in the Kayin, regardless of malaria endemicity. This finding indirectly documents that malaria endemicity is less important than race in the maintenance of a high frequency of HbE in Myanmar.

A previous report studied HbE prevalence in a minor Myanmar ethnic group, the Ao Nagas of the Mokochung District of Naga land, in extreme northeast India [7]. That report found that malaria was an important ecologic factor in maintaining the high frequency of glucose-6-phosphate dehydrogenase (G6PD) deficiency and HbE among the Ao Nagas. However, that report did not compare that group with other major or minor indige-

nous races of Myanmar.

It has been known that hemoglobin E is a thalassemic hemoglobin and that the thalassemias are the most common monogenic diseases in man, with a broad distribution throughout the Mediterranean, the Middle East, the Indian sub-continent, and Southeast Asia including Myanmar [8, 9]. Although in some parts of the world malaria and hemoglobinopathies do not and have never coexisted, selection by malaria remains the force responsible for the prevalence of hemoglobinopathies [1].

Many recent findings have pointed out the great importance of HbE against malaria, including protective effects [1], effects on antimalarials [4], effects on reproductive health [5, 6], and effects on the clinical course of the disease [2, 3]. So the issue of HbE and its role in malaria should be considered highly important, since Myanmar is a multi-racial country (8 major and 137

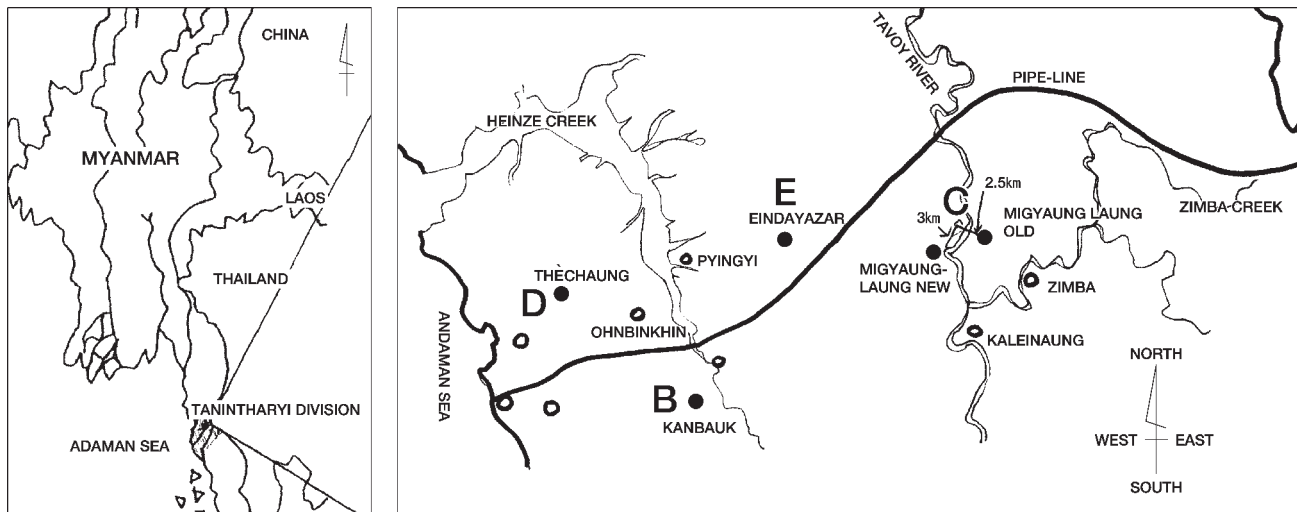


Fig. 2 Map of Coastal villages of Southeastern Myanmar (Location B, C, D, and E).

Table I Distribution of HbE carriers and Hb pattern in different malaria population of 2 indigenous races of Myanmar, Bamar and Kayin (n = 456)

Location (Predominant race)	Number	Hemoglobin Pattern							E carrier (%)
		AA	AF	AE	AH	AEF	EF	EE	
A (Bamar)	237	166	39	23	1	5	1	2	31 (13.1)
B (Bamar)	65	42	9	12			1	1	14 (21.5)
C (Kayin)	67	57	6	3				1	4 (6)
D (Kayin)	37	34		2	1				2 (5.4)
E (Kayin)	50	45	4	1					1 (2)
Total	456	344	58	41	2	5	2	4	52 (11.4)

Location A, Oo-Dao Village; B, Gant-Gaw-Taung Village; C, Mi-Gyaung-Laung Village; D, Thè-Chaung; E, Ein-Daya-Zar Village

minor ethnic groups) with a very high endemicity of both clinical entities.

In conclusion, future detailed clinical, epidemiological, and molecular studies of malaria in relation to thalassemia and prevalence studies in different indigenous races of border areas in Myanmar are suggested. The findings of such studies will be of great value in the national prevention and control programme for severe thalasseмии (congenital red cell disorder) and malaria (acquired red cell disorder) as well.

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References

1. Flint J, Harding RM, Boyce AJ and Clegg JB: The population genetics of the hemoglobinopathies. *Baillieres Clin Haematol* (1998) 11: 1-51.
2. Hutagalung R, Wilairatana P, Looareesuwan S, Brittenham GM, Aikawa M and Gordeuk VR: Influence of hemoglobin E trait on the severity of *Falciparum* malaria. *J Infect Dis* (1999) 179: 283-286.
3. Brown AE, Webster HK, Fucharoen S and Bunyaratvej A: Haemoglobin-E trait and the clinical course of malaria in Thai soldiers. *Eur J Haematol* (1990) 45: 120-121.
4. Hutagalung R, Wilairatana P, Looareesuwan S, Brittenham GM and Gordeuk VR: Influence of hemoglobin E trait on the antimalarial effect of artemisinin derivatives. *J Infect Dis* (2000) 181: 1513-1516.
5. Lisa A, Astolfi P, Degioanni A, DiPasquale C and Zei G: Differential fertility as a mechanism maintaining balanced polymorphisms in Sardinia. *Hum Biol* (1994) 66: 683-698.
6. Astolfi P, Lisa A, Degioanni A, Tagarelli A and Zei G: Past malaria, thalassemia and woman fertility in southern Italy. *Ann Hum Biol* (1999) 26: 163-173.
7. Kar S, Seth S and Seth PK: Prevalence of malaria in Ao Nagas and its association with G6PD and HbE. *Hum Biol* (1992) 64: 187-197.
8. Hill AV: Molecular epidemiology of the thalasseмии (including haemoglobin E). *Baillieres Clin Haematol* (1992) 5: 209-238.
9. Win N, Harano T, Harano K, Myint TT, Mra R, Okada S, Shimono S and Myint: A wider molecular spectrum of beta thalassemia in Myanmar. *Br J Haematol* (2002) 117: 988-992.
10. Lachant NA and Tanaka KR: Impaired antioxidant defense in hemoglobin E containing erythrocytes: a mechanism protective against malaria? *Am J Hematol* (1987) 26: 211-219.