Hemoglobin J in Thais

Abstract. Hemoglobin J in Thailand, a "fast" hemoglobin with an anomaly in its beta chain different from the anomalies previously reported, was the major hemoglobin component in the blood of nine subjects among 1923 Thais from northeastern Thailand. After hemoglobin E, J is the second most frequent of the anomalous hemoglobins among Thais.

A survey was made in 1962 among a group of normal Thai adults from northeastern Thailand to determine the distribution of hemoglobin types and to compare it with the distribution of anomalous hemoglobins in the same population. Among 676 subjects tested, one individual had, in addition to the normal hemoglobin A, another exhibiting the increased anodal mobility characteristic of hemoglobin J (3, 4).

Subsequent studies among members of the individual's family who were living near Nakornratchasima (Korat), in Korat province, northeastern Thailand, revealed an interesting group of individuals with the following combinations of hemoglobins: E, A+J, A+J+L, and J+E (5). Pending completion of our analytical studies, which should establish the exact nature of the structural anomaly, the "fast" hemoglobin from this family has been identified provisionally as J (2, 5).

A survey currently is in progress to determine the relative frequency of occurrence of J in northeastern Thailand. Preliminary results of the study suggest that heterozygosis for hemoglobin J is no means rare.

Blood samples have been analyzed (6) from 1923 Thai adults; almost all the individuals originated from northeastern Thailand, and most of them are residents of Korat province. Hemolyses made from the blood clots (7) were analyzed electrophoretically by the vertical starch-gel method of Smithies (8); the tri-EDTA-borate buffer, pH 9, of Aronson and Crowell (9), at the lower concentrations described by Goldberg (10), was used in the analyses.

In contrast to the first survey, in which just one individual among 676 exhibited A+J hemoglobins, nine individuals or 0.47 percent of the 1923 subjects were heterozygous for hemoglobin J. Among these nine subjects, six had A+J hemoglobins, two had J+E, and one had J alone with an unidentified "slow" hemoglobin with a mobility slightly faster than E and approximately equal to that of D.

All nine subjects, visual inspection of the starch gels indicated that the J component comprised more than 50 percent of the hemoglobin present. Blood samples from an additional 36 subjects among the 1923 studied showed evidence of a fast component apparently identical with J; however, we think additional blood samples from these subjects should be examined before a final decision is made concerning its identity.

The subjects do not represent a random population sample chosen specifically for a survey of abnormal hemoglobin incidence. Nevertheless, they do provide a small sampling from northeastern Thailand. In almost all instances only one member of a family group is included. The size of the sample precludes reliable estimates concerning the incidence of J in various parts of northeastern Thailand; however, its occurrence in approximately 0.5 percent of the entire sample is noteworthy. Our results suggest that the incidence of hemoglobin J in northeastern Thailand may be shown in future detailed studies to be appreciable in some portions of Thailand. It appears quite likely, then, that hemoglobin J, the most frequent anomalous hemoglobin among normal Thais, is also the most frequent among abnormal Thais.

The occurrence of particular anomalous hemoglobins in several ethnic groups may prove to be of some etiological importance. Therefore it is of interest to compare the structural relationship of hemoglobin J with that of the J-type hemoglobins reported previously. Following Thorpe's initial report, hemoglobin J in an American Negro (24), other reports have appeared concerning hemoglobin J in Negroes (11-15), European Caucasians (14-17), Algerians (17), Gurbet Indians (18), tribesmen from northwestern Pakistan (19), Indonesians (20), Chinese (21), and others of obviously mixed ancestry (22). Clearly, not all of the hemoglobins J are identical some are alpha-chain anomalies and others are beta-chain anomalies (11-16; 23). Two of them, J and J, have established structures. The structure for hemoglobin J, found in an American Negro family by Weatherall (13) and in an English Caucasian family by Holman et al. (15), was found by Bagioni and Weatherall (12) to be oB2B4. The same structure was found independently by Holman et al. (15) in their English family. Hemoglobin N, from a French Caucasian family (24), also has a structure identical with that of J and Liddell et al. (16) found that J has an analogous replacement of glycine by aspartic acid at position 15 of the alpha chain: oB4B4; the same structure was reported (25) for hemoglobin J.

Although its precise structural anomaly has not been established, hemoglobin J is different from both J and J; our preliminary work (26) indicates that the anomaly in J resides in the sequence encompassing positions 41 to 59 of the beta chain (trypic peptide BT5), where an aspartic acid replaces either phenylalanine or glycine. This region of the beta chain is also affected (26) in hemoglobin J, a J hemoglobin found in a Halkannese Chinese family in Taiwan (27).

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References and Notes
6. Blood samples were collected at numerous academic institutions and hospitals and were sent to the Central Medical Laboratory in Europe for subse-

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