Haemoglobin-C gene in India?

Mammalian haemoglobin (Hb) molecule is a conjugated protein consisting of four or five different peptide chains. These are designated as alpha (α), beta (β), gamma (γ) and delta (δ) with an alleged fifth type represented as epsilon (ε). Each chain has intimately but reversibly bound to it a molecule of the iron containing porphyrin ring called haem. Human haemoglobin contains two identical alpha chains; other two chains are either β, γ or δ. The various polypeptide chains, similar in the overall length, differ in the number and type of amino acid residues. The α-chain has 141 residues and β, γ and δ chains have 146 residues each.

In erythrocytes (red blood cells) of a normal adult human, two types of haemoglobin are found. The majority, about 97%, is normal adult haemoglobin (Hb-A), and a small fraction, of about 2.5% is haemoglobin A_2 (Hb-A_2); traces of haemoglobin F (Hb-F) are found predominantly in a foetus.

During human evolution, some mutations (substitution/deletion) that occurred resulted in a change in the molecular structure of genes responsible for haemoglobin synthesis. These genetic changes commonly occur in the natural selection process. Expression of abnormal or mutant genes either results in the formation of several abnormal haemoglobin variants (e.g. Hb-S, D, E, C, etc.) or decrease in the synthesis rate of globin chains (e.g. β-thalassaemia). Such genetic changes were mostly regional and race-specific. One of the β-globin chain Hb variants, Hb-C only occurs with a frequency comparable to that of Hb-S in a quite restricted area, west river Niger, in West Africa. The incidence of *Hb-C* gene in northern Ghana is about 20% (refs 1 and 2); there is also observed and reported 2% incidence in African Negroes[3]. It has been found in Morocco, occasionally in South Africa and rarely in white individuals[4–6]. That the incidence is so restricted suggests that the gene is of relatively recent origin[7].

In India, the rarest Hb variant (Hb-C) has also been detected. The occurrence of this mutant gene was first observed in the Bohra-Muslims of southern Rajasthan in 1991 (ref. 8). The method adopted for its identification was electrophoresis. Later, this abnormal Hb was also detected in tribals and other ethnic groups of Rajasthan[9–11]. In 2007, Kumar and his co-workers[12] identified and reported this mutant *Hb* gene in double heterozygous form (Hb-C/β-thalassaemia) in a 22-year-old female patient and erroneously claimed that this is the first case of Hb-C in India. From India, another recent report on the evidence of *Hb-C* gene in the population of West Bengal is also available[13]. Hb-C thus is not restricted to native Africans but is also found in other geographical regions and diverse ethnic groups. Whether *Hb-C* gene has any correlation with falciparum malaria as found in case of sickle cell haemoglobin (Hb-S), more surveys are highly suggestive on its distribution in different geographical areas or ecosystems having different malaria endemicity. However, *in vitro* study is more suitable and reliable to find out a correlation between Hb-C and falciparum malaria.

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Citedness of Indian science journals indexed by *SCIE*

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