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Sickle Cell Gene (HbS) Scenario in Tribal India

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Abstract

In India, a very high prevalence of sickle cell trait (SCT) has been reported from central, southern and western states, the frequency ranges from 0 to 48% with sporadic cases in eastern and north-western states. Of the total 6675 screened individuals for haemoglobin S (HbS) from Maharashtra, Kerala and Orissa, 748 samples of eight tribal populations were considered for present study.

A very high frequency of 20.3% has been observed for HbS among the Pardhan followed by the Gond (15.7%) and the Gowari (7.3%). The Banjara and the Halba show a similar pattern of HbS distribution being 5.9% and 5.04% respectively. The gene is found to be completely absent among the Mana of the same region. The Khutia khond of Orissa state show a lowest frequency for HbS gene (0.9%) of all the studied tribal groups. The Mullukuruman exhibits moderate frequency of 10.8% as compared to other tribal groups in southern India.

The tribal people of central and southern had a geographical unicentric origin and had unicentric origin of the mutated gene when these tribal populations were in direct contact and underwent panmixia or gene flow. But now they dispersed and live distantly isolating themselves and maintain strict endogamy leading to high frequency for HbS gene.

Keywords: Sickle cell trait; Haemoglobin S; Frequency; Tribal groups; Endogamy; Unicentric origin; Maharashtra; Kerala; Orissa; India

Introduction

FBecause of its bio-diversity that exists within and between populations, the Indian subcontinent offers unique opportunity for genetic and anthropological studies that throw light on various aspect of human being. Indian tribal populations constitute 8.2% of the total population [1]. Sickle cell anaemia is an autosomal genetic disorder caused by a defect in HBB gene (β 6Glu \rightarrow Val). The beta (β) globins gene is located on short arm (i.e., P-arm) of chromosome 11 and there are over 475 allelic variants. HbS (Haemoglobin S) is responsible for sickle cell disease, one of the most prevalent genetic diseases, affecting millions of people in India. Individuals who are sickle cell carriers are referred as sickle cell trait and do not express symptoms of sickle cell disease. Either double copy of the HbS variant on both the chromosomes or one copy of HbS and one copy of HbC variant on different chromosomes results into disease manifestation. In addition SCD results in combination with mutation for beta thalassaemia on other chromosome.

The presence of Sickle Cell Trait (SCT) provided a selective advantage in hyper endemic malarial areas [2]. The sickle cell gene in homozygous condition was found to be lethal in Africa [2], the homozygous apparently suffer from severe anaemia and die before attaining reproductive age without contributing to the gene pool of the population. Majority of the children born with SCA die before the age of 5 years. Haldane in 1949 described the role of balanced polymorphism for high incidence of SCD in Africa. The incidences of SCT are higher among the tribal groups than other caste populations [3].

Prof. James Herrick of Chicago was discovered and elongated sickle shaped red blood cell in 1910. Subsequently, Huck in 1923 showed that SCD was inherited as a simple Mendelian dominant. It was found that the trait was present to the extent of about 10% in African origins. The absence in other racial group led to the belief that sickling was an exclusively an African gene. Lehman and Cutbush [4] reported the presence of the trait in considerable frequencies in some of the tribal populations in and around Nilgiri Hills in South India. Buchi [5] was of the opinion that "the sickle cell cannot be a character of Weddis as a whole". He further pointed out that "the possibility of direct contact with the African for the introduction of the trait in India than independent mutation". SCD is by no means an African characteristic alone. Ingram [6] obtained the molecular change in the haemoglobin molecule of SCD –

Chemical structure	Position of amino acid
Normal HbA	$H_{3}N - Val - His - Leu - Thr - Pro - Glu - Glu$
Sickle HbS	H,N – Val – His – Leu – Thr – Pro – Val – Glu

Sickle cell disease is generally known by its prevalence in African and American origins. The hypothesis that falciparum malaria is mainly responsible for the maintenance of high frequencies of this trait is widely accepted. One of the evidence presented by Allison [2] in support of this hypothesis is that the distribution of hyper endemic falciparum malaria in Africa holds good for India too [7]. In India also, sickle cell trait is generally found in regions where malaria is present or was still present till recent time. Lower mortality risk is associated with HbAS due to its protection against malarial related mortality [8].

Material and Methods

A massive screening programme was conducted for haemoglobinopthies to identify high risk population and to create community awareness followed by counseling to the affected individuals/ families about sickle cell disease for its prevention and management. Several camps were organized in various schools and community halls during July 2005-December 2007 in three states namely Maharashtra (Western India), Kerala (Southern India) and Orissa (Eastern India)

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of India during which 6675 unrelated individuals comprising 3572 males and 3103 females were screened for Haemoglobin S (HbS) using solutions of qualitative solubility test. Necessary consents approved by the Ethical Committee of Anthropological Survey of India were obtained from individuals before subjecting them to the tests. In order to exclude any bias, the data was strictly adhered to indigenous population of unrelated and healthy individuals and free from any kind of ailment. Out of 6675 samples, Halba (139); Gond (230); Pardhan (64); Mana (31); Gowari (55); Banjara (17) from Maharashtra and two primitive tribal groups Mullukuruman (102) from Kerala and Khutia Khond (110) from Orissa state were considered for present study. The age of the subjects of both males and females ranges from <9 to 42 years (Table 1). 20 µl blood samples were drawn from finger prick for test and mixed thoroughly with solutions and readings were taken following WHO's guidelines. 2 ml intravenous blood from positive sample was drawn in BD Vacutainer® (USA) and brought to the DNA lab at CRC, Nagpur for further analysis. Laboratory investigations were carried out following standard procedure described by Dacie and Lewis [9]. All the samples were subjected to haemoglobin electrophoresis using cellulose acetate membrane in alkaline TEB buffer at pH 8.9 for pattern confirmation. The known samples (control) of HbS along with present samples were run for electrophoresis. DNA has been extracted from all HbS samples for its genomic study.

Results and Discussion

The age and sex wise distribution of sickle cell trait (SCT) is given in table 1. The highest frequency of SCT was found among the Pardhan (20.3%) followed by the Gond and the Mullukuruman being 15.7 and 10.8% respectively. The Gowari show the frequency of 7.3% for SCT while in Banjara and Halba it is found to be 5.9% and 5.0% respectively in western India. The Khutia Khond from eastern India, however, found to be lowest (0.9%) for HbS gene. Interestingly, SCT is found to be completely absent among the Mana community belonging to western India. However, the magnitude of SCT was found to be higher among the Pardhan of all the studied tribal groups (Table 1). The wide spread Pardhan in central and western India and few pockets of southern India exhibits more or less a similar pattern with earlier studies for sickle cell indicating stable frequency of the HbS gene irrespective of time and region (Table 2). Similarly, a very high frequency of HbS gene has been reported among various tribe like Gond, Kolam, Madia, Halba, Irula, Kurumba, Koya Dora, Panka and Otkar with varying degrees across the country. The Mullukuruman of the present study exhibit moderate frequency of 10.8% as compared to other tribal groups in southern India e.g., Adiyan, Yerava, Soliga, Betta Kuruba, Mullukrumba etc. It is found that the Mullukuruman exhibits higher frequency of sickle cell gene than the Kurichian but lesser than the Paniyan and Adiyan of the Wynad district in Kerala state. Sickle cell anaemia in the hilly, forested and once-malarial Wynad district was prevalent among two social groups- the tribal people, particularly the Paniyan and the Kattunayakan and the Wynadan Chetty, a small agriculture caste group, classified among the other backward castes.

The Panka shows highest incidences (48.6%) of SCT followed by Halba (27.3%), San-Bhatra (19.3%) and Bison-horn Maria (18.6%) amongst the tribal group of Chhattisgarh [7]. The Gond has similar high frequency of the trait (15.1 to 20.5%), the Kanwar who are in no way related to Gond show a low frequency of the trait (3.3%). This is in conformity with the findings of Negi [10]. The frequency of the Kanwar is comparable to that found in the Binjwar, the Manjwar, the Dhanwar and the Kamwar of Chhattisgarh (Table 2). The trait was introduced into the Kanwar through Raut and Cherwa by means of hybridization [11].

The Otkar, the Pawar, the Bhill, the Madia Gond, the Madia, the Kolam and the Halbi tribal groups of the eastern Vidarbha region show a very high frequency of HbS gene [3,12-14]. Table 2 portrays more than 30 % of the HbS gene among the tribes of central India viz., the Pardhan, the Otkar, the Panka, the Bhilala, the Dhanka and the Garasia in western India, and the Irula in southern India. Above 25% of the gene HbS was found among the Powar (Maharashtra), the Halba (Chhattisgarh), the Koya Dora (Andhra Pradesh), and the Gamit (Gujarat). The Warli, the Dhodia, the Madia Gond, the Kolam (Maharashtra), the Kurumba-Jenu, the Kurumba-Beta, the Soliga (Karnataka), the Gond (Madhya Pradesh), the Hill Maria (Chhattisgarh), the Paniyan, the Kurumba (Tamil Nadu) have been reported more than 20 % of sickle cell trait. It is evident from the Table 2 that the frequency of sickle cell trait is higher in southern Indian states i.e., Tamil Nadu, Karnataka, Andhra Pradesh, Kerala, followed by central India i.e., Chhattisgarh, Madhya Pradesh, and western India i.e., Maharashtra, Gujarat and Rajasthan.

In India, a very high prevalence of HbS gene has been reported in different tribal groups from central, southern and western India, the frequency ranges from 0 to 48% with sporadic cases in eastern and north-western states. The tribal populations from 9 states depict a very

Population	N	Sex	Age cohort							
			5 - 9	10 - 14	15 - 19	20 - 24	25 - 29	30 - 34	35 +	Total (%)
Banjara	17	М	-	-	1	-	-	-	-	1 (5.9)
		F	-	-	-	-	-	-	-	-
Gond	230	М	-	11	9	1	-	-	-	21 (9.1)
		F	1	6	6	1	-	1	-	15 (6.5)
Gowari 55	55	М	-	1	1	-	-	-	-	2 (3.6)
		F	-	1	1	-	-	-	-	2 (3.6)
Halba	139	М	-	1	1	-	-	-	1	4 (2.9)
		F	-	2	3	-	-	-		3 (2.2)
Khutia Khond	110	М	-	-	-	-	-	-	-	-
		F	-	-	-	-	-	1		1 (0.9)
Mana	31	М	-	-	-	-	-	-	-	-
		F	-	-	-	-	-	-	-	-
Mullukuruman	102	М	-	-	1	2	2	-	-	5 (4.9)
		F	-	-	2	-	-	1	3	6 (5.9)
Pardhan	64	М	-	1	2	-	-	-	1	5 (7.8)
		F	-	4	-	2	1	-	1	8 (12.5)

Table 1: Distribution of sample for HbS by age and sex.

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Community	Place	Category	No.	AA	AS	SS	% AS	Source
Maharashtra		0,						
Warli	Thane	ST	128	-	-	-	19.53	
Dhodia	Maharashtra	ST	65	-	-	-	21.54	
Konkana	Maharashtra	ST	83	-	-	-	3.61	
Pardhan	Nanded/ Yeotmal	ST	146	97	49	-	33.56	
Pardhan	Vidarbha	ST	38				31.6	
Kolam	Vidarbha	ST	28				20.3	
Rai Gond	Vidarbha	ST	57				11 7	
Naikgond	Vidarbha	ST	11				10.6	
Otkar	Gadchiroli	ST	-	_	_	_	35	
Pardhan	Yeotmal/ Nanded	ST	_	_	_	_	16.8	
Pardhan	Yeotmal/ Nanded	ST	_	_	_	_	33.7	
Pawara	Dhule Jalgaon	ST	_	_	_	_	25.2	
Madia Gond	Gadchiroli	ST	_	_	_	_	20.8	
Bhil	Nandurbar	ST	_	_	_	_	20.6	
Halbi	Gadchiroli	ST					13.0	
Malbar koli	Thana	et .					13.0	
Raigond	Gadchiroli	ST					10.9	
Kajyunu	Amrovati	ot ot	-	-	-	-	0.40	
Tandui	Annavau	от Ст	- 70	-	-	-	9.49	
Tanovi	Jaigaon	51 CT	12	-	-	-	8.33	
Kolam	Thoras	51	30	-	-	-	8.33	
vvarii	Inane	SI	-	-	-	-	8.04	
Katkari	Pune, Ratnagiri	SI	-	-	-	-	5.9	
Kokana	Dhule,Nashik	SI	-	-	-	-	3.5	
Andh	Nanded	SI	-	-	-	-	1.97	
Mahadeo Koli	Pune, Nashik	ST	-	-	-	-	0.81	
Madia	Gadchiroli	ST	140	115	25	-	17.86	
Maria Gond		ST	167	-	-	-	16.17	
Halba	Nagpur	ST	139	137	2	-	5.04	
Gond	Nagpur/Gondia	ST	230	203	27	-	15.65	
Banjara	Vidarbha	ST	17	14	1	1	5.88	
Mana	Nagpur	ST	31	31	-	-	0	
Gowari	Gondia/Yeotmal	ST	55	51	4	-	7.27	
Pardhan	Nagpur	ST	64	51	13	1	20.31	
Chhattisgarh								
Binjwar	Raipur	ST	14	14			0	
Binjwar	Bilaspur	ST	13	12	1	-	3.85	
Dhanwar	Bilaspur	ST	31	31			0	
Gond	Raipur	ST	157	132	25		7.96	
Gond	Raipur	ST	63	44	19		15.08	
Gond	Bilaspur	ST	129	104	25		9.69	
Gond	Ambikapur	ST	127	101	26		10.24	
Gond-Bade Bhatra	Bastar	ST	153	138	25		16.34	
Bison Horn Maria	Bastar	ST	442	360	82		18.55	
Bison Horn Maria	Bilaspur	ST	218	171	47		10.78	
Dhurwa	Bastar	ST	60	50	10		16.67	
Dhurwa	Bastar	ST	348	336	12		3.45	
Gond Muria	Bastar	ST	143	128	15		10.49	
Gond Muria	Bastar	ST	169	142	27		15.98	
Hill Maria	Bilaspur	ST	85	68	17		20	
Gond Raj	Bastar	ST	70	60	10		14.29	
San Bhatra	Bastar	ST	88	71	17		19.32	
Halba	Bastar	ST	77	56	21		27.27	
Halba	Raipur	ST	33	29	4		13.32	
Halba	Raipur	ST	122	105	17		6.97	
Kamar	Raipur	ST	21	20	1		2.38	
Kanwar	Ambikapur	ST	100	100	-		0	
Kanwar	Bilaspur	ST	91	88	3		3.3	
Kawar	Ambikapur	ST	114	113	1		0.46	
Kawar	Rainur	ST	72	68	4		2 78	
	i taipui	.	· -		1		2.10	

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				1	1	1	1	
Kol	Bilaspur	ST	10	10	-		0	
Manjwar	Bilaspur	ST	17	17	-		0	
Oraon	Bilaspur	ST	6	6	-		0	
Panka	Raipur	ST	26	17	9		17.3	
Panka	Bilaspur	ST	35	18	17		48.57	
Madhya Pradesh								
Bhilala	Jhabua	ST	384	263	121		31.75	
Gond	-	ST	157	-	-	-	15.92	
Kawar		ST	72	-	_	_	5 45	
Halba		ST	122	_	_	_	13.93	
Gond		ST	127	_	_		20.47	
Oraon		ST	127		-	-	2 1 2	
Kewer		01	422	-	-	-	2.13	
Nawai		51	114	-	-	-	0.00	
Bhii		SI	145	-	-	-	17.24	
Korku		SI	102	-	-	-	1	
Bhilala		ST	977				19.04	
Gujarat	1	1	1	1	1			1
Dhanka	Surat	ST	30	21	9		30	
Gamit		ST	251	-	-	-	17.13	
Gamit	Surat	ST	137	103	34		25	
Gamit		ST	207	-	-	-	31.4	
Naik		ST	174	-	-	-	16.09	
Bhil		ST	206	-	-	-	15.53	
Dhanka (Bhil)		ST	215	-	-	-	19.07	
Dhodia		ST	213	_	_	_	17 84	
Raiasthan		01	210				11.01	
Garasia	Sirohi	ет	75	50	25		33 33	
Carasia	Udoinur	et .	25	24	11		21 42	
Galasia	Ouaipui	51	35	24	11		31.43	
Anunra Pradesn) (in her a her a her a her	OT	100	00	20		14.0	
Relli	visnakapatanam	51	138	99	39		14.2	
Pardnan	Adiiabad	51	101	65	35	1	34.65	
Yerukula		SI	125	-	-	-	0.8	
Yerukula		SI	40	-	-	-	0	
Savara		ST	132	-	-	-	1.52	
Jatapu		ST	157	-	-	-	1.27	
Jatapu		ST	260	-	-	-	5.94	
Sugali		ST	61	-	-	-	4.92	
Konda Reddy		ST	92	-	-	-	2.17	
Koya		ST	159	-	-	-	12.57	
Raj Gond		ST	133	-	-	-	14.28	
Koya Dora		ST	99	-	-	-	24.24	
Koya Dora		ST	547	-	-	-	7.86	
Naik Pod		ST	90	-	-	-	4.44	
Lambadi		ST	154	-	-	-	2.6	
Pardhan		ST	122	-	-	-	31.79	
Rai Gond		ST	197	-	-	-	11.17	
Kolam		ST	215	-	_	_	14.92	
Chenchu		ST	139	_	_	_	0.72	
Kova		ST ST	238				6.59	Dingle [23]
Manna		0T	155	-	-	-	0.50	
Kalana		51	100	-	-	-	2.00	
Kolam		51	142	-	-	-	7.04	
Kaj Gond		51	140	-	-	-	14.28	
Karnataka	-							
Kurumba-Jenu	Coorg	ST	76	46	29	1	20.66	
kurumba-Beta	Coorg	ST	67	39	27	1	21.6	
Soliga	Mysore	ST	102	61	40	1	20.59	
Kerala								
Adiyan	Cannore	ST	75	51	24		16	
Paniyan	Kerala	ST	61	40	21		17.21	
Mullukuruman	Wynad (Kerala)	ST	102	91	11	-	10.78	
Adiyan	Cannore	ST	75	51	24		16	
-		1	1		1			

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Paniyan	Kerala	ST	61	40	21		17.21
Kurichian	Wynad (Kerala)	ST	106	104	2	-	1.88
Mullukuruman	Wynad (Kerala)	ST	102	91	11	-	10.78
Tamil Nadu							
Paniyan	Nilgiri	ST	109	62	45	2	22.47
Irual	Nilgiri Hills	ST	124	85	39		15.73
Irual	Nilgiri Hills	ST	254	168	81	5	17.91
Irual	Nilgiri Hills	ST	130	84	46		35.38
Kurumba-Mullu	Nilgiri	ST	101	62	38	1	19.8
Toda		ST	98	-	-	-	1.02
Kurumba		ST	43	-	-	-	20.93
Toda		ST	60	-	-	-	3.33
Irula		ST	15	-	-	-	40
Kurumba		ST	43	-	-	-	23.25
Kota		ST	549	-	-	-	0
Irula		ST	175	-	-	-	26.28
Irual	Nilgiri Hills	ST	124	85	39		15.73
Irual	Nilgiri Hills	ST	254	168	81	5	17.91
Irual	Nilgiri Hills	ST	130	84	46		35.38
Kurumba-Mullu	Nilgiri	ST	101	62	38	1	19.8
Toda		ST	98	-	-	-	1.02
Kurumba		ST	43	-	-	-	20.93
Toda	Nilgiri	ST	60	-	-	-	3.33
Irula	Nilgiri	ST	15	-	-	-	40
Kurumba	Nilgiri	ST	43	-	-	-	23.25
Kota		ST	549	-	-	-	0
Irula		ST	175	-	-	-	26.28
Orissa (Odisha)							
Gadaba-Ollaro	Koraput	ST	225	218	7		3.11
Khutia khond	Phulbani dist	ST	110	109	1	-	0.91
Bado Gadaba	Koraput	ST	100	100	-	-	0
Baren Paroja	Koraput	ST	100	100	-	-	0
Kondh		ST	-	-	-	-	5.5
Kutia kondh		ST	-	-	-		1.7
Oraon		ST	-	-	-	-	0.2
Saora		ST	-	-	-		0.2
Santal		ST	-	-	-	-	0
Bihar							
Oraon		ST	130				0.77
Oraon		ST	56				0
West Bengal							
Santhal		ST	336	4	-	-	1.19
Santhal		ST	164	0	-	-	0
Oraon		ST	204	2	-	-	0.98
Kaora		ST	202	1	-	-	0.5

Table 2: Community and state wise frequency distribution of sickle cell trait.

high percentage of incidences of HbS gene. The frequency of 17.2% among the Paniyan for HbS gene starts from Kerala of southern India goes up to 48.6% among the Panka in Chhattisgarh and then follows a declining trend to western and north-western states of Gujarat and Rajasthan. One of the reasons for occurring in such a high incidences could be attributed to the practice of consanguinity among most of them and the similar findings were reported by Mukherjee and Das [15]. But it is surprising to note that why the other neighboring populations who live in the similar ecological niche do not exhibit similar trend for the SCT? In some of the tribal groups the HbS gene is completely absent. The Khutia khond, the Korku, the Chenchu, the Oraon, the Kolam, the Lambadi, the Halba, the Kawar, the Andh, the Toda, the Savara, the Jatapu, the Konda reddy, the Yerukula, the Manne etc., show a

negligible frequency for SCT. The frequency of HbS gene was found to very low among the tribes of Orissa, Bihar and West Bengal.

Piel et al. [16] showed the presence of HbS allele frequency of >0.5% in the African continent, the Middle East and India. The highest frequency of HbS allele in tribal groups of India indicates a similar pattern of prevalence of SCT from southern Senegal to northern Liberia to southeast Tanzania to Saudi Arabia up to Chhattisgarh and southern Karnataka region of India. Mukherjee and Das [15] are of the view that the highest gene frequency of HbS occur in region where malaria is highly endemic. HbS was largely absent in the areas of Horn of Africa and south of the Zambezi. Piel et al. [16] shown a similar pattern of distribution of HbS frequency in malaria-free, hypoendemic

and mesoendemic zones but higher in hyperendemic and holoendemic areas. In Asia, no relation between HbS allele frequency and malaria endemicity was found. Globaly, the HbS frequency increases with the increase in malarial endemicity but a reverse trend has been noticed in Asia.

Based on the high magnitude of sickle cell, the gene HbS could be located from four zones across India: (1) Tamilnadu, Andhra Pradesh and Karnataka, (2) Chhattisgarh and Madhya Pradesh, (3) Maharashtra, Gujarat and Rajasthan and (4) Kerala, besides sporadic occurrence of gene in Orissa, Bihar and West Bengal. The concentration of HbS gene is in southern and western states spreading across the country with intermediate frequencies in the neighboring state and slow down it in farther states. From this two inferences can be drawn, 1. The mutation for HbS gene has taken place independently in different populations during the course of time and/or 2. The lethal gene HbS has introduced in India through southern and western sea-routes during ancient time as the frequency of HbS is higher in those corresponding states.

Rao et al. [17] had shown a trimodal distribution of HbS concentrations among the tribes of Maharashtra. Many tribal groups practice area inbreeding and the deleterious gene may be confined within the smaller endogamous groups that maintain more or less a stable frequency among them as the pattern of SCT was found to be same reported by earlier studies.

Conclusion

The tribal people of central and southern had a geographical unicentric origin and had unicentric origin of the mutated gene when these tribal populations were in direct contact and underwent panmixia or gene flow. But now they dispersed and live distantly isolating themselves and maintain strict endogamy leading to high frequency for HbS gene. A high frequency of SCT was reported among the food gatherers and jhoom/slash burn agriculturist in India [18]. But the distribution pattern of HbS does not conform to the earlier studies as the HbS in high frequencies is prevalent among the tribes with varied life styles. For example, the Pardhan, the Gowari, the Bhilala and the Gamit are agriculturist; the Irula is an artisan; the Kurumba, the Madia, are hunting tribes and the Banjara is a nomad, show equally high frequencies of HbS amongst them. On the contrary, the Mana is an agriculturist, the Halba is weaver and the Khutia khond is hunters and gatherers show a complete absence or negligible HbS gene.

Lehman and Cutbush [4] first detected the sickle cell gene among the Irula of Nilgiri Hills in South India. The frequency of this lethal gene varies from complete absence to 48.57% among the tribal population which is quite alarming as compare to other ethnic groups in India. Once upon a time the Mullukuruman were hunters and gatherers but now they are settled agriculturist. The moderate frequency of 10.78% sickle cell gene among the Mullukuruman justify the hypothesis put forward by Allison. On the contrary, some tribal groups whose economy is still depend on hunting and gathering and some of them are settled agriculturist but do not have high incidences of sickle cell trait and in some of them the trait is completely absent. The Allison hypothesis says that the sickle cell trait shows its presence in the hyper endemic region of falciparum malaria and the population whose economy revolves around agriculture and settled population. A correlation between sickle cell trait and malarial endemicity has been found among some populations of central India and Maharashtra [19,20]. But how far the malarial hypothesis is tenable for the spread of sickle cell among different populations need in-depth study in Indian context as the prevalence of SCT is not uniform across the country where the malaria still exist.

HbS is in some of the indigenous populations of northern India, eastern India and north east India the HbS gene is either completely absent or present in very low frequency. Therefore, the malaria-sickle cell hypothesis does not hold well in India so far as the occurrences of HbS in different indigenous tribal as well as caste groups in varied ecological zones are concerned. In India most of the part are highly malarial endemic, despite this HbS is absent in more than half of its region.

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