ADVANCES IN BIORESEARCH, Vol 1 [2] December 2010: 69 - 71

http://www.soeagra.com
ISSN 0976-4585

RESEARCH PAPER

Advantageous Alleles, Parallel Adaptation, Geographic Location and Sickle Cell Anemia among Africans and Indians

Clyde Winters
Uthman dan fodio Institute
Chicago,Illinosis 60643
Email: olmec982000@yahoo.com

ABSTRACT
Parallel phenotypic adaptation in nature is considered unlikely because of the multiple genes involved in parallel evolution. Recently researchers claimed that Indian sickle cell anemia is an example of parallel genotypic adaptation. Findings from archaeogenetic evidence fails to support this proposition.
***KEY WORDS**: Phylogenetic, parallel adaptation, evolution, haplogroup, advantageous alleles*

INTRODUCTION

It has been assumed that parallel genotypic adaptation via advantageous alleles is unlikely. Recently researchers have found circumstantial evidence that parallel adaptation occurs at taxonomic levels [1]. This suggest that parallel genotypic adaptation probably takes place in the natural world.

Recently Ralph and Coop [2] claimed that sickle cell anemia in India is probably a case of natural parallel adaptation. In this paper we will determine if the presence of sickle cell anemia in India demonstrates parallel adaptation.

Ralph and Coop [2] are not the first researchers to use parallel evolution to explain Indian phylogenetics. It was also used to explain the shared haplogroup M1 HVS-1 signature motifs: 16129,16189, 16249 and 16311, in the Indian M haplogroups [3].

Ralph and Coop [2] argue that environment and geographical location can influence parallel adaptation of advantageous alleles across species . These researchers maintain that parallel mutation best explains independent convergent evolution at orthologous genetic loci [2].

Ralph and Coop [2] suggest that sickle cell alleles among geographically diverse human populations are evidence of the wave of advance of advantageous alleles. They reason that the sickle cell allele HbS gene in humans has evolved into four distinct haplotypes across different African geographic regions: Central African Republic (Bantu), Benin, Senegal and Cameron, and the Arab-Indian sickle cell in Eurasia. The Senegal haplotype is found mainly in Senegal and above the Niger River. The Benin haplotype mainly exist in Nigeria and Benin [4].

Ralph and Coop [2] conclude that eventhough there are few solid representations of parallel adaptive mutations within species, they predict that the acquisition of advantageous alleles through parallel adaptation will be common among large geographically spread populations. Intuitively this hypothesis appears appropriate.

In reality parallel mutations may be infrequent among human populations and geographical location may have little if any effect on the adaptive response of a human population to selection pressures [3]. This view is supported by the spread of sickle cell anemia.

The spread of sickle cell anemia does not support the Ralph and Coop [2] geographical based model for the advance of advantageous alleles across and within human populations. Sickle cell anemia has spread from Africa across Arabia into India. The Ralph and Coop [2] model of geographical parallel adaptation predicts that HbS would show diversity in Eurasia given the varied geography for each region.

The genetic evidence fails to support this prediction . It would appear that in Eurasia only the Arab-India haplotype is autochthnos to the geographical region. In fact the dominant HbS haplotypes in Eurasia originate in Africa.

In Omen for example, the three major haplotypes are 52% Benin, 26 % Arab-India and 21 % Bantu [5]. Some Bantu speaking slaves from East Africa were sold in Oman, but there were no slaves sold in Oman from the region where Benin HbS predominate.

We also find evidence of the Senegal haplotype in India among Tribal Dravidian speaking populations [6]. In many states the prevalence of sickle cell can range among the tribal population from between 10% -35% of the population [7].

This is interesting because the Tribal populations who primarially speak Dravidian languages are recognized as some of the earliest settlers of India This suggest a unicentric origin of the mutation before the Tribal populations separated [7].

The HbS chromosome haplotypes of the Indian Tribals were Arab-Indian with 25% of the haplotypes possessing the epsilon polymorphic site identical to the Senegal6b. The Senegal and Indian sickle cell share haplotypes [8]. The Arab-Indian and Senegal haplotypes share the C!T mutation at position -158 4,7.

In India the Benin HbS is the most common haplotype in western India. To account for the presence of this haplotype in India researchers argue that African slaves took this gene to India.

There are problems with this theory. The major problem with the slave trade solution for the transmission of the Benin haplotype to India, is that the African slaves in India are mainly of Somali-Ethiopian origin—not West African origin .

History provides a solution to the presence of African HbS haplotypes in Eurasia. The Dravidian population of India originated in Africa and belonged to the C-Group culture of Nubia [9].

The Dravidian people share cultural and linguistic features with Africans [10-14]. The archaeological evidence suggest that the Dravidian people belonged to the C-Group people of Nubia and migrated to India 5kya [9,10-11]. The Dravidian origination in Nubia, the original home of the Niger-Congo speakers who carry the Benin and Senegal HbS would explain the existence of African HbS haplotypes in India. These haplotypes in India suggest that they already existed among Dravidian and Niger-Congo speaking populations before they separated 5kya.

The earliest civilization in Southwest Arabia date back to the 2nd Millenium. This culture is called the Tihama culture which originated in Africa [13]. According to Fattovich [13], the pottery from Tihama Cultural Complex and other Ethiopian and Arabian sites shows similarities to the Kerma and C-Group pottery.

The presence of C-Group people in Arabia explains the affinity between HbS haplotypes in Africa and Eurasia. At Tihama and other sites in Arabia we find pottery related to the C-Group people of Nubia [13-16]. The Tihama archaeological evidence indicates that C-Group people, that included Dravidian and Niger-Congo speakers expanded from Nubia to Mesopotamia and the Indus Valley[11-14]. Through demic diffusion Dravidians spread sickle cell anemia across Oman into India.

In conclusion, the phenotypical expression of sickle cell anemia in Eurasia fails to be explained as a case of geographical parallel mutation as suggested by Ralph and Coop [2].The best explaination for the spread of HbS to Eurasia is the demic diffusion of Kushites not parallel mutation[17].

REFERENCE

[1] Wood TE, Burke JM & Riesberg LH. (2005). Parallel genotypic adaptation:when evolution repeats itself. *Genetica* 123(**1-2**): 157-170.

[2] Ralph, P and Coop,G. (2010). Parallel adaptation: One or many waves of advance of an advantageous allele. Genetics, 186: 647-668.

[3] Winters C. (2010).Can parallel mutation and neutral genome selection explain Eastern African M1 consensus HVS-I motifs in Indian M haplogroups. *Indian J Hum Genet*;13:93-6. Available from: http://www.ijhg.com/text.asp?2007/13/3/93/38982

[4] Bain , B. (2006). Haemoglobinopathy Diagnosis, Wiley-Blackwell .

[5] Daar S, Hussain HM, Gravell D. Ngel RL, and Krishnamoorty R.(2000). Epidemiology of HbS in Oman: Multicentric origins for βs Gene. *American Journal of Hematology* 64:38-48.

[6] Labie D, Rao S, Dunda O, Dode C, Lapoumeroulie C, Devi V, Devi S, Ramasami K, Elion J, Ducrocq R, Krishnamoorthy R, Nagel RL. Haplotypes in tribal Indians bearing the sickle gene: evidence for the unicentric origin of the αS-mutation and the unicentric origin of the tribal populations of India. *Hum Biol.* 1989; 61: 479-491. (s)

[7] Kate, SL.(2000). Health Problems of Tribal Population Groups from the State of Maharashtra, Immunohaematology Bulletin. Retrieved December 18 2009:http://sickle. bwh. harvard. edu/ India _ scd.html

[8] Rahimi Z, M. Karimi, M. Haghshenass, A. Merat .(2003).Globin gene cluster haplotypes in sickle cell patients from southwest Iran, *American Journal of Hematology*, 74 (**3**):156-160

[9] Lal BB. (1963). "The Only Asian Expedition in threatened Nubia: Work by an India Mission at Afyeh and Tumas". *The Illustrated Times,* London 20 April.

[10] Sergent , B. (1992). *Genèse de L'Inde.* Paris: Payot .

[11] Winters,CA. (2008). Origin and spread of Dravidian speakers. *International Journal of Human Genetics,* 8(**4**), 325-329.

[12] Winters ,CA.(2007). Did the Dravidian Speakers Originate in Africa? BioEssays,27(**5**):497-98.

[13] Fattovich ,R. (2008). The development of urbanism in the Northern Horn of Africa in ancient and Medieval Times. Retrieved 2/ 19/ 2008 http: //www. arkeologi.uu.se/afr /projects/BOOK /fattowich.pdf

[14] Keall, E. J. (2000).Changing Settlement along the Red Sea Coast of Yemen in the Bronze Age=, First International Congress on the Archaeology of the Ancient Near East (Rome May 18-23, 1998), Proceedings, (Matthiae, P., Enea, A., Peyronel, L. and Pinnock, F., eds), 719-31, Rome.

[15] Giumlia-Mair, A., Keall, E. J., Shugar, A. and Stock, S. (2002) Investigation of a Copper-based Hoard from the Megalithic Site of al-Midamman, Yemen: an Interdisciplinary Approach=, *Journal of Archaeological Science* 29, 195-209.

[16] Keall, E J. (2008)Contact across the Red Sea (between Arabia and Africa) in the 2nd millennium BC: circumstantial evidence from the archaeological site of al-Midamman, Tihama coast of Yemen, and Dahlak Kabir Island, Eritrea . Retrieved 2/20/08 at:http://72.14.205.104/ search?q=cache:SJPE _UY0VWUJ:www.dur.ac.uk/resources/mlac/arabic/RSPIabstracts02.pdf+keall,+Contact+across+the+ Red+Sea+(between+Arabia+and+Africa)+in+the+2nd&hl=en&ct=clnk&cd=1&gl=us

[17] Winters,C. (2010). The Kushite Spread of Haplogroup R*-M173 from Africa to Eurasia. Current *Research Journal of Biological Sciences*,2(5):294-299. Retrieved 16 October 2010: http://www.maxwellsci.com/print/crjbs/v2-294-299.pdf