THE BRAZILIAN ASSOCIATION FOR CREATION RESEARCH

Recently we have received some news about the *Associacao Brasileira de Pesquisa da Criacao*, the Brazilian Association for Creation Research.

The Association was formed in July, 1979. Since 1980 it has been circulating, on a monthly basis, Creationist articles to about four hundred members all over Brazil.

We have known for some time that Creationist work was going on in Brazil. No doubt the Association will be able to be effective in coordinating the work and in making it apparent to many that Creation is the best explanation of the world and the things in it.

The address is: Associacao Brasileira de Pesquisa da Criacao, Caixa Postal 37, 36570—Vicosa, Minas Gerais, Brazil.

QUOTABLE QUOTE

"History is full of instances in which people refuse (sometimes unconsciously but not always) to make an observation which disagrees with a preconceived notion . . . Equivalent errors can be made in interpretıng data . . . usually by over-interpreting or ignoring part of the data. The story of the discovery of Pluto illustrates an example of this problem.

After the discovery of Uranus . . . astronomers observed the orbit . . . A discrepancy was found which J.C. Adams in England and U.J. Leverrier in France independently interpreted as a consequence of an eighth, as yet undiscovered, planet. . . . Neptune was found . . . most of the discrepancy in the orbit of Uranus was removed. Still agreement was not perfect . . . a ninth planet was predicted to exist by Percival Lowell in 1906. In 1930, . . . Pluto was discovered . . . near the predicted position. Further observations showed, however, that Pluto was too small to have a significant effect on the orbit of Uranus and finding it near the predicted position was sheer luck. The discrepancies on which Lowell had based his calculations were due to inaccuracies in the observations. His prediction of the existence of another planet was based more on his desire for another planet to exist than on the gathered data."

Dearborn, David S., 1978. Tales of the travels of Newton Schwartz. *The Astronomy Quarterly* 2(8):229-236. (The quotation is from p. 234.)

THE SICKLE CELL TRAIT

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The sickle cell story is often put forward as a demonstration of evolution. Here recent medical knowledge concerning the trait is reviewed. It is seen that in sickle cell nothing has evolved, improved, or increased in complexity. All that happens is that in this case protection against one disease, malaria, is conferred by another disease, sickle cell.

History of Sickle Cell and Malaria

Among infectious diseases malaria is one of the greatest scourges to ever affect mankind. In the fifth century before Christ, Hippocrates differentiated the disease into fever occuring daily (*falciparum*), on alternate days (*vivax*), on every fourth day (*malariae*), and recognized the association of malaria (bad air) with stagnant swamps and marshes. Malaria was one of the causes of the decline of the Roman Empire. And during World War II, Pacific malaria-battle casuality ratio was 8-1 in American troops in New Guinea and the Solomons.

Malaria is an infection by the protist *Plasmodium falciparum* which is carried to man by the Anopheline mosquito. Introduced during the mosquito's bloodmeal, the parasite proceeds through asexual stages within human erthrocytic (red blood cells, rbcs) and non-erythrocytic tissues.

Sickle cell refers to rbcs that are not shaped like normal bi-concaved discs but rather appear elongated, C-shaped, or sickled. The sickling is produced because of a genetic defect that codes for an amino acid subsitution (valine for glutamic acid in the beta chain's sixth position) in hemoglobin protein. Thus this abnormal hemoglobin has a tendency to precipitate into tactoids when de-oxygentated deforming the rbc. Those having both sickle genes (SS) have Sickle Cell Anemia (1/500 or within 50,000 of the nations 22 million Blacks) that causes vascular occlusion, infarction, premature rbc destruction, ischemia, pain, dysfunction, necrosis,[1] and with many death in childhood.[2]

Sickle Cell was first described clinically by Dr. James Herrick, a Chicago physician in 1904. In 1949 Pauling and associates found through electrophoresis a way to differentiate the hemoglobins. This led to Sickle Cell being the first demonstrable molecular disease. The precise protein change between the hemoglobins (Hbs) came to light in 1959 when Vernum Ingram devised partition chromatography, the technique for detecting amino acide differences in proteins. Today so much is known about the molecular arrangements within Sickle Cell that it has been called the first 'atomic' disease.

Case for Evolution

Sickle Cell has been stated to be the best example that students of human evolution have of genetic adaptation to an infectious disease.[3] In *Scientific America* there is

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this statement, "For anyone interested in population genetics and human evolution, the Sickle Cell story presents a remarkably clear demonstration of some of the principles at play. It affords, for one thing, a simple illustration of the principle of hybrid vigor."[4]

Now we will examine, perhaps, why some scientist offer such comments. With the high mortality rate seen among homozygotes for the sickle cell gene, the frequency of this mutant gene would be expected to decline within a population's gene pool. Because among some African communities a stable 20% have the gene (as trait carriers, Hb AS) then a beneficial relationship must exist.

Allison in 1954 first reported the association between the malarial parasite Plasmodium falciparum and resistance to this infection in the presence of abnormal Hb AS.[5] In malarial environments three genotypes often exist, the normal Hb AA which suffers malaria infection, the Hb SS that die from the anemia. and Hb AS heterozygotes which seem able to survive in this situation. The estimated fitness of Hb AS in malarial environments is 1.25 times over the normal Hb AA.[6]

The authors in *Pathology of Sickle Cell Disease* point out that there are two outstanding explanations for the persistance of the sickle gene in the face of strong negative natural selection, (1) a balanced polymorphism (i.e. the heterozygote AS survives, while the homozygotes AA and SS face difficult experiences), and (2) a high spontaneous mutation rate.[7] Along these lines the literature states, ". . . the Sickle Cell situation shows that mutation is not an unmixed bane to the human species . . . Similarly other mutant genes that are bad in one situation may prove beneficial in another. Variabilty and mutation permit the human species, like other organisms, to adapt rapidly to new situations."[8]

How does Sickle Cell Trait, SCT, or those with Hb AS receive protection against malaria? In these individuals with malaria, it was observed that the parasite caused rbc's to sickle faster. The parasite lowers the pH of the infected cell by about 0.4 pH units which can increase sickling about 20-fold[9] (normally 2-4% sickled cells are present). With the decreased pH about 40% of the infected cells will sickle. These rbc's are sequestered in low-O_2 vascular environments[10] and phagocytosized[11], killing in the process the parasite. Thomas H. Maugh in *Science* remarks, "The effect of the mutant gene is thus not completely protective, but it does lessen the severity of the disease and may be sufficient to prevent death."[12]

The importance of this example is emphasized as noted again from *Pathology of Sickle Cell Disease,* "The heterozygous state (AS), on the other hand seems to be dichotomous, being detrimental under some conditions and protective under others . . . The relationship between SCT and infection lies at the crossroads of genetics, evolutionary theory, ecology, hematology, epidemiology, and preventive medicine."[13] And finally these comments by the biochemist Lubert Stryer, "The finding of mutant hemoglobins has enhanced our understanding of evolutionary processes. Mutations are the raw materials of evolution and the studies of sickle-cell anemia have shown that a mutation may be simultaneously beneficial and harmful. The disease of an individual may be a concomitant of the evolutionary process . . .

Subjectively, to evolve must most often have amounted to suffering from a disease. And these diseases were of course molecular. The appearance of the concept of good and evil, interpreted by man as his painful expulsion from Paradise, was probably a molecular disease that turned out to be evolution."[14]

Sickle Cell Trait

In August of 1980 there was an unfortunate report of a previously healthy football athlete at Abilene Christian University having died from a Sickle Cell Trait crisis.[15] Dr. Tames I. Duff in Abilene, with Clinical Pathology Associates, stated in his "Protocol of Necropsy" about this case, "Sudden death associated with SCT (all organs showed distention and plugging of vessels with masses of sickle cells)."[16]

In 1970 Colorado University also had an athlete experience a similar SCT crisis; and they presently, along with several Texas Universities, perform blood tests on their Black athletes for the trait.

Between March 1968 and February 1969 four deaths

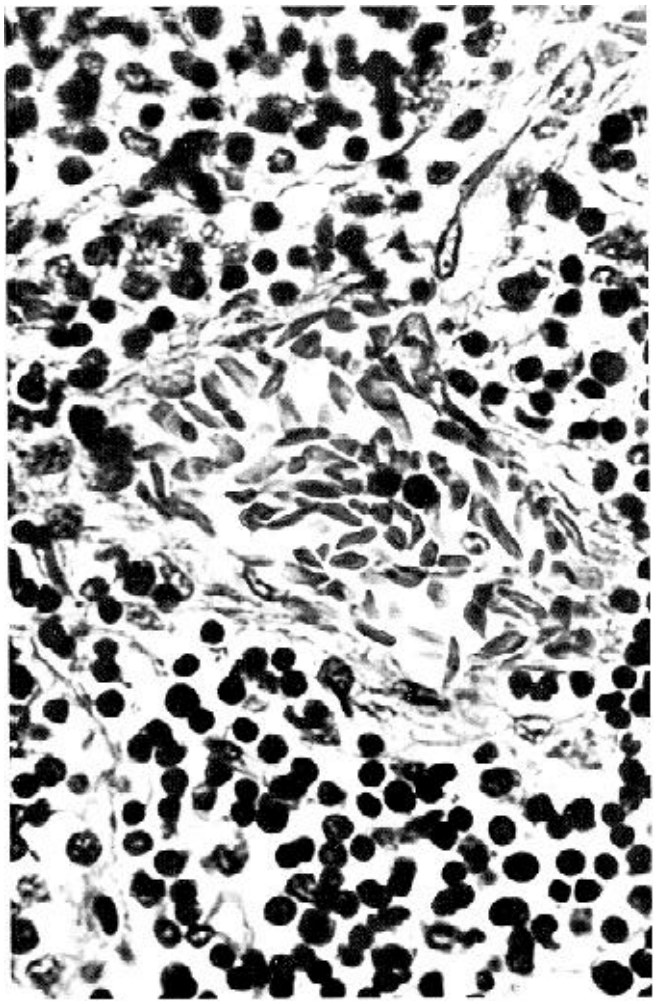

Figure 1. Lymph node in a patient with SCT crisis. Notice the sickled RBC's within the blood vessel.

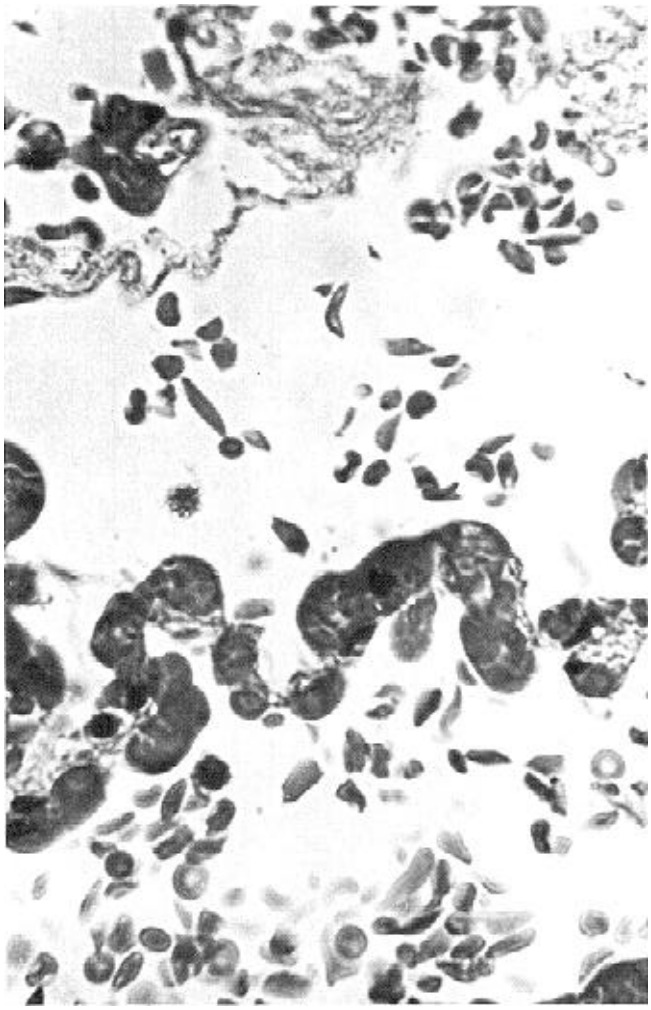

Figure 2. Lung in a patient with SCT crisis. Notice the extravagated and sickled RBC's in the respiratory spaces.

were attributed to SCT crisis among 4,000 Blacks at an Army basic combat training post. In his investigation on these incidences Dr. Stephen R. Jones commented, "it is generally not appreciated that certain complications of SCT may be fatal."[17] And between 1970 and 1974 four recruits experienced SCT crisis, one leading to death, at two Air Force installations. Drs. Koppes, Clotman, and Butkus in their report in the *American Journal of Medicine* state, "The hypothesis is then developed that rhabdomyolysis and endotheial damage, terminating in severe coagulopathy, may more likely occur in patients with sickle cell trait who are subjected to vigorous physical exertion."[18]

A growing concern over the proper view of SCT is reflected throughout medical literature in varied opinions. The 1979 *Textbook of Medicine* says about the trait, "Carriers are discovered only as a result of routine testing or surveys, and should be assured of the trivial nature of the abnormality as well as its genetic implications."[19] Other sources are now reporting, as does Dr. Robert M. Nalbandian the noted author of many works on Sickle Cell, "the heterozygous state for S Hb has been shown to be responsible for significant clinical disease and even death and should no longer be considered an asymptomatic state."[20]

This paper will now review some of the clinical and medical manifestations that may be found in the SCT carrier. Recent evidence on the trait has now caused some alert among the medical community as the authors of *Sickle Cell Hemoglobin* express, "There is a perceptive and growing body of information showing that the heterozygous S state is not without risk and hazard under conditions of physiological stress."[21]

There is the strong suggestion that acute intravascular sickling in persons with SCT may constitute a serious threat to life, since the sickling crisis may be precipitated by mild illness or physiologic stress in an otherwise healthy person.[22]

What conditions precipitate a SCT crisis? Factors known to be important in initiating and hastening sickling include hypoxemia, acidosis, dehydration, and the presence of reducing agents such as lactate.[23] Once sickling begins the process can have a cyclic effect in that occlusion of capillary beds by the deformed rbc's causes blood stasis and thus hypoxemia and acidosis that induce additional affected blood cells to sickle. Also entrapment of sickled cells in lung capillaries causes blood to be shunted which adds to the hypoxemia.

The following is a list of clinical manifestations attributed to SCT; however, many individuals who are trait carriers may live totally unaware of their condition. Carriers have been shown to have greater risks with local or general hypoxemia in general anesthesia, pulmonary infarction at both high altitude and at sea level, splenic infarction, massive hematuria (bloody urine), renal papillary necrosis, renal medullary necrosis, renal failure, retroperitoneal fibrosis, higher incidences of leg ulceration, lower birth weight in infants of SCT mothers, retinal arterial occulsion following ocular trauma, and significant increased associations between maternal SCT carriers and perinatal mortality when mothers were subjected to anoxic stress.[24]

In *Sickle Cell Hemoglobin* other impairments are mentioned, "Aseptic femoral head necrosis, superior longitudinal sinus thrombosis, and serious ocular pathology."[25] Carriers are reported to have higher incidences of Salmonella infections.[26] Although SCT may infer some protection against leprosy, it also predisposes carriers to acute exudative tuberculosis.[27] There appears to be a high correlation between pulmonary emboli, oral contraceptives, and SCT.[28] And following minor leg infections, carriers often have recurrent ulcers because of impaired healing.[29] Dr. David A. Sears writing in the *American Journal of Medicine* includes increased abnormalities within SCT carriers for splenic infarction, bacteriuria, phelonephritis in pregnancy, and in his discussion adds, "Even those who minimize the risks of AS hemoglobinopathy suggest that certain real hazards do exist for carriers of the trait."[30]

A large controversy rages over suggested correlations between SCT carriers and physical/mental abilities. Drs. Bowman[31], Orball, and Be1l[32] oppose the findings of Michael K. McCormack, Ph.D., who in a controlled study compared 19 carriers to 155 same-sex Blacks and

showed lower scores in maturity of skeletal age and in 4 of 5 intellectual measurements.[33]

In the McCormick study of 120 autopsies with SCT, examination at necropsy showed that in 17.5% of the cases sickling was either the cause of death or a major contributing factor.[34]

Within the military the Department of Defense, DOD, issued a directive that all incoming personnel will be tested for Hb AS as part of an occupational medicine program. (Dr. Nalbandia even suggests screening all AS individuals for the purpose of occupational counseling that would emphasize avoiding physiologically stressful careers.)[35] The DOD does not regard those with SCT to be diseased but fit for all military duty except for certain restrictions of assigment that might induce hypoxemia.[36] See Table 1.

In the Air Force Academy no individuals with SCT can qualify for admission; however, they may join the Air Force ROTC. Table 2 is a compilation of evidence from the *American Journal of Medicine* article entitled "Exertion-Induced Rhabdomyloysis, with Acute Renal Failure, and Disseminated Intravascular Coagulation in Sickle Cell Trait" regarding the four recruits mentioned previously experiencing SCT crisis in their training.[37]

There is however the study in 1976 on the National Football League which showed that of the 579 Black athletes involved only 6.7% had SCT with no reports of crisis.[38] This 6.7% is below the national average which is approximately 8%.[39] And in a 1974 article, examination of Olympic Black athletes showed only 11 out of 760 (1.4%) had SCT and yet world-class performances were turned in.[40] It is interesting conjecture at this point to note the low percentage (1.4%) of SCT participants considering 1/13 Black Americans have the trait, with higher ranges (over 20%) in certain African populations, and up to a 30% frequency among certain people of southern India.[41]

Table 1

Army	Air Force	Navy
Flight Duty	Flight Duty	Flight Duty
Diving Duty		Diving Duty*
Airborne Duty		
Ranger Duty		

*includes parachuting

To conclude this section three comments are worth mentioning. From *Sickle Cell*, "While individuals with the trait are generally asymptomatic under normal physiologic conditions, intravascular sickling, vaso-occlusion, and infarction can occur if oxygen tension is sufficiently lowered."[42] These effects may be witnessed in states of hypoventilation, anesthesia, unpressurized high altitude flying (note conflicting reference article 43), exercise at high altitudes,[44] pneumonias,[45] shock,[46] and onset of stressful physical exertion.[47] Dr. Stephen J. Jones offers in his summary statements, "Why sudden death occurs in only a few who possess sickle cell trait is puzzling but may be explained by the relative amount of S-Hb present—the greater the amount, the greater the facility for sickling."[48] And finally, it has been considered that any presence of sickle gene as in SCT should be considered under the broader term of 'Sickle Cell Disease.'[49]

Sickle Cell Gene

In this section several points concerning the mutant gene for Sickle Cell will be discussed. First it must be

Table 2
Clinical Manifestations and Conditions in Military Personnel with SCT Crisis[37]

Date	Case 1	Case 2	Case 3	Case 4
Age, sex	19, M	19, M	19, M	17, M
Race	Black	Black	Black	Black
Past Health	Good, 18th day of training	Good, 25th day of training	Athlete, well conditioned	Athlete, well conditioned
Altitude (ft)	661	661	7,200	7,200
Location	San Antonio, Tex	San Antonio, Tex	Colorado	Colorado
Ambient Temperature	79 °F	83 °F	57 °F	75 °F
Presentation	Mile run, severe muscle cramps and swelling	Obstacle course collapse, severe muscle cramps and swelling	2 mile run collapse, muscle cramps and swelling	Obstacle course collapse, severe muscle cramps and swelling
Pulse rate	88	110	140	128
Hemoglobin (%)	39 S 60 A	42 S	37 S 62 A	40 S 58 A
Bleeding	Hemoptysis, GI tract	Intravenous sites	not clinical	4+ guaiac stools
Urine	Color brown	Color brown	Color red	Color brown
Outcome	Normal recovery	Death, hyper kalemia and hemorrhage	Normal recovery with elevated temperature for 6 wks	Normal recovery with elevated temp. for 6 wks

emphasized that although hemoglobin is specifically designed to carry molecular O_2, it seems capable of performing this function throughout highly varied forms. Research is continuing to find new alterations in hemoglobin (in 1970, there were 59 known varieties of the alpha and beta chains,[50] with some 30 forms known to be less stable than normal Hb AA[51]).

Recent evidence using recombinant DNA techniques have shown that there are two distinct sickle genes that arose apparently from independent mutations in malaria-prone areas.[52] One gene arose in West Africa and spread north; and a second gene originated in East Africa and spread to the Middle East and Asia.

However, other archeological and anthropological data conclude that the sickle gene may have arisen from a population in the Fertile Arabia area.[53] David M. Kurnit in his paper "Evolution of Sickle Variant Gene"[54] reports that the gene arose only 60-80 generations ago with a change of agricultural techniques in Africa some 1500-2000 years ago.

The appearance of the sickle gene in populations of Italy, Sicily, Greece, East Africa, West Africa, Southern and Southeast Africa, and along tropical islands, represents cross cultural, racial and geographical occurances for the mutant gene. Because of this distribution the authors of *Pathology of Sickle Disease* state, "It has thus become increasingly difficult to explain the distribution of SCT on the basis of ancestral origins and racial affinity alone."[55] And the maximal estimate given for sickle gene mutation is high, 1.7×10^{-3} per gene per generation.[56] This evidence allows speculation that here in action is a highly mutatable gene locus that may have allowed independent and isolated aberrations to occur.

And sickling is not restricted only to humans. To mention only a few known examples, it occurs among rachitic white rats, guinea pigs, and white-tailed deer.[57]

Nor is the mutant Hb AS the only abnormal Hb or condition associated with resistance to malaria. Also implicated in resistance are Hb C in West Africa, Hb E in South-East Asia, beta-Thalassaemia in Near East, South-East Asia and Africa, and Glucose-6-Phosphate Dehydrogenase deficiency among tropical and subtropical regions.[58] Protection is also seen in fetal Hb,[59] in conditions of reduced ATP content in rbc's,[60] and in Latin America the absence of the Duffy substance from rbc membrances has recently been shown associated with protection.[61]

When Blacks were first brought from Africa to America some 250-300 years ago, the estimated frequency of SCT was 22%.[62] Although America once had endemic areas of malaria, it was eradicated through control of the breeding places for the vector *Anopheles*. Removal of malaria or movments of populations into non-malarious areas is associated with a reduction in gene frequency for AS.[63] This decrease has been witnessed in this country where the SCT frequency has dropped from the original 22% to the present 8%.[64]

How is this reduction explained? First, back mutation to normal hemaglobin is admittedly too slow a process for so rapid a change. Secondly, dilution effects in the gene pool may have played a role and have been investigated. A recent study used the Duffy allele as a gene marker, for this gene is believed to have no known causes for selection and it is in high frequency among Northern European Caucasians but totally absent in African Blacks. Admixture between these two groups revealed interesting frequencies for the Duffy allele which ranged from 26% among Detroit Blacks to 3.7% in Charleston, S.C.[65] Northern Blacks appear to have had higher admixtures in their ancestry than Southern Blacks. However Dr. Phillip Herrick of the University of Kansas found elevated frequencies for two gene loci that did not experience dilutional effects from expected admixture results. These two gene sites are known to undergo selection and are—Glucose-6-Phosphate Dehydrogenase deficiency and Hb AS.[66]

The third explanation, and what appears to be the most proper, is the genetically demonstrable increase in the number of those with Hb AA over Hb AS. The AS individual is slightly disadvantaged within this country with a fitness estimated to below the AA normal.[67] And the incidence of the AS trait may continue to decrease until equilibrium is reached with spontaneous new mutations.

Questions and Problems

1. Why the decrease of SCT frequency in this country?

This decrease is most significant because if this single amino acid mutation is a major and prime example of evolution then several points need clarification. The environment changed (no malaria in this country) and the gene S frequency quickly began to decrease. But if evolution had occurred, if improvement of the organism, if higher order, if greater complexity, if new information, if all of these or any one of these advantages had arisen—then why the drop-out? If an organism is going to have a mutation and then struggle through selection to establish that new gene only to have the environment change, and then this new event proceeds to remove this important phase of its evolution—how will anything advance? For natural selection on an evolutionary scale requires an environment that is constantly changing and challenging. Evolution must surely involve the retention of the newly gained segment of information in one situation in order to build upon that new order for the next challenge. In this regard Sickle Cell apparently fails.

2. Was new information added with the S mutation?

The only 'evolutionary' change that took place in Sickle Cell was one amino acid substitution; but Hb and the red blood cell looked at independently show no new information. Hb was not made to function better in its important capacity of transporting O_2; and the rbc structure was certainly not improved. In fact, the design seen within the rbc was itself drastically altered so that deformed cells were eliminated from the system; or else the sickling caused pathological situations with occlusions, infarctions, crisis in SCT, and death in anemia.

Creationists raise serious thoughts concerning this and the above questions, as, "The evolutionist is also challenged to show not merely that the alleged mutation survives, but more importantly that it has increased the information content of the organism or population regardless of environment."[68]

3. Does SCT represent an example of 'hybrid vigor'?

Hybrid vigor or heterosis is most often defined as the offspring's increase in size, yield, and performance over the parental types. Within this frame of reference it is difficult to have SCT comply. Previously cited are many examples of medical problems that can be faced by the carrier. And this theme is repeated: "morbidity and mortality resulting from sickle cell trait has been recognized with increased frequency during the past two decades . . ."[69]

It is acknowledged that SCT is but one of many conditions that may infer protection against malaria. And Hb S, unlike normal Hb A, when combined with other Hb variants is often seen with increased, not decreased, clinical disease. As in the doubly heterozygous state SC which causes a serious hazard and jeopardy for the neonate.[70] De-oxygenated AS shows 1 ± 0.46 c.p. units increase in blood viscosity over AA de-O_2,[71] which can cause complicating hemodynamic problems.

And this point must be rendered that no matter the environment (malarial or non-malarial) these with 'hybrid vigor' will still reproduce offspring of which one quarter are born too defective to survive (SS).

4. Why does the more primitive type of Protozoa infect man?

Plasmodium have been shown to infect (this is an incomplete list) man, birds, monkeys, lizards,[72] *p. falciparum* occurring in man, birds,[73] and monkeys.[74] It is curious that all bird and amphibian blood protozoa have typical protozoan mitochondria, double membraned with the intra-mitochondrial structure composed of microvilli. All mammalian malaria parasites are deprived of typical mitochondria but rather have a two-membrane structure similar to bacteria mesosomes.[75]

The researchers involved in this evidence comment, "The reappearance of this primitive type of mitochondrion would fit well into this evolutionary hypothesis and would apply also to Plasmodia where the parasitic way of life might be connected with a loss and then reappearance of certain organelles. It is difficult, however to explain the presence of typical mitochondria in bird malaria parasites and of a primitive type in the closely related mammalian species; as both groups belong to the same genus, Plasmodium, and both are intracellular parasites inhibiting erythrocytes."[76]

This indeed seems peculiar within an evolutionary scenario, considering that the more primitive amphibians and birds host the more advanced form of protist, while man (the relative newcomer) is infected by the supposedly more primitive parasite.

5. How should we view the recent origin of the sickle mutation?

Generally overlooked here (but especially by evolutionists) is the massive and complex interplay between this triad of man, mosquito, and protist. In the evolutionary view, mosquito and protist have struggled on this planet hundreds of millions of years before man; and their life cycles would certainly have necessitated satisfactory relationships before his arrival. Man has supposedly been *Homo sapiens* for the last 2 MY-250,000 years, depending upon which authority you address. Yet it is only within the last 10,000-5,000 years that man is truly seen with agriculture and development. Creationists contend that man and agriculture have always been contemporary, and that recent origins for most matters involving man are to be expected. The total combination then of man, the clearing of tropical regions for agriculture, the endemic spread of malaria, and the mutation (a prediction from the Fall of Man with the advent of disorder as seen in the 2nd Law of Thermodynamics) allows us to see recent natural events as consequences of a hostile environment for which and to which man must be responsible.

6. Was the S mutation purely random?

There is much that is unknown about mutations, and the vast majority of what our knowledge points out is that in most mutations random processes alone are responsible. But in the case of Sickle Cell could other factors be involved?

Mutation research is a scientific hot-bed and the list of naturally occurring mutagens is rapidly expanding.[77,78,79] Toxic influences exist in malaria[80] and cholesterol products, the main sterol of malaria parasites,[81] in autoxidation processes have recently been shown to be frameshift mutagens.[82]

This broad speculation also stems from the occurrence of the sickling phenomenon as seen in different races of man, arising independently in different geo-regions, and from its presence in different animals, coupled with our most incomplete knowledge of the inter-relationships between parasite/host and mutagen/mutation. New discoveries are being made as this statement from *Pathologic Basis of Disease* declares: "The evidence grows stronger that the chromosomal aberrations are not merely random but affect particular chromosomes and specific loci within the karyotype, and indeed, in experimental neoplasms, some relationship between the inducing carcinogen and the cytogenetic change is beginning to emerge."[83]

Discussion

Most evolutionists, including the noted genetist J.B.S. Haldane, view disease as one of the most important agents of natural selection in human evolution. Darwin recognized a role for disease in human evolution.[84] Statements are often encountered as, "The existence of any disease is the expression of a transient evolutionary process that ought to be explainable."[85]

Stuber has already stated that the origin of good/evil itself most probably evolved as a consequence of a molecular disease. Allison makes this comment in his "Sickle Cell and Evolution," "Finally, the sickle cell findings, offer a cheering thought on the genetic future of civilized man. Eugenists often express alarm about the fact that civilized societies, through medical protection of the ill and weak, are accumulating harmful genes: e.g., those responsible for diabetes and other hereditary diseases. The sickle cell history brings out the other side of the story: improving standards of hygiene may also eliminate harmful genes—not only the sickle cell but also others . . ."[86]

However if disease is one of man's most important evolutionary prods; then the question arises what defined roles have preventive medicine, health organizations, and medical research had in attempting to

eradicate the very challenges that will purify our species, strengthen the fittest, and elevate man to higher complexities. Without the rudder of disease will not man be adrift in the directionless swirl of an evolutionary sea?

A Creationist view of disease, however, might regard cases such as Sickle Cell as natural consequences of the processes in force since the Fall of Man. Disease will not improve the organism or the population, nor is it a transient evolutionary process. For, as Creationist may predict, disease states will increase with time, via chance and accumulation, without intervention by man, i.e., the intelligent use of the wisdom endowed to him by the Creator.

Man's proper view of disease (as most naturally witnessed in the world) should contain two major elements, compassion and responsibility. With compassion man will continue to seek to alleviate suffering and with responsibility man will direct energy and talents toward improving his dominion over this world. But in this regard man has not been left alone, for by following the Creator's plan for life one can truly eliminate a major host of illness and disease.[87]

In conclusion, this paper has attempted to look in depth at the entire problem encountered by SCT. The individual with the trait should not be made alarmed but rather aware of the special conditions and genetic consequences involved with this condition. Medical personnel might heed this statement by Drs. Nalbandian and Murayama, "It is poor medicine and perhaps irresponsible to mislead sickle-cell trait individuals to believe that they are as invulnerable to unusual or severe physiological stresses as non-S trait individuals."[88]

Sickle Cell Trait and malaria may probably best be viewed as one less serious impairment causing non-expression of a major and more serious injury. Dr. Gary E. Parker[89] expressed this same idea and comments from a letter with Dr. Duane T. Gish are most enlightening. "While the sickle cell gene, when paired with a normal gene, is a benefit to the individual who is infected with malaria, it has no overall benefit to the population. Possession of one of these genes does increase the survival rate to malaria, but such individuals survive to reproduce, increasing the number of individuals dying of sickle cell anemia, so the number of individuals who survive malaria as a result of carrying this trait is balanced by the increase in the number of individuals who die of sickle cell anemia. There is thus no overall benefit to the population. It is the rare situation in which one injury, namely the sickle cell trait, helps the individual to survive another injury, namely infection by malaria."[90]

And finally, perhaps, evolutionists themselves are beginning to tire of the case presented by sickle cell trait. In his defense of the evolutionary neoclassical theory against Darwinism, Lewontin has pointedly remarked, "So it is no use trotting out that tired old Bucephalus, sickle-cell anemia, as a proof that single locus heterosis can exist. Anyone who has taught genetics for a number of years is tired of sickle cell anemia and embarassed by the fact that it is the only authenticated case of overdominance available. 'If balancing selection is so common,' the neoclassicists say, 'why do you always end up talking about sickle cell anemia?'"[091]

Acknowledgements

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A CRITIQUE OF AFAR MAN AS THE ALLEGED COMMON ANCESTOR OF LATER AUSTRALOPITHECINES AND HOMO

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Claims have been made by Dr. Donald C. Johanson (Cleveland Museum of Natural History) that fossil finds at Laetoli and Hadar in Ethiopia reveal that 3.5 million years ago, a creature named as *Australopithecus afarensis* existed which had sufficient features to prove that it was the common ancestor of *A. africanus* and early *Homo*.[1,2] (Although I do not believe the time scales quoted, I shall not argue against them in this article). Johanson's position in 1976 was that true *Homo* fossils were found in the same stratum as *Australopithecus* thus proving that "early" man and *Australopithecus* coexisted at 3.5 m. years BP. (This is still the viewpoint of Mary and Richard Leakey, who insist that no Homo evolved from any Australopithecine).[3,4] However, by 1979, Dr. Johanson had second thoughts and believed that the Hadar and Laetoli fossils were not separate species but instead belonged to a single species, the long-missing ape-man, *Australopithecus afarensis.*

Let us now examine the position in regard to Johanson's change of mind; and also his continuing clash with the Leakeys.

Firstly we shall examine the evidence presented in Johanson's first assessment in 1976, 19 months after the Hadar finds.[1]

On page 801, Johanson is quite clear on one thing. The 40% of a full hominid skeleton known as Lucy is "without doubt" that of an Australopithecine. On page 801a he writes that "Lucy is far from the genus *Homo*" and that the mandible is V-shaped like that of *Australopithecus* and not parabolic as in modern man. In the article Johanson constantly stresses the stark contrasts

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