HOMO SAPIENS ASCORBICUS, A BIOCHEMICALLY CORRECTED ROBUST HUMAN MUTANT.

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ABSTRACT

Homo sapiens' gene pool contains a defective gene for the synthesis of the active enzyme protein, L-gulonolactone oxidase (GLO). The absence of GLO in the human liver blocks the normal mammalian conversion of blood sugar into ascorbate, leading to the potentially-fatal "inborn error of carbohydrate metabolism", the genetic disease, Hypoascorbemia (in the older nomenclature - scurvy). To survive, humans need exogenous sources of daily ascorbate. Most mammals have the intact gene for GLO synthesis and produce generous daily amounts of the liver metabolite, ascorbate; for instance, an unstressed 70 Kg goat is capable of producing over 13 grams of ascorbate daily and much more under stress. The recommended dietary allowance of 45 milligrams of ascorbate a day for human adults, now proposed and used by nutritionists, is grossly inadequate to restore Homo sapiens to a normal mammalian ascorbate physiology. To correct fully this human genetic defect and banish epidemic chronic subclinical scurvy requires daily intakes of ascorbate equivalent to, at least, the amounts synthesized by the other mammals. Humans kept on a long term regime of full correction of this birth defect show great salutary benefits in health maintenance, disease therapy and slowing of the aging process. This can be regarded as the creation of a new and more robust, longer-living, tough human sub-species, Homo sapiens ascorbicus, by the biochemical reversal of a primate mutation occurring some 60 million years ago. Some of the practical benefits and pathways of future clinical research are discussed.

Key Words: Ascorbic Acid; Ascorbic Acid Deficiency; Scurvy; Genetics, Human; Genetics, Biochemical; Anthropology, Physical.

THE PROBLEM

The gene pool of Homo sapiens contains several defective genes not present in other mammals, which produce enzymic dysfunctions. Probably the most important of these defective genes, which severely limits our health and life-span capabilities, is the gene controlling the synthesis of the liver-enzyme protein, L-gulonolactone oxidase (GLO). This enzyme is the final member of a four-enzyme biochemical system, whose function is to convert blood glucose into ascorbate in the liver. While the daily stress-related production of large amounts (many grams) of ascorbate is common to most mammals, the daily production of ascorbate is zero in the members of the present day Primate Suborder, Anthropoidea, because of this missing enzyme. These members include the higher monkeys, the apes and Homo sapiens (1). The more primitive monkeys, members of the other Primate Suborder, Prosimii, carry the intact gene for GLO and like other mammals are capable of producing their own daily ascorbate.
THE HUMAN BIRTH DEFECT FOR GLO

Unless exogenous ascorbate is available continuously in sufficient amounts to the members of the Suborder, Anthropoidea, their survival will be limited to no more than several months, depending upon incident stresses. Ascorbate, the reducing member of the oxidation-reduction system, ascorbate-dehydroascorbate, is a small simple molecule that appears basic to the living process. It has so many functions in modern living plants and animals that we have only skirted the fringes of this knowledge. The list of ascorbate functions is constantly growing.

It is likely that this molecule was intimately associated with the early development of life on this planet. Its probable main function in these early life forms was to maintain optimal low redox potential (rH) in the living protoplasm and to protect it from the increasing oxygen toxicity due to photosynthesis. As the primitive life forms became more complex, their daily requirements for ascorbate increased. The natural history of ascorbate in the over 400 million years of vertebrate evolution is a fascinating story, and one showing increasing requirements and production of ascorbate in the sequential evolution of the amphibians, reptiles, birds, mammals and primates (1).

A mutation on the gene for synthesizing the liver-enzyme protein, GLO, appears to have occurred some 60 million years ago in ancestors of the present-day members of the Suborder, Anthropoidea, destroying their ability to produce their own ascorbate. This makes Homo sapiens a mammalian mutant (2).

CHRONIC ASCORBATE SCARCITY IN HOMO SAPIENS

This birth defect appears to be present in all members of Homo sapiens, and deprives humans of an important mammalian protective biochemical mechanism against stress (3). A feedback mechanism, increasing the liver production of ascorbate under stress (4), has been serving the mammals as an antistressor for the past 165 million years.

In more recent times, both in human prehistory and in historical times, this defective gene has probably been responsible, either directly or indirectly, for more deaths and disease, more human misery and a shortened life span than any other single cause. If it were not for the severe limitations on population growth imposed by this human defective gene, it is likely that our current overpopulation problems would have overwhelmed us centuries ago.

Before the 1930's it was impossible to correct fully this universal human birth defect, because the only source of the then unknown "antiscorbutic factor" was our foodstuffs, where it only existed in minute trace quantities. To get enough through our foods, comparable to the amounts normally produced each day by other mammals, would require the ingestion of such large quantities of foodstuffs as to be far beyond the handling capacity of our digestive system. Boiled potatoes, for instance, which were depended upon for centuries to keep populations of entire countries free of the terminal symptoms of scurvy, require 44 pounds to get one level teaspoonsful, 3 grams, of ascorbic acid (5). All we can expect from eating even the best of antiscorbutic foods, is merely preventing the appearance of the classical terminal signs of frank clinical scurvy, while leaving chronic subclinical
scurvy, the CSS Syndrome, untouched and rampant (6), especially when it is realized that a 150 pound unstressed goat is capable of the daily production of over 13 grams of the liver metabolite, ascorbate (7).

**BIOCHEMICAL REVERSAL OF THIS HUMAN BIRTH DEFECT**

It was only some 45 years ago, when our chemical technology caught up and advanced to a point where ascorbate could be synthesized and produced rather cheaply, that we were in a position to do anything about fully correcting this 60 million year old chronic subclinical scurvy problem. Except for the past decade and the work of a few dedicated individuals, these 45 years have passed without much assistance or encouragement from the medical or nutrition establishment to utilize fully our accumulating knowledge about the genetics of scurvy (8), daily ascorbate requirements, and their medical and health implications (9). One of the first steps required to modernize the thinking about "scurvy", is to get away from the old misleading tenets of the "Vitamin C-Dietary Deficiency Disease" hypothesis as its underlying etiology and put it in its proper place as a genetic liver-enzyme disease. It is also necessary to discard the term, "vitamin C", for the mammalian liver metabolite, ascorbate. The current daily Recommended Dietary Allowances for ascorbate also require drastic upward revision.

The technology is now available for those who are interested in fully biochemically correcting this human "inborn error of carbohydrate metabolism", Hypoascorbemia.

Full correction may be approached by two pathways:

1. The genetic approach, in which the gene is repaired or replaced so it will be capable of directing the synthesis of the active enzyme GLO. This would be a convenient solution to the problem, but the present "state of the art" in genetic engineering is not capable of doing this. Perhaps another 50 years will see this accomplished. Humans would then be able to perform the endogenous synthesis of ascorbate, like the other mammals. If enough is synthesized in response to stress it will reduce the threat of the CSS Syndrome, but may not insure complete freedom from it. Many mammals with the intact gene for GLO and capable of liver synthesis of ascorbate, still suffer from the CSS Syndrome during their lives (10,11). They favorably respond to additional exogenous ascorbate.

2. The pragmatic approach, which has been available for over four decades and successfully practiced by many individuals. This is simply to ingest daily spaced doses of ascorbate in the range normally synthesized by the mammals, and increase the intake in response to stress. A problem here is our imprecise knowledge of the amounts of ascorbate synthesized by the mammals or the amounts required by Homo under particular stresses. This is not too serious a problem as more clinical tests could rapidly fill in our gaps of knowledge. Also the virtual lack of any toxicity of ascorbate would make "overdosage" a relatively safe procedure.

**HOMO SAPIENS ASCORBICUS**

The long term, daily full correction of the ancient primate mutation resulting in Homo sapiens' genetic defect for GLO, by the spaced ingestion of the required levels of ascorbate would have so many salutary effects (3, 12, 13) on so many different human physiological and pathological
processes that it would be tantamount to creating a new human sub-species, a sort of biochemical "Superman", who would be more robust, tougher and more resistant to diseases and stresses and have a much longer life span. The effects of the biochemical reversal of this mutation have been reviewed from the scant data available up to 1971 (3).

To this biochemically induced "sub-species" I have given the name, "Homo sapiens ascorbicus" to distinguish them from the rest of the scorbutic population of Homo sapiens, in which this genetic defect for GLO is still in full bloom (5).

This full correction should start with the conception of the individual, which necessitates the involvement of the mother. The mother should be on the regime suggested by Klenner many years ago and recently published (14). This comprises the ingestion of about 5 to 15 grams of ascorbate daily throughout pregnancy and lactation and the administration, after weaning, to the neonate each day of ascorbate reaching 1 gram by the end of the first year. During childhood the daily intake of ascorbate is increased to 1 gram per day per year of age until age 10 and then at least 10 grams per day thereafter. Under this regime the fetus develops in the normal uterine environment of ascorbate abundance and avoids the present common stressful birth after 9 months of intrauterine subclinical scurvy. Over the years, Klenner has managed many hundreds of successful pregnancies, with easy labor, in which the present pitfalls of maternal hemorrhaging and infant respiratory distress have been avoided. He also delivered the Fultz quadruplets, the first quads to survive in the southeastern U. S. The distinct and easily noticeable result of this procedure has been the good health and robustness of the neonate. If any unexpected stress does occur, the level of ascorbate intake is adjusted accordingly.

SUDDEN INFANT DEATH SYNDROME (SIDS)

An early fatal hazard and the most common cause of death of the Homo sapiens infant in the Western World is the sudden infant death syndrome (15), also known as, "Crib Deaths", or in Australia as, "Cot Deaths". SIDS usually afflicts infants under 1 year and generally before 3 months of age. The apparently healthy, symptom-free baby is put to sleep in the normal manner, and is found dead when next observed. The mortality is about 17 per thousand in Australia. In the U. S. A., the estimate is 8,000 to 10,000 infants dying per year, which is probably low.

After long and extensive work in his hospital in the Australian outback, Kalokerinos (16) and Dettman (17) have summarized and confirmed the conclusion reached many years earlier by Klenner (14) that SIDS is a fatal manifestation of infantile scurvy, which can be simply and harmlessly prevented with ascorbate.

The medical research establishment in both Australia and the U. S. A. has consistently ignored the findings of these three investigators, their simple preventive measures and the rapid urine tests they suggested for detecting potential SIDS victims, and have not allotted any money from the ample research funds available for SIDS to test these findings and conclusions. By their apathy and hostility to these ideas, they may have permitted this annual slaughter of babies to continue unchecked. From the scant available evidence, Homo sapiens ascorbicus infants do not appear to succumb to SIDS.
SUDEN ADULT DEATH SYNDROME (SADS)

Sudden and unexpected death has been known for centuries (and as often forgotten) as the characteristic final pathognomonic symptom of scurvy. Dr. Linz in his 1753 book on scurvy cites many examples of scorbutic sailors suddenly and unexpectedly dropping down dead in the midst of whatever they were doing. He stated (18), "At sea, where no greens, fresh meats or fruits are to be had, the prognostics in this disease are sometimes deceitful; for people that appear to be but slightly scorbutic, are apt to be suddenly and unexpectedly seized with some of its worst symptoms. Their dropping down dead upon an exertion of their strength, or changes of air, is not easily foretold; though it generally happens after a tedious confinement in a foul air."

"Foul air" in Linz's day meant the air in the unventilated living quarters in the ship's hold. Our "foul air" is the highly polluted atmosphere that we have to breathe all the time and cannot escape by going "topside".

A recent paper (19) explores SADS as the basic cause of the millions of "sudden deaths" occurring each year throughout the world, blaming it on the cumulative life-long insults and the long term biochemical stresses due to the chronic inadequate correction of the human, "inborn error of carbohydrate metabolism", hypoascorbemia (8). This chronic subclinical scurvy could promote the genesis and permit the long term unchecked progression of the diseases that are now reported as the "cause of death"; the coronaries, the cancer, the strokes, the infections, the "Mystery Deaths" and other conditions that now annually terminate the lives of millions. Would the early conversion of the Homo sapiens individual to Homo sapiens ascorbicus reduce this mortality and permit the members of this sub-species to avoid this physiological abuse throughout their lives and live in full health to their increased, statistically-unknown, allotted life span (20)?

THE VIRAL DISEASES

Among the many useful properties of ascorbate is its strong, non-specific ability to detoxify viruses, when used in the proper manner. Medicine with its almost sole preoccupation with its frustrating use of highly specific vaccines in the prevention of the viral diseases, has almost completely ignored ascorbate's important therapeutic contributions, for the past 30 years. Many doctors appear not to realize that they are putting these stressful foreign proteinaceous vaccines into the blood streams of a population of Homo sapiens suffering severely from the CSS Syndrome. In chronic subclinical scurvy, the individual's immune system is operating poorly and the introduction of this foreign protein can do more harm than good (21), and may even cause deaths as recorded in the recent Swine Flu flasco. In Homo sapiens ascorbicus there is no lack of ascorbate to keep the immune system at optimum performance, and the immune response will not only be improved and less dangerous, but the individual may not pick up the disease in the first place. A classic pathognomonic symptom of scurvy (and the CSS Syndrome) is lack of resistance to infection.

There is a large volume of medical literature on ascorbate's ability to detoxify and treat the viral diseases, which was partially reviewed in 1972 (22).

From a more practical standpoint, Klenner (23) has been successfully using massive doses of ascorbate for over 30 years in the therapy of a wide spectrum of viral diseases and other conditions (24), in some cases giving as much as as much as 300 grams of sodium ascorbate a day, intravenously.
Klenner's work has been confirmed by Cathcart (25), who in the past 6 years has treated over 6,000 viral disease cases, usually getting rid of the symptoms in about 3 days, which he calls, "symptomectomy". Medicine has paid little attention to these eminently successful clinical results, rejecting them as "anecdotal", but how long can thousands of "anecdotal" successes be ignored?

As far back as 1933, tests on scorbutic guinea pigs showed degenerative liver changes and sporadic later work confirmed this and ascorbate's ability to prevent and successfully treat hepatitis and help Homo sapiens recover from the liver damage (26). In spite of all these reports and the wide availability of inexpensive ascorbate, this simple and harmless procedure has been ignored by most doctors and hepatitis has remained a major and lethal problem for the past 40 years. The latest work of Morishige and Murata (27) shows how easily viral hepatitis B can be prevented by ascorbate in transfused hospital patients. It seems that hepatitis would not be a problem for Homo sapiens ascorbicus, and because of ascorbate's non-specificity other viral diseases also. The complete control of the viral diseases appears to be well within our grasp.

This section on the viral diseases cannot be closed without a comment on the oft-quoted poor showing of ascorbate's antiviral effects in the large scale double-blind studies on the the common cold (12, 22). The investigators designing the protocols of these tests were unaware of the size of the dosages of ascorbate required for effective prophylaxis and therapeusis and the importance of properly timing these doses. They also left out completely the "abortive dose" phase of the procedure, apparently because it was too difficult to do in a large scale double-blind study. It was simply a matter of "too little and too late", because in previous unpublished "anecdotal" tests it was at least 95% effective (28).

CANCER

The use of ascorbate in the treatment of cancer and leukemia goes back many years to the 1930's when ascorbate first became commercially available. This early work is confusing because these early investigators had no idea of the size of effective dosages required and were using it as a "vitamin" at trace levels. As soon as higher amounts were employed, the clinical results improved (29).

In cancer and leukemia, as in other serious diseases, humans suffer from the cumulative stresses of a second disease, chronic subclinical scurvy, which is a major contributor to the lethality of the first disease. It has been proposed that this chronic genetic condition be fully corrected with the required dosages of ascorbate before and during the treatment of the first disease (30), in order to aid the effectiveness of the treatment and insure survival of the patient.

The rationale for the use of ascorbate in cancer has been discussed (31) and the major contribution of the CSS Syndrome to the lethality of cancer (32) and Leukemia (33) further explored. Ewan Cameron and Linus Pauling for many years have been investigating the theoretical and clinical effects of ascorbate in cancer therapy and have published widely. Brief summaries have appeared in 1977 (34, 35). A comprehensive review of the use of ascorbate in cancer by Cameron, Pauling and Liebovitz is scheduled for publication in 1979 (36).

From the results obtained up to this point on the use of ascorbate in
cancer prophylaxis and therapy, it would appear that members of Homo sapiens ascorbicus would have much less to worry about cancer, have a significantly lower risk of contracting the disease, and if contracted the customary therapy would be much less lethal.

HEART AND VASCULAR DISEASES

This section on heart disease will be brief with a few review references (37, 38, 39). This is not being done because of a paucity of evidence that ascorbate is essential in the prevention and treatment of heart disease, vascular pathology and strokes. To the contrary, the evidence indicates that the proper use of ascorbate may be the long-ignored "missing link" in the prophylaxis and current therapy of these diseases, and would be a life saving measure.

This would be especially true in the recently developed, highly organized special emergency units for the rapid response and stressful collection and transportation of these helpless victims to the highly stressful environment of the hospital's emergency care unit. These scorbutic victims are usually given no ascorbate to combat these stresses nor is any attempt made to fully correct their life-long genetic defect for GLO.

It has been pointed out (37) that, "In intensive care units for coronaries, ascorbic acid is conspicuous by its absence." Each emergency unit responding to these calls should be equipped to start dripping into the patient a parenteral solution containing 30 grams per liter of Ascorbic Acid Injectable, U.S.P., to at least assure that the patient arrives at the hospital alive (19).

It is likely that the biochemical conversion to Homo sapiens ascorbicus, as early in the life of the individual as possible, would eliminate the scorbutic genesis and pathology leading to these diseases, and be a major factor in reducing the incidence and mortality of these killer diseases.

DRUG ADDICTION

New clinical data obtained since 1976 indicates that Homo sapiens ascorbicus would not be troubled with addiction to heroin and other abused addictive substances. Addiction is now a major health, economic and police problem, affecting millions and touching the lives of many innocent victims.

In Homo sapiens treated with sodium ascorbate, the non-specific detoxication of the addicting substance is so fast, no "high" is produced on the injection of a pharmacologically active dose of heroin (40). This simple procedure is now being used with remarkable success in the rapid treatment and detoxification of addiction. These clinical results have been checked (41, 42). This non-toxic technique is the only one that restores the addict to good health in about a week and will eventually supplant all present drug addiction treatment procedures involving the use of narcotics or other harmful drugs. Under the influence of sodium ascorbate, the addict can be taken off the addictive drug completely, or in the drug culture slang, "go cold turkey", without showing any withdrawal symptoms. After about a week on the sodium ascorbate the ex-addicts are different persons both physically and mentally.

Actually what we are doing with our treatment (40) is rapidly converting sick, scorbutic Homo sapiens individuals into robust, healthy members of Homo sapiens ascorbicus!!
AGING AND THE LIFE SPAN OF HOMO SAPIENS ASCORBICUS

The reduction in the incidence, morbidity and mortality of various present serious medical problems, by the full early and continued correction of the human genetic defect for GLO, will have a profound salutary effect on the healthy life span of these corrected individuals. The extent of this effect is not yet known due to the lack of statistical mortality data for a population of this human sub-species, but educated guesses indicate it may be substantial (43).

As early as 1947 it was pointed out by McCormick (44) that the continuous significant drop in the incidence, morbidity and mortality in a wide spectrum of diseases over the past century was due to, "some unidentified major prophylactic factor .... operating in bringing about such a uniformity in reduced mortality from so many infectious diseases in the same period of time .... The author believes that the major factor in the protective influence .... has been the greatly increased intake of vitamin C. The major change in nutrition in this period has been brought about by the tremendous increase in the production, distribution and consumption of citrus and other vitamin C fruits, made possible by the gradual development of better transportation throughout the century- steamships, railways and motor highways."

In discussing the possible life extending qualities of ascorbate, the question always arises as to why with their apparently high liver production of ascorbate, the mammals have such short lives. The answer to this is that these mammals are dependent upon a sensitive biological enzyme system, which may not respond to heavy stresses with sufficient ascorbate production, fast enough. Also the efficiency of the endogenous enzyme system may deteriorate with age. In our work in veterinary medicine (10), we found that dogs and cats suffer from chronic subclinical scurvy during most of their lives and benefit from ascorbate supplementation. In the larger breeds of dogs, hip dysplasia, long regarded as a genetic defect, is merely due to a chronic insufficiency of ascorbate (11).

Unlike the other mammals, Homo sapiens ascorbicus is not dependent or limited by an endogenous sensitive enzyme system. A supply of ascorbate can always be available, it is only necessary to know when to reach for the bottle and know how much to take.

CONCLUSION

The simple biochemical reversal of the human genetic defect for GLO, resulting in the conversion of Homo sapiens into Homo sapiens ascorbicus, with a return to a more normal mammalian physiology of ascorbate abundance, provides the basis for humans to live happier, healthier and longer lives. It will take a long time to collect sufficient statistics to obtain a reliable estimate of the increased life span and extent of disease resistance due to the elimination of the current epidemic CSS Syndrome. This can be regarded as an inexpensive form of biochemical life and health insurance. We look forward to the cooperation of Medicine, Health and other scientific disciplines in gathering this needed data.
REFERENCES


43. Stone I. Reference 3, Chapter 18, Aging.