
Sickle Cell disease is a condition which was exclusively a Black racial trait which has spread to various parts of the world through non-White racial migration - forced or voluntarily - and which has then manifested itself in populations where Blacks have either settled, or have mixed with elements of the local population. The following overview of sickle cell, its nature, origin and spread, has been culled from non-political, academic and medical sources. Included in the review is the relationship between malaria and sickle cell.

SICKLE CELL ANEMIA IS AN INHERITED BLOOD DISORDER


"Sickle cell anemia is caused by an error in the gene that tells the body how to make hemoglobin. The defective gene tells the body to make the abnormal hemoglobin that results in deformed red blood cells." - (National Institute of Health, Bethesda, MD. http://www.nhlbi.nih.gov/health/public/blood/sickle/sca_fact.pdf)

SICKLE CELL ANEMIA IS TRANSMITTED GENETICALLY, FROM PARENT TO CHILD

Sickle cell anemia is genetic. It can only be transmitted through direct genetic inheritance.

"Children who inherit copies of the defective gene from both parents will have sickle cell anemia. Children who inherit the defective sickle hemoglobin gene from only one parent will not have the disease, but will carry the sickle cell trait. Individuals with sickle cell trait generally have no symptoms, but they can pass the sickle hemoglobin gene on to their children." (National Institute of Health, Bethesda, MD. http://www.nhlbi.nih.gov/health/public/blood/sickle/sca_fact.pdf)

SICKLE CELL ANEMIA ORIGINATED IN AFRICA

"The beta-globin gene exists in a region of chromosome 11 called the "beta globin locus." The substitutions in the flanking regions of the gene (the haplotypes) show that Hb S arose separately at least four times in Africa, and once in Asia, possibly in India (Nagel and Fleming, 1992)." (Harvard University, http://sickle.bwh.harvard.edu/scdmanage.html)

Left: Analysis of the sickle gene in different regions of Africa and the Middle East showed that the gene arose several times independently. The haplotypes are named for the geographic regions where they were identified: Senegal, Benin, Central African Republic (CAR) and Asian (Middle East). The sickle cell genes spread over much of the world with migration. In India, the Asian haplotype is found almost exclusively. In the Americas, primarily the three major African haplotypes exist.

SICKLE CELL SUFFERERS EXIST IN LANDS WHERE BLACK INFLUENCE HAS BEEN FELT

"Sickle cell anemia is common among people whose ancestors come from sub-Saharan Africa; Spanish-speaking regions (South America, Cuba, Central America); Saudi Arabia; India; and Mediterranean countries, such as Turkey, Greece, and Italy." (National Institute of Health, Bethesda, MD. http://www.nhlbi.nih.gov/health/public/blood/sickle/sca_fact.pdf)

"Sickle cell disease is a worldwide health problem. In Africa alone there
are approximately 200,000 infants born with Hb SS. In the United States there are approximately 2,000 infants born annually. Therefore, Hb SS is a major health problem both in the U.S. and the world. Worldwide sickle cell disease affects individuals living in southern Italy, northern Greece, southern Turkey, the eastern province of Saudi Arabia, India, and equatorial Africa. Thus, as immigrants from all over the world arrive in the U.S., it is no longer just Africans who require medical management of SCD." – ‘Sickle Cell Disease: A Brief Overview’ By Ernest A. Turner, M.D. Director of the Comprehensive Sickle Cell Center, Meharry Medical College, Nashville, TN:
(http://blackhealthnetwork.com/articles/article.asp?articleid=26)

"I Haemoglobin S (Hb S) : This is extremely common in Africa, particularly in countries south of the Sahara (but not South Africa), and in some Asian Indian tribes. It is also found in areas where beta thalassaemia is common, such as the Middle East, northern India, Pakistan, Greece, Sicily and southern Italy, Albania, southern Turkey and southern Portugal." – (GEOGRAPHICAL DISTRIBUTION OF HAEMOGLOBIN DISORDERS, Bernadette Modell, Welcome Principal Research Fellow, Dept of Obstetrics and Gynaecology University College London Medical School http://www.thalassaemia.org.cy/articles/01Geog_Distrib.htm)

"In Hb Lepore-Washington-Boston, which has been commonly found in Italy, Spain and the countries from the Balkan peninsula, the breakpoint lies between codons 87 and 116a-87 aall6-146. The cross over breakpoint of Hb Lepore-Baltimore is between codons 50 and 86 aall-50 aall6-146 and this variant is quite often found in Brazil, Central Portugal and Italy [5]. Hb Lepore ab2 is one of the most common abnormal haemoglobin in Caucasians [5]. The majority of such variants i.e. Hb Lepore-Baltimore aall-50 aall6-146 and Hb Lepore-Washington-Boston a-87 aall6-146 are prevalent in Central Portugal and in the Spanish Alta Extremadura. (Case Report Complex interactions of db hybrid haemoglobin (Hb Lepore-Hollandia), Hb E (b b 26 G®®A ) and a a + thalassaemia in a Thai family, Vip Viprakasit, Parichat Pung-Amritt, Lerlugh Suwanthon, Kevin Clark, and Voravarn S. Tanphaichitr, all from the MRC Molecular Haematology Unit, Weatherall Institute of Molecular Medicine, John Radcliffe Hospital, Oxford, UK, and Haematology-Oncology Division, Department of Paediatrics, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand http://www.ejh.dk/articles/637accepted.pdf)

"Health Notes/Traditional Medical Practices: Common health problems in the Portuguese community are heart disease, high blood pressure, alcoholism and depression. Individuals from the south of Portugal may be carriers of sickle cell disease, often not diagnosed because patients are Caucasians." (Office of Refugee and Immigrant Health Refugees and Immigrants in Massachusetts http://www.state.ma.us/dph/orih/por1.htm)

"Sickle hemoglobin is found in people whose ancestors come from Africa, the Arabian States, South India and from countries around the Caribbean Sea, such as Puerto Rico, Cuba, Haiti and Jamaica, and also from countries around the Mediterranean Sea, such as Italy, Cyprus, Greece, Turkey, and Syria." – University of Rochester Medical Center, http://www.urmc.rochester.edu/genetics/brochures/sc.htm

SICKLE CELL CAME TO EUROPE VIA THE BLACK AFRICAN SLAVE TRADE

"Hb S is common in some areas of the Mediterranean basin, including regions of Italy, Greece, Albania and Turkey (Boletini et al., 1994)"
(Schiliro et al., 1990). Haplotype analysis shows that the Hb S in these areas originated in Africa. The genes probably moved along ancient trading routes between wealthy kingdoms in western Africa and the trade centers in the Mediterranean basin." (Harvard University, http://sickle.bwh.harvard.edu/scdmanage.html)

"Usually, people with sickle cell disease outside Africa (e.g., blacks in the United States) or India have mixed haplotypes for their sickle cell genes." (Harvard University, http://sickle.bwh.harvard.edu/scdmanage.html)

"Templeton gives a modern-day analogy: the presence of a gene for sickle cell anemia in Caucasians in Portugal. The gene traces back to a mutation that occurred in Africa and spread through interbreeding between Africans and Europeans. "The Africans didn't come up, reconquer the Iberian peninsula, kill off all the Europeans, and that's why there are sickle cell alleles in Portugal today," he says. The presence of the sickle cell gene in Portugal "means that Portuguese and Africans have met and they've interbred, just like humans tend to do." - "Out of Africa" - Ruth Flanagan, Contributing Editor, Earth Magazine, http://www2.mc.maricopa.edu/anthro/learning/origins/hominid_journey/outofafrica/OutofAfrica5.html

Sickle cell is thus transmitted genetically as a result of racial population movement. This holds true for all geographical regions:

"Sickle cell was taken with African slaves to North and South America and the West Indies in the 17th to 19th centuries, and as many as ten per cent of all black people in these countries now carry it. In more recent years it has been brought to Western Europe by migrants from the Caribbean and Africa, and is first becoming established in most industrial cities in the developed world. In Britain, about ten per cent of all Afro Caribbean and over 20 per cent of all Africans carry it. It is also found in the Indian, Pakistani, Cypriot, Italian, Greek and Portuguese communities, and very occasionally indeed in northern Europeans." - (GEOGRAPHICAL DISTRIBUTION OF HAEMOGLOBIN DISORDERS, Bernadette Modell, Welcome Principal Research Fellow, Dept of Obstetrics and Gynaecology University College London Medical School http://www.thalassaemia.org.cy/articles/01Geog_Distrib.htm)

"The migration from and exploration of Africa is a key reason for the spreading of the gene to India, Saudi Arabia, Spain, Southern Italy, and other Mediterranean regions. The slave trade was a major catalyst in bringing the gene to North America." - Howard University, Center for Sickle Cell Disease, http://www.huhosp.org/sicklecell/history.html

SICKLE CELL AND MALARIA

Often the argument is made that the reason why sickle cell is found in Southern Europe is because of the presence of malaria. The argument follows the line that sickle cell provides resistance against malaria, and by a process of natural selection, this syndrome then becomes present in large numbers in the population living in the malaria prevalent region.

This argument is false on two grounds: firstly because malaria was prevalent in all of Europe, not just southern Europe, yet the sickle cell syndrome is not found in all of Europe; and Secondly, because malaria can never by itself “cause” sickle cell to become prevalent - all it can do, through the law of natural selection, is allow a PRE-EXISTING condition to become dominant.

MALARIA PREVALENT IN NORTHERN EUROPE: YET NO SICKLE CELL INCIDENCE
Conclusive evidence that malaria per se does NOT cause sickle cell, comes in the fact that malaria was common all over Europe, including northern Europe, right up until the 19th Century. Despite this fact, sickle cell syndrome is unknown in the vast majority of the European population. The fact that malaria was present in these regions — and was only eradicated shortly before the disease was eradicated in all of southern Europe — makes nonsense of the allegation that the disease is prevalent in southern Europe only because of malaria.

The US based Center For Disease Control has the following to say about the incidence of malaria all over Europe:

“Until the second half of the 20th century, malaria was endemic and widespread in many temperate regions, with major epidemics as far north as the Arctic Circle. From 1564 to the 1730s—the coldest period of the Little Ice Age—malaria was an important cause of illness and death in several parts of England. Transmission began to decline only in the 19th century, when the present warming trend was well under way. The history of the disease in England underscores the role of factors other than temperature in malaria transmission. There are numerous accounts of malaria in all the northern European countries in the 18th and early 19th centuries. The wealth of records in this period confirms that the disease was common at many coastal sites in England and in some parts of Scotland, with occasional transmission as far north as Inverness (57°20’). The northern limit was roughly along the 15°C July isotherm—not the 15°C winter isotherm, as stated by some authors. Thus, there was endemic transmission in southern Sweden and Finland, with occasional devastating epidemics that extended to the northern end of the Gulf of Bothnia, close to the Arctic Circle. In North America transmission occurred throughout most of the United States and in some parts of Canada.

In 1827, John Macculloch wrote "We may take the average of life among ourselves, in round numbers, at fifty with sufficient safety for this purpose. In Holland it is twenty-five; the half of human life is cut off at one blow, and the executioner is malaria, for there is no other cause for the superior mortality of that country."

- "From Shakespeare to Defoe: Malaria in England in the Little Ice Age" by Paul Reiter, Centers for Disease Control and Prevention, San Juan, Puerto Rico http://www.cdc.gov/ncidod/eid/vol6no1/reiter.htm

NATURAL SELECTION, MALARIA AND SICKLE CELL

The erroneous belief that malaria "causes" sickle cell is based on a misunderstanding of the concept of natural selection. Natural selection occurs when environment factors allow a PRE-EXISTING genetic string to become dominant. It does NOT “create” new genetic strings.

A theoretical example will illustrate the point: if a disaster had to occur with the earth’s ozone layer, and only people with red hair could survive the disaster, then, fairly obviously, all people without red hair would die off, and the only survivors would be red-haired people.

This is natural selection at work: it would allow only people with red hair to survive. The gene for red hair would then become dominant amongst humans, NOT because it was new, but because it was best suited to survive the changed environmental conditions.

Natural selection is therefore where environmental conditions change so that only certain pre-existing traits survive. Natural selection does NOT “create” new genetic strings, it merely lets certain pre-existing strings become dominant.

This theoretical example is exactly what happened with malaria and sickle cell. As the sickle cell condition does indeed provide a measure of protection against
malaria, those populations in which sickle cell was already present, saw an exponential growth in the number of sickle cell sufferers, as, obviously, those without the gene died off from malaria.

However, and this is the key, the sickle cell gene had to be pre-existing in the affected populations. Malaria did not “create” the sickle cell gene, it merely allowed it to become prevalent in groups which already possessed the gene. As discussed above in the origin of sickle cell, modern haplotype analysis has shown that the sickle cell syndrome is native to Blacks in sub-Saharan Africa, and that the presence of this disease in Europe and elsewhere has been conclusively tracked back, through genetic research, to Africa itself. There is thus no evidence to show that sickle cell was pre-existing in any pure White population group, and all attempts to link the genetically based condition exclusively to malaria, are pure invention.

SICKLE CELL DOES NOT VANISH EVEN IF MALARIA DOES

“Carriers who migrate to non-malarial countries no longer need their natural protection against malaria, but the traits do not disappear. Even though they are redundant as protection against malaria, the thalassaemia and sickle cell genes continue to be handed down in families for generations, just like skin colour or hair colour.” - GEOGRAPHICAL DISTRIBUTION OF HAEMOGLOBIN DISORDERS, Bernadette Modell, Welcome Principal Research Fellow, Dept of Obstetrics and Gynaecology University College London Medical School, http://www.thalassaemia.org.cy/articles/01Geog_Distrib.htm

It is of course an incorrect presumption that all people of Mediterranean origin are equally exposed to the risk: in reality, people of pure White Mediterranean extraction run no risk at all of having the disease, as only individuals of racially mixed ancestry can be carriers - and not all Mediterranean types are of mixed ancestry, as discussed in other sections of this appendix.

CONCLUSIONS

1. Sickle cell anemia originated amongst the sub-Saharan Black African population.
2. It is transmitted genetically - that is, from direct parent-to-child.
3. The incidence of sickle cell in Southern Europe is directly linked to the absorption of sub-Saharan Africans into elements of the native White populations. There is no other way the disease could have spread into these regions. The spread of the disease into Turkey, Saudi-Arabia, India and elsewhere in Asia is explained by the more than 1000 years of Arab slave trading into these regions.
4. Sickle cell is NOT caused by malaria. This is proven by the fact that malaria was prevalent in all of Europe, right into Scandinavia, yet sickle cell is unknown in these regions. All that malaria does, is by way of natural selection, allow pre-existing strains of sickle cell to grow in numbers. Modern genetic analysis of haplotypes has proven conclusively that sickle cell is native to sub-Saharan Africa - even the types of sickle cell are named after regions in Black Africa.
5. The genetic footprint left by sickle cell therefore conclusively shows the accuracy of historical events which led to the absorption of non-White races into elements of the previously all-White populations.
6. While it is of course obvious that this racial mixing did not affect all members of these societies, it did affect enough to hamper the development of parts of those societies, and laid those lands open to further incursions by other groupings, White and non-White.
Black African Genetic Footprint: Sickle Cell Disease
Part 3: Racial Mixing
Brought the Hemoglobin D disorder to Britain and Ireland
Part 4: The Mendelian Laws of Genetics – dominant and recessive racially mixed genes
Part 5: European Footprint: Hereditary Hemochromatosis – a genetically inherited disease
Part 6: Genetic Evidence of Avar and Hunnish Admixture in Central Europe
Part 7: Western European Genetic Remnants in Egypt
Part 8: Genetic Evidence of Racial Mixing in Greece
Part 9: Genetic Evidence of Racial Mixing in Italy
Part 10: Genetic Evidence of Racial Mixing in Portugal
Part 11: Genetic Evidence of Racial Mixing in Spain
Part 12: Genetic Homogeneity in Poland
Part 13: Genetic Homogeneity in Norway
Part 14: Finland, the Lapps and the Tat-C Controversy
Part 15: Y-Chromosomes as Racial Markers