HEMOGLOBIN VARIANTS IN INDIA

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The occurrence of different types of hemoglobin variants in India has been well-documented in urban populations and in certain tribal areas. Beta-thalassaemia and hemoglobins D, E, H, S, and rare variants like Hb Lepore, have been found in varying percentages. There is a higher incidence of Hb E in the Eastern region and of Hb D in certain Northern populations. The association of Hb E with beta thalassaemia G6PD deficiency has been established. The relationship with other environmental factors, like malaria, is not yet clear.

INTRODUCTION

The most extensive data on genetic anomalies available in India are those on different types of hemoglobin variants. They include both case reports and population studies, using techniques ranging from chemical to electrophoretic ones (Sharma et al. 1976).

A significant amount of the available data is from tribals and other small isolated populations in different parts of the country, and may be regarded as fairly representative of the populations under study. The reports from hospitals, however, are not representative in as far as population studies are concerned, since such hospitals are located in urban areas and the cases admitted are necessarily heterogeneous. Family studies have also been carried out in certain regions, and to some extent give a comprehensive picture.

Apart from thalassemias, which have been reported all over India, the other hemoglobin variants so far observed in this country include Hb D, E, H, J, K, L, M, S, Lepore, Norfolk, and the hereditary persistence of Hb F.

Techniques ranging from chemical to electrophoretic and microchromatographic ones have been used by the various authors for the detection of the thalassemia trait and hemoglobin variants. Peptide analysis and finger printing for protein have also been employed.

In India, as in other countries with records of both thalassemia and hemoglobin variants, both may be inherited in certain cases, leading to genetic interaction and enhanced production of hemoglobin variants with consequent clinical disorders of varying severity. Sometimes, in such double heterozygotes, hemoglobin variants may coexist without genetic interaction as well. Such genetic interaction and their clinical expression are influenced by several factors, including environmental ones.

GEOGRAPHIC DISTRIBUTION OF HEMOGLOBIN VARIANTS IN INDIA

Several hemoglobin variants were first detected in individuals of Indian origin, such as Hb D, J, K, L, and Koya Dora Rampa. Hemoglobin variants are frequent in many racial and tribal groups of India over wide areas, though Sinha et al. (1973) report their total incidence in the general population of India as less than 0.5 per cent.

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The results of all published surveys to date are presented in the appendix (Table and map). Earlier reviews have given details of isolated case reports and histories, and these will not be repeated here (Chatterjea 1966, Saha and Banerjee 1973, Sukumaran 1975).
Considering India and its neighboring countries with populations of a common origin, the various forms of hemoglobin variants may be classified as follows:

(1) Hemoglobin variants of universal occurrence;
(2) Hemoglobin variants of limited occurrence;
(3) Hemoglobin variants of rare occurrence.

Most of the major hemoglobin variants, namely, S, E and D, are widespread or occur in several subpopulations in different parts of the country. Of relatively rare occurrence are Hb H, J, K, L, M, Q, and Hb Lepore and Norfolk.
Of the thalassemias, β-thalassemia major, easier to detect, has been recorded all over the country, whereas β-thalassemia minor, the heterozygous state, has been reported mainly in northern and eastern India.
α-thalassemia minor — the heterozygous state of a condition in which the α-chain synthesis is reduced, depressed, or absent — is expressed as increased Hb Barts in cord blood and has, as such, been recorded sporadically. Thalassemia has been observed in conjunction with other hemoglobin variants as well, such as S-thalassemia, E-thalassemia, D-thalassemia, J-thalassemia, K-thalassemia and Q-thalassemia in varying frequencies in different regions (Mitra 1978). Hereditary persistence of fetal hemoglobin occurred in several cases, as, for example, the 13 instances with 3 double heterozygotes for Hb F and thalassemia major observed by Sukumaran et al. (1961). Chatterjea (1966) recorded a similar interaction in two families from Bengal; Barkhan and Adinolfi (1962) in a family of mixed Indian and Portuguese ancestry; Bird et al. (1964) in a male in Poona, and Parekh et al. (1963) in families in Bombay.

POPULATION STUDIES

Hemoglobin S

Both sickle cell anemia and the sickling trait are recorded in significant frequencies in endogamous aboriginal tribals in different parts of India, except the northwest and extreme south. For other population groups, scattered reports are available.
The Pre-Dravidian aboriginal tribes from the Nilgiri hills show Hb S in frequencies varying from 3 to 30 per cent in different subpopulations. The Dravidian nonaboriginal tribes of the same area, surprisingly however, indicate an absence or very low frequency of the trait. The percentage of incidence reported by different workers amongst the former, from time to time for the same subgroups, may vary, but all reports agree in the absence of Hb S amongst one aboriginal tribe, the Kotas (Chaudhuri et al. 1964*a*, *b*, Kirk et al. 1962*a* and 1962*b*, Lehmann 1954, Lehmann and Cutbush 1952, Lehmann and Sukumaran 1956). The Paniyans of the same region have a very high incidence (265 in 955: Das et al. 1967; 22 in 74: Büchi 1955). In Orissa, Andhra Pradesh, Bihar, Madhya Pradesh and Gujarat, the tribals have shown relatively high figures for Hb S, though its absence has also been reported (Das et al. 1967, Kumar and Ghosh 1967, Negi 1962, 1963 and 1964; Roy and Roy Chaudhuri 1967, Parikh et al. 1969, Sarup and White 1974, Sharma J.C. 1968, Sukumaran 1975 and 1978, Vyas et al. 1962). The tribals from Assam show a similarly high frequency of this trait (Batabyal and Wilson 1958), though Flatz et al. (1972) do not consider it to be characteristic for the

autochthonous populations of Assam. Population surveys of nontribals from the same region seldom show a frequency of Hb S trait higher than 10 per cent, particularly amongst the upper castes (Bhatia et al. 1955, Chaudhuri et al. 1963, Kumar and Ghosh 1967, Lele et al. 1962, Mital et al. 1962, Sanghvi 1962, Sayed and Amin 1966, Shukla and Parande 1956, Shukla and Solanki 1958, Sukurmaran and Master 1974, Sukumaran et al. 1956). Very low incidence or complete absence has been observed in some cases, e.g., in the Cutchi: in Bombay (9/599: Mehta et al. 1972); in the Goans (1/379: Trincao et al. 1963); in Uttar Pradesh, Maharashtra, Kerala and Andhra Pradesh (absent: Dube et al. 1959, Parekh et al. 1967, and Reddy and Baruah 1964); and in Bengal (1/10,000: Chatterjea 1959). The Parsis in Bombay showed an incidence of Hb S of 5/1290. The Iranis of the same region, though having a common origin, showed a complete absence (Undevia 1969). Muslims from Bangladesh, having a presumably common ancestry with Muslims of West Bengal, had an incidence of 2/424 (Swarup and Chatterjea 1958) while the incidence in West Pakistan was found to be 1/76 (Lehmann et al. 1961). The latter is unusual, since no report is available for Muslims of northwest India.

The presence of Hb S in higher frequencies in tribals than in the non-tribals of the same region presents an interesting problem. It would, however, be preferable to obtain more data on the nontribals before drawing any conclusions.

Hemoglobin E

Hemoglobin E trait occurs frequently in combination with β-thalassemia disease and trait. Their distribution is widespread, though reports are more frequent from eastern India and Bombay (Chatterjea 1970). Some scattered observations of Hb E trait are available from Uttar Pradesh as well (Atal and Mital referred to in Chatterjea 1966, Gupta et al. 1970, Mathur et al. 1962, Mehrotra et al. 1968, Pande et al. 1972).

The incidence of Hb E is higher in Assam, amongst the Assamese, Ahoms and Khasis, and shows a significant association with Hb EE (Das et al. 1971, Flatz et al. 1972). The Toto tribals gave 23 cases of Hb E trait out of 116 (Chaudhuri et al. 1964). Bhutan has a lower frequency (2/31: Glasgow et al. 1968). Hb E was suggested as a marker for the Mongoloid element in northeast Indian populations (Das and Flatz 1975, Flatz 1978, Flatz et al. 1972; Seminar on Human Variation in Calcutta: 29 June - 2 July, 1977).

However, data from Calcutta hospitals are more variable and do not give any definite relationship between the Hb E trait and different subpopulations or castes (Bhattacharjee 1956, Chaudhuri et al. 1967*a*, Chatterjea et al. 1957). The variability may be due to the ethnic heterogeneity of the cases studied. Refinements in method of detection may further increase the frequency of incidence of the heterozygous state (Ajmani et al. 1976, 1977, Sharma et al. 1976).

β-thalassemia major has been reported in almost all States of western India (Mehta et al. 1973, Sharma R.S. et al. 1963, Udani et al. 1961). Cases of β-thalassemia as well as Hb E trait have been observed in Uttar Pradesh, Sind and Punjab, and also in immigrants of Indian origin abroad (Saha 1970, Saha and Banerjee 1971, Sen 1960, Siddoo et al. 1956, Vella 1957 and 1962, Vella and Field 1958). Amongst the armed forces personnel as well, a higher frequency of the Hb E trait is seen in the recruits from the eastern zone. The Hb E trait persists amongst the neighboring countries in populations with known common origin. For example, 25 Hb E trait and 50 E-thalassemia cases were diagnosed in 424 samples of Bengali Muslims

in Bangladesh (Swarup et al. 1960). In Ceylon, Veddahs from different areas have different degrees of Hb E, occurring frequently with Hb EE (Aksoy et al. 1955, Graff et al. 1954, Kirk et al. 1962*b*, Nagaratnam et al. 1971, Wickremasinghe et al. 1963). The preponderance of Hb E-thalassemia over β-thalassemia is explained by the fact that it is in general milder than the latter and the subjects live longer.

The overall incidence of β-thalassemia and Hb E trait suggests that their frequency in this country may be higher than earlier estimated and may be revealed by a thorough survey.

Hemoglobin D

Hemoglobin D has a relatively low incidence, taking the country as a whole, and a tendency to cluster in particular populations. It is most frequent in the northwestern part of India, particularly amongst the Sindhis, Sikhs and Punjabis, having occasional association with Hb DD (Bird and Lehmann 1956, Bird et al. 1956, Chaudhuri et al. 1967*b*, Dutta et al. 1972, Gupta et al. 1972, Jain 1971, Saha and Banerjee 1971). Other reports from Gujarat, Goa, Uttar Pradesh, Mysore, Bombay, etc., indicate mainly sporadic cases, possibly arising through mutation (Hakim et al. 1972, Jacob et al. 1956, Mehrotra et al. 1968, Pande et al. 1972, Raper 1957, Saha 1970, Sen 1960, Sinha et al. 1973, Swarup et al. 1959 and 1966*a*, Trincao et al. 1963, Vella 1962). A pocket of Hb D in a Sindhi community in Maharashtra was detected. Three members were homozygous and 12 out of 238 individuals carried the Hb D trait (Phadke 1977). This group had migrated to this area from Sindh-Punjab which has a high incidence of Hb D. The Lohanas of Gujarat possibly have some ethnic affinity with Punjabi Khattris (Thakur 1959) and may have acquired the Hb D gene through them. Association with thalassemia has been observed in certain cases (Sukumaran et al. 1960), reported mainly in individuals from the north-western regions. Amongst the neighboring countries, in north-western Pakistan, there is both a high incidence of Hb D trait and association with Hb JD (Bolton et al. 1964, Stern et al. 1968).

Other Hemoglobin Variants

For the other hemoglobin variants, including Hb Punjab, K, L, M and Q, a screening of the armed forces gives an overall incidence of 0.96 per cent (Chandrasekhar et al. 1974, Das et al 1975, Sharma N.P. et al. 1976, Sinha et al. 1973). Hb Norfolk has been recorded in Gorkhas of the lower Himalayas (Mehrotra et al. 1975). Hb J with both α- and β-chain variations has been diagnosed in Gujarati Lohanas, Gujarati immigrants in Uganda and also Bengalis and Punjabis (Hakim et al. 1972, Raper 1957, Sanghvi et al. 1958, Subhedar et al. 1961, Sukumaran et al. 1969, Swarup et al. 1966*b*), No definite association with a subpopulation or region has yet been established, though the highest frequency appears to be shown by the Gujaratis. A report on Gorkhas from Nepal showed a frequency of 1.33 per cent hemoglobin variants in 1198 individuals, with 5 Hb AE, 4 Hb AD, 5 Hb A Norfolk and 2 Hb AJ (Gupta et al. 1977).

Three reports of Hb K from Bengal, Madras and Pondicherry indicate its possible distribution on the eastern coast (Labie et al. 1961, Swarup et al. 1963). Hb L shows a sporadic occurrence among Punjabi Khattris and Gujaratis. Records of α-thalassemia, Hb Barts and Hb H are fragmentary, mainly from large referral hospitals in Bombay and Calcutta (Chouhan et al. 1970, Nagaratnam and Sukumaran 1967, Parekh et al. 1967, Sukumaran and Master

1974, Swarup et al. 1965, Vora et al. 1975). A comprehensive survey is required for a complete picture. Data on Hb Lepore are meagre too, only one report being available (Chouhan et al. 1971). These scattered instances of rare hemoglobin variants may have originated through independent mutations.

RELATIONSHIP WITH OTHER CONDITIONS

The possibility that hemoglobin variants may represent an advantage in heterozygotes in some cases has been debated at length (Allison 1954, Edington and Lehmann 1956, Raper 1955), the classic instance being that of hemoglobin S heterozygotes having a higher survival rate after infection by *Plasmodium falciparum* malaria than normal homozygotes in Africa. In India, experiments following inoculation with *P. vivax* and *P. falciparum* of Hb E-thalassemia double heterozygotes gave a certain amount of resistance to *P. vivax* amongst the carriers in comparison with control (Chatterjea 1964, Ray et al. 1964). The results, however, are inconclusive, particularly in view of the fact that Hb E carriers in Thailand do not show a similar resistance to *P. vivax* (Kruatrachue et al. 1962, 1969). However, Flatz (1967, 1978) and Kruatrachue et al. (1969) have suggested an advantage of Hb E gene (Hb β E) in a malarious environment from distribution and parasitemia studies. In Assam, where the highest frequencies of this gene were found, malaria had been said to have been nearly eliminated after World War II.

In evaluating the Hb β E frequencies, a competing effect of Hb β T should be considered. In southeast Asia, a reciprocal distribution has been suggested of these two genes in different population subgroups (Flatz et al. 1965, Wasi et al. 1967). In Assam, Flatz et al. (1972) have indicated a similar reciprocal relationship. The high frequency in the Ahoms (0.3488) — a Thai group related to the Shan in Burma — possibly indicates the gradual elimination of Hb β T by Hb β E in a favourable environment. Frequencies of Hb E in other Assamese subgroups are high as well, indicating that Hb β E may have spread to other Mongoloid groups on the southern slopes of the Himalayas, after its introduction by migration from southeast Asia in prehistoric times.

Hb S has not been shown to have any definite correlation with malaria, unlike its counterpart in Africa. Its occurrence, principally amongst the tribals in west and central India, does not correspond, as yet, to any specific environment, particularly since nontribals in the same area, as mentioned before, often have a very low frequency of the trait. Its incidence amongst the tea-garden workers in Upper Assam (Batabyal and Wilson 1958, Dunlop and Mozumdar 1952) has been ascribed to the origin of these populations mainly in Orissa, where Hb S is not uncommon, followed by migration to Assam (Roy and Roy Chaudhuri 1967). Flatz et al. (1972) have suggested that the Hb S gene is being introduced into the general Assamese population due to the current lowering of social barriers.

A positive correlation between the frequencies of glucose-6-phosphate dehydrogenase deficiency — another prevalent genetic disorder in India — and hemoglobin variants, has been recorded in southeast Asia and mediterranean populations (Allison et al. 1963, Flatz et al. 1965, Siniscalco et al. 1961). The data from India are as yet insufficient for any generalisation. In Assam, the frequency of G6PD deficiency is lower than that of abnormal hemoglobins. In a relatively recent survey, Misra et al. (1976) found 8 of 42 cases of thalassemia studied (19%) to be G6PD deficient. Since G6PD deficiency exists in adult populations nearly all over India in frequencies varying from less than 1 to 15.8 per cent (Azevedo et al.

1968, Bapat and Baxi 1973, Baxi et al. 1961, Chatterjea, 1966; Dube et al. 1976, Mehta et al. 1971), no significant correlation can be established with any particular hemoglobin variant as yet.

Family studies on hemoglobin variants in India have been many and the pattern of inheritance has been traced in most cases (see earlier reviews by Chatterjea 1966, Saha and Banerjee 1973, Sukumaran and Master 1974, Talukder and Sharma 1978, Verma 1978, for details). Consanguinity is not an important factor in most of India, consanguineous marriages being mainly confined to certain regions, like Andhra Pradesh, and to certain subpopulations. Observations, in such cases, mainly involve studies on sterility, mental retardation, blood groups and, less frequently, congenital defects (Chakravartti and Chakravartti 1977, John and Jayabal 1971, Joshua 1974, Majumdar 1972, Reid 1976, Sunder Rao et al. 1971).

FUTURE WORK

Hemoglobin variants, though relatively extensively studied in this country, yet have scope for a great deal of further research. Most of the data represent case reports or population studies of small *ad hoc* groups. Surveys for the screening of hemoglobin variants are few, considering the dimensions, multiracial and multienvironmental, of the country. The major problem faced in surveys is primarily the lack of adequate birth, marriage and death records in rural areas — which harbour 80 per cent of the general population. The available records are principally from urban referral hospitals, where the samples are necessarily heterogenous, and from small tribal groups where anthropological measurements have been carried out. An additional limitation is the lack of simple screening methods for the heterozygous state of most abnormal hemoglobins, as is available for Hb S.

A thorough survey for the entire country is required, dividing it into distinct zones and screening the populations for hemoglobin variants. The populations may be further divided into ethnic groups, particularly into inbreeding subpopulations on the basis of religion, or caste, wherever available. Family data will be analysed after identifying individual cases of hemoglobin variants.

The effect of, and relation with, various environmental factors, are as yet uncertain. The data, wherever available, are fragmentary and ambiguous. The interaction of an abnormal gene with other genes and with climate, nutrition, geographical and other environmental parameters requires a thorough analysis. Screening programs should therefore be developed in collaboration with population geneticists. Such programs would need carefully planned sampling techniques with a detailed understanding of the historical, demographic and genealogical relationships of people living in different environmental conditions.

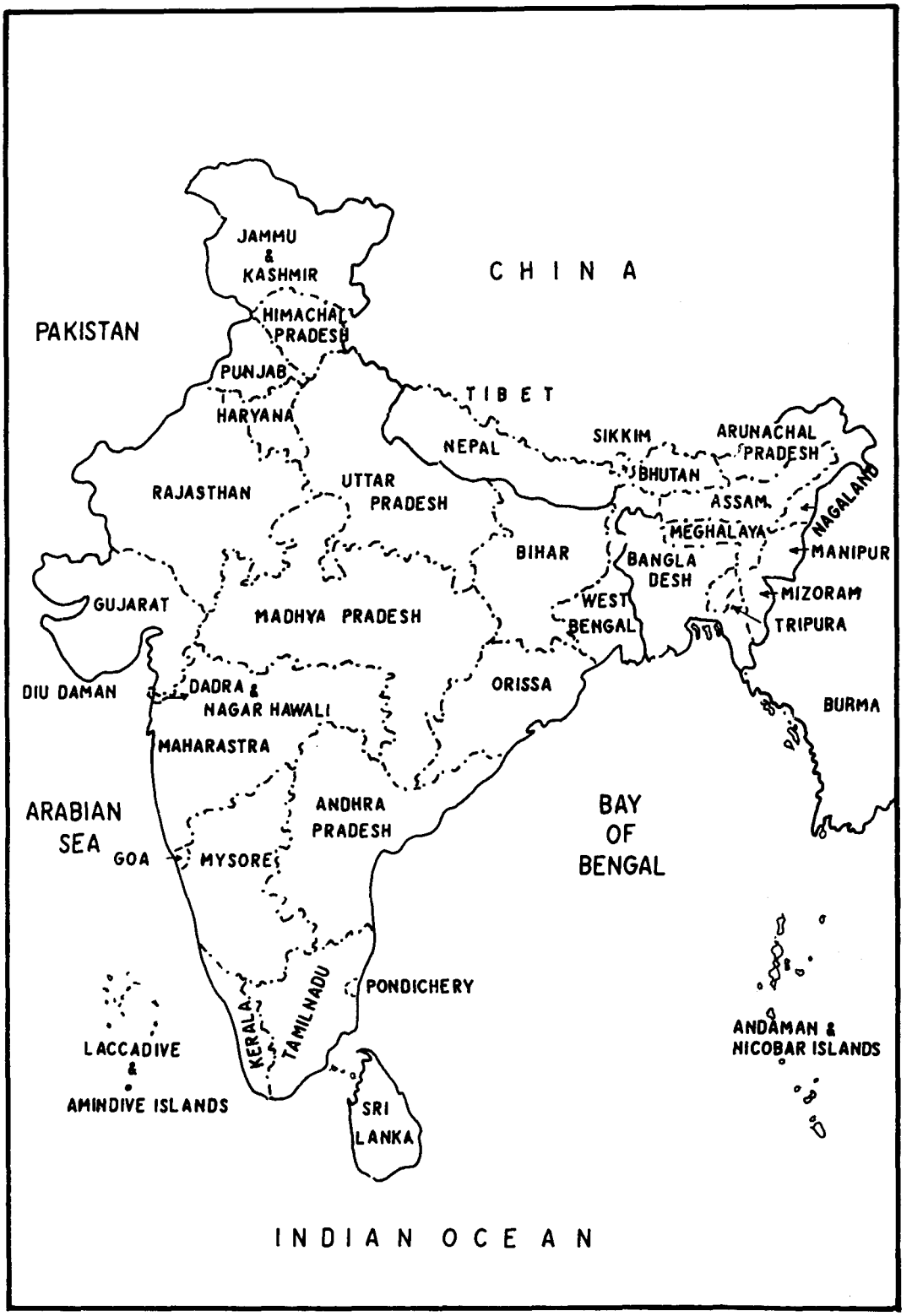

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Table. Distribution pattern of abnormal hemoglobins in different populations from the Indian subcontinent

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
HEMOGLOBIN D						
Punjab	Sikh	109		1		Bird and Lehmann (1956)
		62		1		
Northwest India	Sikh	279	1	4		Bird et al. (1956)
Uganda	Gujaratis	174		2		Jacob et al. (1956)
	Gujaratis and a few Sikhs and Punjabis	326		2		Raper (1957)
Mysore (Dist. Shimoga)	Indians	618		1	Harijan	Swarup et al. (1959)
Singapore and Malaya	Indians	2500		13		Vella (1957)
U.K.	Bengalis	564		2	1 Brahmin 1 Vaidya	Sen (1960)
Gujrat	Lohanas	603		3		Sanghvi (1962)
	Brahmins	203		—		
Singapore and Malaya	Indians	3341		15		Vella (1962)
Goa	Indians	1843		5		Trincao et al. (1963)
Diu	Indians	379		2		
Northwest Pakistan	Brahuis	4		1		Bolton et al. (1964)
	Baluchis	9		2		
	Pathans	18		1	Hb JD	
Ludhiana	Sikh Punjabi Hindus	125		3		Saha and Banerjee (1965)
		225		1		
Calcutta	Bengalis	10000		9		Swarup et al. (1966*a*)
				1	Hb DE	
				6	Hb D-β-thal	
	Sikhs	427		5		Chaudhuri et al. (1967*b*)
Uttar Pradesh	Indians	755		2	Hindu Thakur	Mehrotra et al. (1968)
Northwest Pakistan	Pathans	129		1		Stern et al. (1968)
Allahabad	Indian armed forces personnel	1378		1	Dogra	Pande et al. (1970)
Singapore	Indians	143		2		Saha (1970)
Singapore and Punjab	Gujaratis and Sindhis	150		1		Saha and Banerjee (1971)
	Sikhs	378	1	5		

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Table - *Contd.*

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
Singapore and Kerala	Tamils	1310		1		Saha and Banerjee (1971)
Singapore	Hindus	123		1		
Uttar Pradesh	Indigenous population of the state	709		3		Gupta et al. (1972)
				1	Hb D-thal	
Bombay	Mixed Muslims	854		2		Hakim et al. (1972)
Allahabad	Blood Bank donors	535		2	1 from Punjab 1 from Madhya Pradesh	Pande et al. (1972)
	Indian armed forces personnel	2075		13	5 from other Indian castes (one each from Jammu and Kashmir, Kerala, Maharashtra, Mysore and Uttar Pradesh) 6 Brahmins (From Delhi, Orissa, Punjab, Uttar Pradesh) 1 Khatri 1 Christian from Orissa	Sinha et al. (1973)
				1	Hb D-thal	
	Pakistani armed forces personnel	5000		21		Sharma N.P. et al. (1976)
HEMOGLOBIN E						
Ceylon	Veddahs	9		2		Graff et al. (1954)
		158		3		Aksoy et al. (1955)
Hooghly	Rahri Brahmins	100		—		Bhattacharjee (1956)
	Muslims	100		—		
Calcutta	Bengalis	7000	2	25		Chatterjea et al. (1957)
Bangladesh	Bengali Muslims	424		25		Swarup and Chatterjea (1958)
				50	Hb E-β-thal	
Singapore	Gorkhas	560		3		Vella and Field (1958)
Calcutta	Nepalese	109		1		Chatterjea (1959)
U.K.	Bengalis	564		10	7 Kayastha 1 Brahmin 1 Vaisya 1 Muslim	Sen (1960)
			1		Brahmin	
				1	Hb E-β-thal Brahmin	

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Table - *Contd.*

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
Karachi	Bengali Muslims	23		2		Lehmann et al. (1961)
Ceylon	Veddahs	30	2	14		Kirk et al. (1962*b*)
Uttar Pradesh	Indians (anemic children)	512		1		Mathur et al. (1962)
Malaya and Singapore	Indians	3341		15		Vella (1962)
Pollebedda (Central Ceylon)	Veddahs	38	1	4		Wickremasinghe et al. (1963)
Dambana (Central Ceylon)	Veddahs	27		—		
Linidamana (Central Ceylon)	Veddahs	87	2	26		
Adampore (North Ceylon)	Veddahs	32		5		
Totopara (Assam)	Totos	116		23		Chaudhuri et al. (1964*a*)
Uttar Pradesh	Indians	97		1		Atal and Mital (quoted by Chatterjea 1966)
Calcutta	Sikh	427		1		Chaudhuri et al. (1967*b*)
Uttar Pradesh	Indians	755		1	Hindu Brahmin	Mehrotra et al. (1968)
Luana and Thimpu (Bhutan)	Bhutanese	31		2		Glasgow et al. (1968)
	Bengalees	235		—	Brahmins	Chaudhuri et al. (1967*a*)
Calcutta		229 129		6 1	Kayastha Vaidya	Chaudhuri et al. (1967*b*)
Singapore	Indians	143		1		Saha (1970)
Uttar Pradesh	Indians	1217		3 3	 Hb E-α-thal in one family of indigenous population of the state	Gupta et al. (1970)
Allahabad	Indian armed forces personnel	1378	 1	3	2 Kayastha 1 Khatrya Assam tribal	Pande et al. (1970)
Assam	Khasis Ahoms	80 82	2 11	31 37		Das et al. (1971)
Singapore and Punjab	Gujaratis and Sindhis	150		1		Saha and Banerjee (1971)
Singapore and Kerala	Tamils	1310	2	6		
	Malayalis	314		2		

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Table - *Contd.*

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
Allahabad	Blood Bank donors	535		1	Bengali	Pande et al. (1972)
Assam	Assamese	182	5	29		Flatz et al. (1972)
	Ahoms	129	15	60		
	Khasis	140	6	51		
	Kacharis	5	1	4		
Allahabad	Indian armed forces personnel	2075		3	2 Assamese 1 Rajpur	Sinha et al. (1973)
			3		2 Assamese 1 Bengali Brahmin	
24-Parganas (West Bengal)	Kaoras	102		1		Das et al. (1971)
Dibrugar and Sibsagar in Assam	Assamese	315	10	55		
	Ahoms	399	40	181		
	Kacharis	555	145	276		
Allahabad	Pakistani armed forces personnel	5000		9	2 Punjabis 5 from Northwest Frontier Province 2 from Pakistan occupied Kashmir	Sharma N.P. et al. (1976)
			1		from Pakistan occupied Kashmir	
HEMOGLOBIN S						
Nilgiri Hills of South India	*Pre-Dravidians* Aboriginal tribes:					Lehmann and Cutbush (1952)
	Badagas	191		16		
	Todas	60		2		
	Irulas	80		24		
	Dravidians Non-aboriginal tribes:					
	Telegus	109		—		
	Malayalis	110		—		
	Canarese	95		—		
	Tamils	128		—		
Nilgiri	Paniyans	61		21		Lehmann (1954)
	Kurumbas	16		3		
	Kotas	86		—		
	Irulas	124		39		
	Badagas	191		26		
	Todas	84		3		
Nilgiri, Wynad Tinnevelly	Paniyans	74		22		Büchi (1955)
	Pallars	112		1		
Manipur	Dhanukh caste	335		32		Bhatia et al. (1955)

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Table - *Contd.*

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
Nilgiri	Kurumbas	26		7		Lehmann and Sukumaran (1956)
	Kotas	22		—		
	Badagas	30		2		
	Irulas	18		4		
	Todas	50		1		
Panch Mahal	Bhils	206		32		Sukumaran et al. (1956)
Surat	Dhodias	107		22		
	Dublas (Talavia)	211		20		
	Kolis	51		—		
	Naikas or Naikadas	90		20		
	Anavil Brahmins	53		—		
Kheda	Leva Patidars	150		—		
Bombay	Marathas	201		—		
	Mixed group	222		—		
Assam	Oriahs	100		15		Batabyal and Wilson (1958)
	Griza Oriahs	100		29		
Nagpur	Mahar	450		100		Shukla and Solanki (1958)
	Kunbi	116		11		
	Teli	80		9		
	Koshti	46		—		
	Gonds	53		—		
	Muslim	68		—		
	Brahmin	26		—		
	Miscellaneous	171		—		
Bangladesh	Bengali	424		1		Swarup and Chatterjea (1958)
	Muslims			1	Hb S-β-thal	
Calcutta	Bengalis	10000		1		Chatterjea (1959)
Lucknow	Indians	235		—		Dube et al. (1959)
Mysore, Shimoga district	Indians	618	1		Harijan	Swarup et al. (1959)
Nagpur	Mahars	482		87		Das et al. (1967)
Karachi	West Pakistanis	76		1		Lehmann et al. (1961)
Kerala	Malayalis	190		—		Bird et al. (1966)
Nilgiri	Kurumbas	43		10		Kirk et al. (1962*b*)
	Irulas	15		6		
	Todas	60		2		
Aurangabad	Scheduled Caste	700	2	36		Lele et al. (1962)
				1	Hb S-β-thal	

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Table - *Contd.*

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
Bastar-Konta	Dorlas	200		26		Negi (1962)
Jagdalpur	Dhurwas	218		7		
	Northern Dhurwas	60		10		
Palghar	Sorathis	325	6	81		Mital et al. (1962)
				12	Hb S-thal	
Maharashtra	Marathas	201		—		Sanghvi (1962)
	Gurjars	203		—		
	Pajnas	200		—		
	Chamars	208		—		
	Mahars	200		4		
	Mixed	222		—		
	Thakurs	264		—		
	Koknas	190		8		
	Katkaris	262		21		
	Warlis	225		36		
Panchmahal	Bhils	206		32		Vyas et al. (1962)
Surat	Gamils	207	2	65		
	Dublas	211		20		
	Kolis	182		8		
	Naikas	174		28		
	Dhodias	213		38		
	Dhankas	215	3	41		
Bastar-Jagdalpur	Bade Bhatra	153		25		Negi (1963)
	Moyhela Bhatra	64		7		
	San Bhatra	88		17		
Khondagaon	Eastern Muria	143		15		
	Western Muria	169		27		
	Mahars	123		47		
Goa	Indians	379		1		Trincao et al. (1963)
Midnapore	Santhals (from 119 families)	336		2		Chaudhuri et al. (1964*b*)
Nilgiri	Kotas	12		—		Chaudhuri et al. (1964*a*)
	Todas	12		—		
Bastar-Bijapur	Bison-Horn Marias	185		29		Negi (1964)
	Raj-Gond	68		8		
	Dorla	27		3		
	Halba	34		9		
Bhopalpatnam	Mahars	30		12		
Bastar	Telanga	19				
	Raut	9		4		
	Banjara	5		1		
	Sonar	1		1		
	Gond	19		—		
Indore	Bhils	46		11		Kumar (1966)
	Balais	73		4		
	Chamars	22		2		
	Others	95		—		

Table - *Contd.*

Region	Population	No. tested	No. of subjects with Hb variant			Reference
			Homoz.	Heteroz.	Notes	
Bastar-Korapur	Tribals	10600		106		Roy and Sen (quoted by Chatterjea 1966)
Baroda	Bhils	220		38		Sayed and Amin (1966)
Wynad, Nilgiri	Paniyans	955		265		Das S.R. et al. (1967)
Korapur	Bada Gadabas	99		—		
	Pareng Gadabas	225		28		
	Ollaro Gadabas	225		6		
	Bareng Projas	104		—		
	Konda Projas	225		30		
Unspecified	Balais	104		5		
	Bhilalas	139		39		
	Bhils	174		21		
Midnapore Jalpaiguri, Cooch Behar	Rajbanshis	300		—		
24-Parganas	Padmaraj (Pods)	100		—		
	Mahishyas	60		—		
Ujjain	Mehtars	72		1		Kumar and Ghosh (1967)
	Brahmins	28		—		
	Rajputs	42		—		
	Jain and Vaishyas	15		—		
Dewas	Dhakars	25		—		
Ujjain and Dewas	Nayata Muslims	65		1		
	Chamars	27		1		
	Muslims	12		—		
	Balais	10		—		
	Others	17		—		
Aurangabad	Mahars	100		24		Deshmukh (1968)
Bastar	Raj Gond	54		15		
	Muria	35		10		
	Bhatra	25		7		
	Halba	26		6		
Bombay	Parsis	1290		5		Undevia (1969)
	Iranis	160		—		
Allahabad	Indian armed forces personnel	1378		8	5 Mahars 1 Maratha 1 Brahmin 1 Christian	Pande et al. (1970)
Bombay	Cutchi Bhanusali	599		9		Mehta et al. (1972)
Allahabad	Pakistani armed forces personnel	5000		9	2 Baluchistani 6 Punjabi 1 From N.W. Frontier Province	Sharma N.P. et al. (1976)

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Table - *Contd.*

Region	Population	No. tested	Hæmoglobin variants	Reference
OTHER HEMOGLOBINS				
Uganda	Gujratis	326	1 Hb AJ	Raper (1957)
Pondicherry	Lower castes	114	3 Hb AK	Labie et al. (1961)
	Unspecified	12	1 Hb AK	
Kualalumpur	Indians (cord blood)	278	3 Hb Bart's	Lie-Injo and Ti (1961)
Gujrat	Brahmins	203	—	Sukumaran et al. (1959)
	Lohanas	603	2 Hb AJ 3 Hb AL	
Singapore and Malaya	Indians	3341	2 Hb AL 1 Hb AJ 5 Hb AK	Vella (1962)
	Indians (cord blood)	222	2 Hb Bart's	
Madras	Indians	101	3 Hb AK	De Traverse et al. (1963)
Goa	Indians	2676	2 Hb AK 4 Hb AQ 20 High Hb F	Trincao et al. (1963)
NW Pakistan	Sindhis	6	1 Hb AJ	Bolton et al. (1964)
	Pathans	18	2 Hb AJ 1 Hb JD	
Calcutta	Bengalis (cord blood)	100	4 Hb Bart's	Swarup et al. (1965)
Malaya and Singapore	Indians (newborn)	226	4 Hb Bart's	Lopez and Lie Injo (1971)
Allahabad	Pakistani armed forces personnel	5000	3 Hb AK	Sharma N.P. et al. (1976)
Nepal	Gorkhas	1198	5 Hb A Norfolk 2 Hb AJ	Gupta et al. (1977)

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