er chain lengths, similar to the two other patients we have studied (*3*). Recent information on the fatty acid composition of MLD myelin sphingolipids from a 10-year-old patient has been obtained by Norton and co-workers (*12*) who found that cerebroside and cerebroside sulfate contained normal proportions of long-chain fatty acids, but that sphingomyelin was deficient in them. Based on this evidence, the overall deficiency of long-chain sphingolipids in MLD myelin is smaller than originally anticipated. The possibility still remains that myelination does not proceed until sufficient quantities of long-chain sphingolipids are present, and that a deficiency of these molecules in white matter in MLD results in an impairment of myelin synthesis (*3, 11*).

Equally significant, however, are the excessive proportions of cerebroside sulfate in MLD myelin. The presence of excessive proportions of this sulfated galactolipid in the myelin membrane will lead to a preponderance of electronegative sulfate groups at the outer surface of the myelin bimolecular lipid leaflet. Unless these electronegative groups are balanced by cationic groups, such as the amino groups of the structural protein of myelin, or by other cations (calcium, potassium, and other), the surface charge of the myelin lipoprotein will be more electronegative than normal. Thus, the surface charge, the ionic environment, and, consequently, the degree of hydration of the myelin lipoprotein may be abnormal in MLD. These abnormalities in charge and hydration may alter the configuration of the structural protein of myelin in MLD; lead to an increased permeability of the myelin membrane, especially for cations; and impair the packing of adjacent myelin lamellae, owing to mutual charge repulsion or excessive hydration effects, or both (*13*). These molecular defects may contribute to the pathogenesis by impairing the synthesis of the myelin membrane, in turn giving rise to a myelin deficiency; hastening the breakdown of myelin due to instabilities in its molecular makeup; and resulting in a slowing of nerve impulse conduction (*14*), due both to deficient quantities of myelin and to the presence of abnormally constituted myelin which is an inefficient insulator. The chemical abnormalities of MLD myelin may also partially explain the ultrastructural morphologic abnormalities of myelin—especially its loose packing and apparent degradation—seen on electron microscopic examination of peripheral nerves in this disease (*15*). Despite the speculative nature of these conjectures, the disclosure of a molecular abnormality of the myelin membrane in a disorder of myelination indicates that other diseases in which myelination is defective may also involve the formation of chemically abnormal myelin.

JOHN S. O'BRIEN
E. LOIS SAMPSON
Division of Chemical Pathology, Departments of Pathology and Medicine, University of Southern California School of Medicine, Los Angeles

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Hemoglobin J_{Korat} in Thais

Abstract. *Hemoglobin J_{Korat}, 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_{Korat} 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 haptoglobin types (*1*) and to compare it with the distribution of anomalous hemoglobins in the same population (*2*). 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 Nakhornratchsima (Korat), in Korat province, northeastern Thailand, revealed an interesting group of individuals with the following combinations of hemoglobins: E, A+E, A+J, 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_{Korat} (*2, 5*).

A survey currently is in progress to determine the relative frequency of occurrence of J_{Korat} in northeastern Thailand. Preliminary results of the study suggest that heterozygotes for hemoglobin J_{Korat} are by 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. Hemolysates made from the blood clots (*7*) were analyzed electrophoretically by the vertical starch-gel method of Smithies (*8*); the tris-EDTA-borate buffer, *p*H 9, of Aronsson and Grönwall (*9*), at the lower concentrations described by Goldberg (*10*), was used in the analysis.

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 were heterozygous for hemoglobin J. Among these nine subjects, six had A+J hemoglobins, two had J+E, and one had J along with an unidentified "slow" hemoglobin with a mobility slightly faster than E and approximately equal to that of D. For 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_{Korat} 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_{Korat} may be shown in future detailed studies to be appreciable in some portions of Thailand. It appears quite likely that, next to hemoglobin E, J_{Korat} is the most frequent anomalous hemoglobin among normal Thais. It also appears possible that considerable heterogeneity will be found within the Thai people with respect to the incidence of J_{Korat}.

The occurrence of particular anomalous hemoglobins in several ethnic groups may prove to be of some ethnological importance. Therefore it is of interest to compare the structural relationship of hemoglobin J_{Korat} with that of the J-type hemoglobins reported previously. Following Thorup's initial report of hemoglobin J in an American Negro (*3*), other reports have appeared concerning hemoglobin J in Negroes (*11–13*), European Caucasians (*14–16*), Algerians (*17*), Gujerati 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, $J_{Baltimore}$ and J_{Oxford}, have established structures. The structure for hemoglobin $J_{Baltimore}$, found in an American Negro family by Weatherall (*13*) and in an English Caucasian family by Holman *et al.* (*15*), was found by Baglioni and Weatherall (*12*) to be $\alpha_2{}^{A}\beta_2{}^{16Asp}$. The same structure was found independently by Holman *et al.* (*15*) in their English family. Hemoglobin $N_{New\ Haven\text{-}2}$, from a French Caucasian family (*24*), also has a structure identical with that of $J_{Baltimore}$. Liddell *et al.* (*16*) found that J_{Oxford} has an analogous replacement of glycine by aspartic acid at position 15 of the alpha chain: $\alpha_2{}^{15\ Asp}\beta_2{}^{A}$; the same structure was reported (*25*) for hemoglobin $I_{Interlaken}$.

Although its precise structural anomaly has not been established, hemoglobin J_{Korat} is different from both $J_{Baltimore}$ and J_{Oxford}; our preliminary work (*26*) indicates that the anomaly in J_{Korat} resides in the sequence encompassing positions 41 to 59 of the beta chain (tryptic peptide β T5), where an aspartic acid replaces either phenylalanine or glycine. The same region of the beta chain is also affected (*26*) in hemoglobin $J_{Meinung}$, a J hemoglobin found in a Hakkanese Chinese family in Taiwan (*27*).

R. QUENTIN BLACKWELL
BOON-NAM BLACKWELL
JEANETTE TUNG-HSIANG HUANG
LI-CHIN CHIEN
U.S. Naval Medical Research Unit 2
Taipei, Taiwan, Republic of China
ARKOM SAMAHARN
Central Medical Laboratory,
Nakhornratchsima, Thailand
CHAMRAS THEPHUSDIN
CHATRA BORVORNSIN
Royal Thai Army Institute of
Pathology, Bangkok, Thailand

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Sterols and Temperature Tolerance in the Fungus Pythium

Abstract. *A fungus of* Pythium *species survives high and low temperatures longer when a suitable sterol is added to the growth medium.*

Certain sterols are required for sexual reproduction of pythiaceous fungi (*1*) and can stimulate their growth (*2*). Evidence now indicates that there is yet another role for sterols. *Pythium* sp. PRL 2142 (*3*) survives high temperatures longer when provided with a suitable sterol (cholesterol, β-sitosterol, and others) than it does when such a sterol is not available. Somewhat comparable effects were noted at the low temperature as well. The temperature at which this organism dies was affected not only by the presence of a sterol (that is, the sterol affects the nutritional state) but also by the speed with which the organism was heated to the critical temperature.

Agar cultures of the *Pythium*, with and without cholesterol, were presumed killed when kept several hours at