is small compared to the spatial scales of the ocean, but this would certainly add more clarity to understanding the Indian Ocean SST relation with the Indian rainfall.

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Prevalence of abnormal haemoglobin E gene in the Dhelki Kharia tribal population

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Tribal communities in India are vulnerable to many hereditary blood disorders. The sickle cell haemoglobinopathy and glucose-6-phosphate dehydrogenase (G-6-PD) deficiency are the most common haemolytic disorders of blood prevalent among the aboriginal or autochthonous people of India. In the present study out of 335 Dhelki Kharia tribals screened following the probability proportionate to size cluster sampling procedure, eleven cases of haemoglobin E gene (ten cases of haemoglobin E trait and one case of haemoglobin E disease) were detected for the first time from Sundargarh district of Western Orissa. The present

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study shows the prevalence of 3.3% of haemoglobin E gene in Dhelki Kharia tribe and 1.5% in the whole Kharia community in Sundargarh district. These cases were studied for haematological, clinical and genetical aspects. Out of eleven cases, only three males were found deficient for G-6-PD enzyme, one of whom was homozygous haemoglobin E disease case, the other two being carriers for haemoglobin E gene. These findings have been discussed in the light of previous studies carried out in Orissa.

THE hereditary disorders of blood in general follow Mendelian inheritance and are among the first abnormalities detected in human beings. The erythrocytes of human blood inherit many genetic defects. The disorders of haemoglobin (Hb) have been attributed to the alterations in single amino acid of the globin moiety. The red cell enzyme defect like glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is the commonest x-linked disorder occurring in human populations. These common hereditary defects including blood group incompatibility cause haemolytic anaemia of varying degree among the vulnerable people in India.

It has been estimated that approximately 50 million people in South East Asia alone carry the gene for haemoglobin E[1]. This abnormal haemoglobin, a beta-chain structural mutant (β^{26} Glu $\rightarrow$ Lys) is particularly prevalent in Bangladesh[1], Indonesia[2], Malaysia[3], Myanmar[4,5], Singapore[1] and Thailand[1]. In India, this abnormal haemoglobin is mostly prevalent in the Eastern states, i.e. West Bengal, Assam, Arunachal Pradesh, Nagaland, Manipur, Tripura and Meghalaya[6] and occurs only sporadically in a few other regions of India[7–9]. It has been believed to be extremely rare especially among the tribal populations of Orissa, which is a Central-East state of India having relatively high prevalence of the sickle cell gene[10–12].

The haematological consequences of haemoglobin E gene either in heterozygous (Hb AE trait) or homozygous (Hb E disease) condition are mild anaemia or without anaemia, microcytosis and hypochromia of erythrocytes with morphological resemblance to erythrocytes in beta-thalassaemia trait[13,14]. However, the haemoglobin E (Hb E) gene can interact with beta-thalassaemia gene, and the results in double heterozygous state which may exhibit phenotypic manifestations of beta-thalassaemia major or thalassaemia intermedia[14].

To the best of our knowledge, there is no study which reports the prevalence of haemoglobin E gene in the tribal population of Orissa[9,15]. This is the first study of its kind from Orissa. Ten cases of haemoglobin E trait and one case of homozygous E disease were detected in a tribal community of Sundargarh district.

The Kharia tribe is an endogamous community, which is mostly confined to Sundargarh district. This district is surrounded by Jharkhand in the North, Chhattisgarh in the West, Keonjhar district in the East and Jharsuguda, Sambalpur and Deogarh districts in the Southern part (Figure 1). However, the sporadic families of Kharia are also seen in Sambalpur, Deogarh and Jharsuguda districts. Some of their families have also migrated to Assam to work in the tea plantations[6]. In Orissa, the total popu-

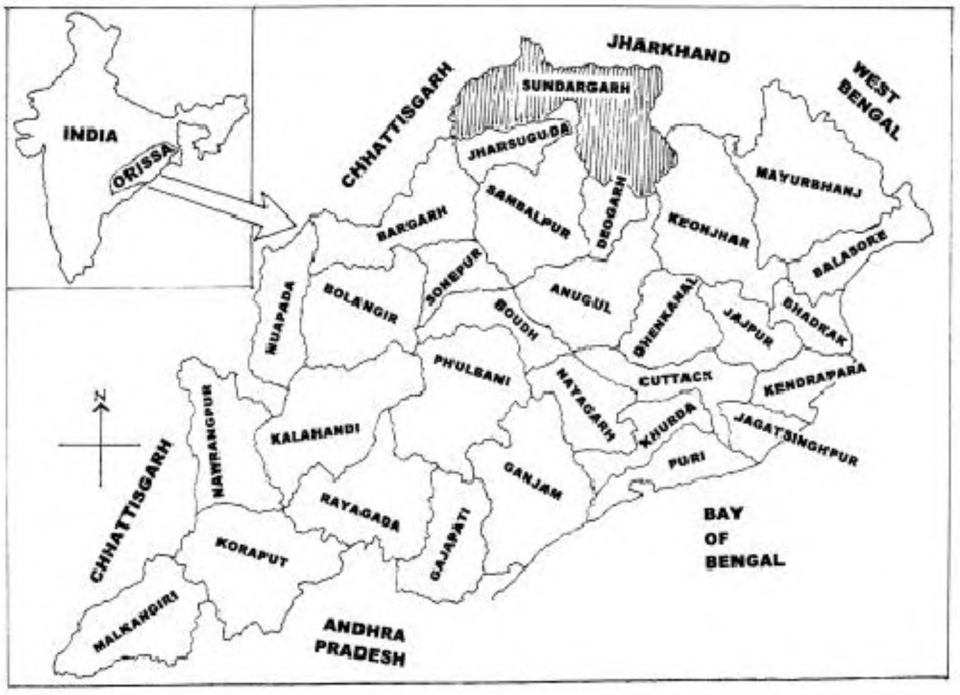

Figure 1. Map of Orissa showing thirty districts and study area (shaded).

lation of Kharia tribe was 1,44,174 persons according to 1981 Census which rose to 2,06,229 in 2001. The sex ratio of Kharia tribe was 1013 females per 1000 males. The literacy rate was 17.9% in 1981 which rose to 33% by 2001.

The Kharia tribe although originally belonged to one ethnic group, has now split into three social groups namely, the Hill Kharia (Pahari Kharia), Dhelki Kharia (Early Comers) and the Dudh Kharia (Pure Kharia), which are distinguished from each other on the basis of three grades of primitive culture. The Hill Kharia, the primitive and backward section, represents the hunting and food gathering stage of economic life along with the practice of rudimentary shifting cultivation and primitive culture. The Dhelki Kharia section represents the more advanced culture with the habit of plough-cultivation and food production. The Dudh Kharias have the most advanced culture, which equates them with other nontribal population of the region. The Dhelki Kharias preponder over the other two groups in population size. The intergroup marriges are not taking place. Reproductively and genetically, they are completely isolated from each other.

Following the probability proportionate to size (PPS) cluster sampling procedure, the blood samples were collected from three blocks, namely, Balisankara, Bargaon and Subdega. A total of 752 Kharia tribals (371 males and 381 females) belonging to subgroups of Dhelki Kharia (175 males and 160 females) and Dudh Kharia (196 males and 221 females) in all age groups were screened for haemoglobinopathies, G-6-PD deficiency, and ABO and Rhesus blood groups. However, out of a total 335 Dhelki Kharia screened, eleven cases of haemoglobin E were detected.

About 2–3 ml of blood was collected using ethylene diamine tetra acetic acid (EDTA) as anticoagulant from each individual after obtaining the informed consent in the presence of a doctor and community leaders. The signs and symptoms related to haemoglobinopathy and G-6-PD deficiency were recorded after clinical examination on the pre-designed proforma.

The blood samples so collected were transported to the laboratory at Bhubaneswar under ice-cold conditions within 24 h of collection. Laboratory investigations were carried out following the standard procedures after cross checking for quality control from time to time. Haematological parameters were studied by using an automated particle cell counter (Model-MS4, Melet Schloesing Laboratories, France).

The routine horizontal haemoglobin electrophoresis was carried out on cellulose acetate membrane (CAM) in Tris-EDTA-Borate buffer at pH 8.6 to identify abnormal haemoglobins by their differential mobility and quantification of A_2 fraction of haemoglobin by elution method at pH 8.9 (refs 14, 16). As Hb A_2 and Hb E have similar electrophoretic mobility on cellulose acetate membrane and cannot be separated by elution or DE 52 column chromatography, a haemoglobin fraction in that position constituting 15% or more of the total haemoglobin content was taken as Hb E[17]. Foetal haemoglobin (Hb F) was estimated by Betke *et al.*'s[18] method as described by Weatherall[14].

The G-6-PD enzyme deficiency was detected by using dichlorophenol indophenol (DCIP) dye as described by Bernstein[19] and, subsequently, confirmed by WHO procedures[20] and Beutler and coworkers[21]. ABO and Rhesus blood groups typing were done by slide method, following manufacturer's instructions. (Tulip Diagnostics Private Limited, Goa).

Out of eleven cases (7 males and 4 females) of haemoglobin E detected among the Dhelki Kharia tribe out of 335 screened, ten were haemoglobin E trait and one was homozygous E disease. All the cases were the inhabitants of Sundargarh district. In none of these families, was there any available history of marriage with persons from regions with a high prevalence of Hb E.

The salient haematological features of eleven cases of haemoglobin E are summarized in Table 1. The table gives the haematological profile and other details of haemoglobin E cases encountered in Orissa state, which will be useful for comparison of data from other sources in future. Moreover, these data help in ascertaining and interpretation of the clinical and haematological profile of the individual cases.

The age of these eleven cases ranged from 5 to 70 years. There were seven males and four females. The haemoglobin level range was from 7.2 to 11.3 g/dl in females and 8.6 to 13.8 g/dl in males and haematocrit from 25 to 43%. Haemoglobin E varied from 24 to 37% in trait cases, Hb F from 0.4 to 1.8%, and haemoglobin A could be detected in all except one (homozygous E) case. The haematological indices such as MCV, MCH and MCHC were recorded low in all these cases.

Red cell morphology showed moderate to marked hypochromia, anisocytosis and poikilocytosis, polychromasia and a variable number of target cells. Normoblasts were present in variable numbers in the peripheral blood of all except two cases. None had received blood transfusion during at least three months before they were detected for haemoglobin abnormality. All except two cases had mild to moderate anaemia. The liver and spleen were found enlarged (2–5 cm) below the costal margin only in three cases.

Out of eleven cases studied, only three males were found deficient for G-6-PD enzyme, one of whom was a homozygous E disease case and other two were carriers for haemoglobin E. None of them was administered any antimalarials. Hence there was no elicitable cause of haemolysis due to antimalarial drugs or otherwise.

The ABO and Rhesus blood groups distribution in eleven cases of haemoglobin E showed no susceptibility towards any specific blood group, except that all cases were Rhesus (D) positive. In general, the frequency of Rhesus negativity is very low (0–2.1%) among the tribes of Orissa.

Table 1. Salient features of eleven Delki Kharia tribals in Sundargarh District of Western Orissa

Code no.	2025	3094	2206	2194	2208	2024	3093	3090	2020	2161	2007
Name	BD	SN	RN	AD	AN	RD	LN	DN	TD	SD	GD
Age (years)	9	16	25	30	5	11	22	32	45	60	70
Sex	F	F	F	F	M	M	M	M	M	M	M
Hb (g/dl)	11.3	7.2	11.6	10.2	8.6	10.6	13.8	12.4	9.1	10.2	9.5
RBC (10^6/µl)	5.2	4.9	5.9	3.7	4.7	5.1	5.3	5.4	5.2	5.9	4.9
MCV (fl)	77.1	50.7	74.1	79.4	65.7	76.5	73.4	75.0	65.0	70.6	72.4
HCT (%)	40.1	24.6	43.4	28.9	30.6	38.7	42.0	43.2	34.0	42.5	35.3
MCH (pg)	21.8	12.3	19.8	20.8	18.4	20.9	26.0	23.0	17.4	18.9	19.5
MCHC (g/dl)	28.3	24.3	26.7	26.2	28.1	27.3	32.8	28.7	26.7	26.7	27.0
WBC (10^3/µl)	8.3	8.8	11.7	6.5	9.6	6.5	6.1	6.4	6.4	7.2	5.4
Electrophoresis	A + E	A + E	A + E	A + E	A + E	A + E	A + E	A + E	E + F	A + E	A + E
Hb A (%)	61.6	72.8	66.8	74.9	71.6	70.6	63.8	66.0	0.0	71.1	66.6
Hb A_2+E (%)	36.9	26.5	32.6	24.4	28.0	28.7	35.2	32.5	98.2	28.1	32.8
Hb F (%)	1.5	0.7	0.6	0.7	0.4	0.7	1.0	1.5	1.8	0.8	0.6
G-6-PD enzyme	N	N	N	N	D	N	N	N	D	D	N
ABO blood group	A	A	B	O	A	O	B	B	A	AB	AB
Rhesus blood group	+ve	+ve	+ve	+ve	+ve	+ve	+ve	+ve	+ve	+ve	+ve

N, Normal; D, Deficiency.

The present article reports ten cases of haemoglobin E trait and one case of homozygous E disease. Haemoglobin E constituted 24–37% of the total haemoglobin in ten cases and corresponding levels of Hb F ranged from 0.4 to 1.8%. No report is available on the haematological parameters of haemoglobin E cases in Orissa. However, these findings are in agreement with the previous studies carried out elsewhere on the haemoglobin E cases in India[4]. Further, the presence of moderate to severe haemolytic anaemia, icterus, moderate to marked morphological abnormalities of red cells, e.g. target cells, hypochromia, anisocytosis and poikilocytosis have all adjuncted to establish the diagnosis in these cases.

The haematological variations, clinical profile, nature and distribution of phenotypes of haemoglobin E gene in different regions of India are largely unknown[7,15] The clinical severity of Hb E is also variable, although in most of the cases the disease is of moderate severity[14]. It has been shown that the haemoglobin E gene behaves functionally like a beta-thalassaemia gene since the E globin chain synthesis in Hb E trait is inhibited as in beta-thalassaemia trait[22,23].

There exists a wide range of variation of haemoglobin E gene in populations of North Eastern India[6,7]. The average allele frequency of haemoglobin E gene has been found to be 10.9% in North Eastern states of India[9]. The present study shows the prevalence of 3.3% of haemoglobin E in Dhelki Kharia tribe and 1.5% in the whole Kharia community in Sundargarh district. These eleven cases of Hb E gene were encountered in Western part of Orissa and suggest that the Hb E gene may not be rare in this region as was generally believed earlier. However, based on sickle cell clinic data in pediatric age group (age 3 months to 14 years) during the period of two years at a Medical College Hospital in Western Orissa, a case of E-beta-thalassaemia[24] and the prevalence of 0.1% for haemoglobin E-beta-thalassaemia has been reported earlier by us[25].

Molecular studies have shown the mutation (GAG → AAG) at the codon of 26 among the people with haemoglobin E[6]. The Sundargarh district is highly endemic for malaria, especially for *Plasmodium falciparum*. This mutation of haemoglobin E may be advantageous in high endemic malaria areas due to heterozygote advantage to the carrier as the parasites are unable to survive for longer duration under such abnormal red cell conditions. As haemoglobin E gene is most common in populations especially of North Eastern India, and has a high frequency in malarious regions, it is essential to find this defect in association with alpha- and beta-thalassaemia. Thus, the detection of haemoglobin E mutation does not exclude the possibility of compound heterozygosity of haemoglobin E and beta-thalassaemia in the populations of Orissa.

It is difficult at this stage to say whether the haemoglobin E gene is an independent mutation in this tribal community or it has penetrated from outside. There is every possibility of an independent haemoglobin E mutation in Orissa, which will be established after molecular analysis. There is also likelihood of infiltration of haemoglobin E gene from West Bengal and/or North Eastern states of India. Haemoglobin E gene is widespread in West Bengal and North Eastern states. The migrations of Kharia tribe from Orissa to Assam and West Bengal have taken place about 1850 AD in search of employment in the tea industry[6,8], which are high endemic regions of haemoglobin E gene. There is equal likelihood of penetration of abnormal haemoglobin E gene in Assam as a result of mixing up with the local populations, who carry this gene. They subsequently, abandoned Assam or West Bengal and returned home. Since Kharia tribe is patrilo-

cal, some of Kharia tribe's forefathers either brought wives from West Bengal or from North East region and settled here.

Further molecular studies are needed on the polymorphism and haplotype studies of haemoglobin variants, especially among the tribals of Orissa.

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Occurrence of a *Hop stunt viroid* (HSVd) variant in yellow corky vein disease of citrus in India

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A viroid was isolated and purified from total nucleic acid extract of *Kagzi* lime (*Citrus aurantifolia*) leaves affected by yellow corky vein disease. It was cloned in pGEMT-easy vector system and sequenced. *In silico* analysis showed that it consisted of 295 nucleotides. In BLAST analysis the sequence aligned with different *Hop stunt viroid* (HSVd) variants showing nearly 100% sequence identity with six citrus cachexia isolates of HSVd. The viroid was tentatively named as yellow corky vein variant of *Hop stunt viroid* (HSVd-ycv). This constitutes the first report of molecular evidence for occurrence of a *Hop stunt viroid* variant from citrus in India.

VIROIDS are low molecular weight, infectious, non-encapsidated, self-replicating, circular, single-stranded RNA molecules (246–463 nt) without any functional ORFs in their genome[1]. Ever since the first report of viroid[2] in potato, 32 viroid species[3] and 160 viroid variants[4] with complete sequence data have been recorded. These rap-

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