The Answer to Crib Death "Sudden Infant Death Syndrome" (SIDS)

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Abstract

(1) Two doctors on opposite sides of the globe eliminated crib death among their patient populations for 40 years using ascorbate supplementation. Unknown to each other they arrived at the same regimen.

(2) Crib deaths nearly disappeared in Japan in 1975 when first inoculations were postponed until the 24th month of life.

These findings and their explanation are explored. SID is traced to a nonspecific or general adaptation stress syndrome defined by Hans Selye. It is precipitated by a deficiency of ascorbate and also of vitamin B6 and zinc.

1. SID

Since appropriate ascorbate supplementation eliminates crib death, it must not result from a "syndrome" of factors but from a vitamin deficiency. The label for sudden infant death is no longer SIDS but SID—without the final S. And the plural, SIDs, means sudden infant deaths.

1. Contrary to the official line, there is a preventive for crib death and has been for more than 40 years. It costs only pennies a day. Two pioneering doctors on opposite sides of the globe—unknown to each other—arrived at the same formula for eradicating among their patients' families this tragedy, which young mothers tell me is their worst fear.

1.1 Archie Kalokerinos virtually eliminated infant mortality among his Aboriginal and white patients in "outback" New South Wales, Australia since 1967. That's the year he began to use ascorbate, the liver metabolite mislabeled vitamin C. A family physician with distinguished education and training, he retired in September 1992 to write a book on SID. Those dark-skinned people scratch out a living in abject poverty in a harsh environment,

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subsisting largely on powdered milk, jam and white bread. When he arrived, infant mortality was close to *50 percent*—whence the title of his book, *Every Second Child}* A large proportion were SIDs: unexpected, and unexplained after autopsy. Aborigines treated by doctors using antibiotics and other medicines continued to "die like flies".[2]

Dr. Kalokerinos got the idea of using ascorbate earlier in his career when he saw a child, dying of scurvy, dramatically recover after an ascorbate injection. He urged 100 milligrams of ascorbic acid daily per month of age to 10 months, always in divided quantities: 100 mg/ day the first month, 200/day the second month and so on. Then 1 gram daily for each year of age to 10 years and 10 grams daily for the rest of life. A sick child was given sodium ascorbate by intravenous injection, the quantity adjusted according to the severity of illness. Some sick children were given oral "bowel tolerance" doses of ascorbic acid.[3]

If doctors objected that so much oral ascorbate might cause diarrhea, he reminded them that is less serious than death; and the quantity can be shaded. Mammals other than primates and guinea pigs normally generate 3-18 grams a day per 150 pounds body weight, and much more when stressed.[2] So an unstressed 8-lb. animal might generate 160 to almost 1,000 mg/day. If some remarked that the RDA of ascorbate for 150-lb. *adults* is only 60 mg/ day, he informed them that native Fijians, untouched by Western dietary influences, ingested 1,000 to 8,000 mg of vitamin C a day in their food.[4] And guinea pigs fed a Western diet containing 60 mg/day of ascorbate (adjusted for body weight) rapidly developed arterial damage that leads in people to heart attacks.[5,6]

For families who couldn't afford the modest cost, he provided the ascorbate purchased in bulk and a little **zinc** and other nutrients for each mother to add to her baby's food daily. Instinctual body language told baby the slight sour taste of ascorbic acid crystals mixed into the food is good.

He made unannounced home visits to spot check the baby's urine (G. Dettman personal communication 1992). Many people assured him, "Yes, I'm giving the vitamin"; but urine tests showed they weren't and so were risking the child's life. Not all commercial tests are accurate; the Merck ascorbate test, designed to measure levels in food, gives a false positive in the presence of uric acid. In one study that test showed very high vitamin C in the urine of babies who had died SID deaths, but the accurate AMEX C-STIX test showed zero ascorbate.[7] Levels of all other micronutrients, as well, are zero or close to zero in SID babies, as they are in people dead of a heart attack or Alzheimer's disease.[7]

"Because the urine reflects the condition of the blood and varies within large limits in order to help the blood maintain homeostasis [internal stability], the urine is a prognostic indicator of what the blood is doing to maintain homeostasis. It keeps what it needs and tosses off into the urine what it doesn't want. Vitamin C is one of the threshold substances; the blood should have enough to toss a certain amount into the urine".[8] This vitamin in the urine doesn't at all indicate that it is being "wasted," as misinformed (or malmotivated) nutritionists and doctors want us to believe. If a specific concentration of ascorbate doesn't appear in urine, the baby isn't getting enough. Dr. Kalokerinos and his partner Glen Dettman, a distinguished biomedical scientist with expertise in microbiology—who concurs with my analysis—did their study without research funding or publicity, constantly harassed and opposed by prevailing medicine, their manuscripts rejected by mainline medical journals.[7] I saw them present their message and data on American television in 1976, and they addressed a radio audience estimated at 80 million in 1982 after publication of Kalokerinos' book. But they couldn't explain his results well enough to convince audiences; only 10,000 copies of the inexpensive book were sold.

1.2 Confirmation is critical. Frederick R. Klenner of Reidsville, North Carolina, also virtually banished infant mortality after hundreds of births supervised from the late 1940s through the 1970s. Each mother took 5-15 grams of ascorbic acid daily in divided quantities throughout life, including pregnancy and lactation; Klenner required breast feeding. The condition of mother and baby was always so incomparably better than usual that "Failure to use this modality in all pregnancies borders on malpractice." And the supplement was certain at least greatly to reduce the number and seriousness of birth defects (see below).[9,10] Klenner's ascorbate schedule for the baby was the same as Kalokerinos'. (For sick children— and adults as well—he too injected sodium ascorbate.)

Breast feeding started immediately after delivery[10]—no preliminary wiping and washing—so that the newborn benefited from the initial flow of colostrum, as well as bonding to the mother. *Hydrogen peroxide* is present in large quantities in all mothers' milk (what a great recommendation!), particularly if ascorbate is supplemented, and especially in the colostrum. Disease organisms *including HIV,* and cancerous tumor cells are anaerobic. So they cannot survive in blood, circulating *or stored,* in the presence of highly active free oxygen atoms that split off from H_20_2 or ozone (0_3) molecules.[1112] As has been known for 40 years, the immune-stimulant ascorbate destroys disease organisms by forming H_20_2.[13] Mothers' milk is also rich in *acidophilus, inositol, immune factors,* a powerful natural *antibiotic*[14] and protective *mucin.*[15] Because babies are born very low or lacking in antibodies to infectious diseases, all these aid the new person's health, as well as the mother's, throughout life—advantages not shared by bottle-fed babies. And as we shall see, formula feeding continues to widen the disadvantage. Further, because the breast-fed baby uses up less ascorbate to destroy disease organisms, more remains to serve the vitamin's many other functions and thus avoid scurvy. These facts partly explain the lower incidence of SID among breast-fed than among bottle-fed babies (see also 3.8). To gain all these advantages the mother, it is said, need only consume about 500 extra calories a day, supplemented by ascorbate, *zinc* and ideally, also *vitamin B6, folate* and at least *magnesium.*

1.3 Now for a bit of perspective: In 1753, James Lind showed how to prevent and cure clinical scurvy with citrus fruits, which contain vitamin C. But 100,000 British Navy sailors dropped dead of scurvy even while working—sudden death!—until all the old

"experts" had died and were replaced by new-thinkers, who chose to make the navy "Limeys." And the British merchant marine waited *118years* before falling into line. New ideas are typically ignored even though proven correct, until one or two old generations of authorities are gone.

Despite appeals publicized all over the world for more than 20 years, no baby given Klenner's/Kalokerinos' ascorbate therapy in the recommended quantities is known to have died.[7] How many thousand babies have to suddenly and tragically expire before the popular media, the medical profession *and the SID "support" groups* stop stonewalling?

What the *Economist* of London called "the SIDS establishment" tries to spare parents of SID victims the agony of blaming themselves for a child's death. "They have made a point of believing that SIDS has no identifiable cause, and therefore no identifiable solution." They reject the "near-SIDS" idea, which implies that a parent on the spot can stop a cot death.[16]

"But both pediatricians and parents quite often see infants who have stopped breathing and have even begun to turn blue. With prompt action they recover [this statement is questioned below]; left on their own they would probably die",[16] and monitors of various kinds are used to warn parents of an impending crisis[17] (cf. 3.6). Some SIDs have in fact been observed.[18] Besides being blue, the babies had cold hands and feet and appeared to be breathing with difficulty. In one case, the observer was unable to save the baby despite CPR, shaking and massive antibiotics.[19]

Warren Guntheroth, a Seattle authority on SID, feels that "encouraging the notion that SIDS is unpreventable and random may in fact make it more difficult for bereaved parents to cope".[16]

As for the medical .establishment: the National Institutes of Health's new program of studying alternative therapies emphasizes double-blind tests, the "gold standard" of medicine. Those on the test substance get a set amount, others a placebo, and neither doctors nor subjects know—at least until side effects appear—who gets what. But in Klenner's/ Kalokerinos' preventive therapy, the quantity is varied according to biochemical individuality and the mother has to know what she is giving the baby. So a double-blind test is hardly practical.

Will N.I.H. use that fact as the pretext for continuing to ignore this proven life-saver? Or will the standard approach be used: "first, discredit the treatment by citing the absence of controlled studies; next, get a panel of experts to argue against conducting such studies; and finally, threaten to destroy anyone who becomes involved".[20] In either case, will the almighty dollar continue to prevail at the expense of helpless babies' lives?

2. What Causes SID?

2.1 A review of the flood of research on crib death published from 1970 to 1981 [21], carrying 142 references, found "no evidence" of vitamin C deficiency in SID babies. But they looked at the RDA (recommended dietary allowance) and ignored the evidence! Most findings cited by Dapena and the referenced articles and books fit perfectly the hypothesis of deficient C or B_6, or both. *Doubtless, most of these babies had been "immunized"* (see 3.6).

Inflammation was often noted in various parts of the upper respiratory tract including the epiglottis, larynx, trachea or bronchi. In an important study in Seattle, "inflammation of the pharynx" was evident in over 90% of crib death autopsies.[22] Some studies found bronchioles thickened by inflammatory infiltrate, of bronchitis rather than viral origin. Others found minor inflammatory processes, which appeared to be responses to a slight viral infection, not severe enough themselves to cause death (see also next section). Minimal evidence of pneumonia was found in one study, clusters of macrophages in alveoli in another. Excess secretion of mucus in the larynx, found in some SID babies, could result from vitamin C deficiency, as can inflammation.[23] All those reported symptoms, it would appear, could have been prevented by adequate ascorbate supplements.

A retrospective study found a statistically significantly greater frequency of acute funisitis, lymphocytic infiltration of decidua, and pigment-laden macrophages in fetal membranes of SID babies than others. All these are associated with preterm deliveries related to amniotic fluid bacterial infections—a common cause of premature delivery. Because such infections can be prevented by enough

ascorbate, it would seem that it could prevent at least some of that type of premature birth. SID incidence is higher among premature than full-term babies (see below).

In one study, significant viral agents were found in 37.5% of SID babies vs. 16.2% of controls. In 41% of another set, at least one viral agent was found from at least one site. In an epidemiological study covering 19 years, influenza A (but no other virus) was found in a statistically significantly greater proportion of SID than other infant deaths. A study of 200 SID babies found infection and absence of thymic reaction to it, suggesting an abnormality of immunologic response. Adequate ascorbate should strengthen the immune system enough to prevent all that. Minute hemorrhages, often found in SID autopsies, can result from ascorbate deficiency. The heart typically shows no lesions.[21]

All these pathological findings match Hans Selye's *nonspecific (or general adaptation) stress syndrome*, which includes enlarged adrenal cortex, intense atrophy of the thymus, the spleen and all lymphatic structures, signs of internal bleeding into the lung, thymus and pericardium, deep ulcers into the lining of the stomach and duodenum, disappearance of eosinophil cells from circulating blood, a number of chemical alterations in the constitution of the body fluids and tissues, and changes in the viscosity and clotting properties of blood.[24] This complex situation, incompatible with life, appears to have resulted from deficiency of ascorbate, B6, zinc and cofactors.

2.2 The apnea hypothesis. "Current medical thought holds that an immature part of the infant's brain fails to recognize the need to breathe and therefore, stops that function".[25] Numerographic studies in Australian hospitals of babies who subsequently died SIDs revealed no apnea;[7] but that small sample suggests only that apnea may not be involved in all cases.

One investigator remarked that minor respiratory illness may trigger sudden apnea. Mucosal swelling in nasal passages, which could result from inflammation, occluded those airways, and "infants of this age simply will not breathe through their mouths. In that crisis... they simply suffocate".[26] Another research team demonstrated, in a large group of living babies of appropriate age, that infants do become apneic in response to mechanical nasal occlusion.[27] Another suggested that obstructive apnea is more life-threatening to babies than apneas of central origin.[28]

In many SID babies examined, an observer found evidence of repetitive apneic episodes leading to hypoxia during their short lives. Hypoxia, often combined with minimal viral infection, could occur before, during and after birth. But ascorbate deficiency could easily explain hypoxia, except during birth, as well as the inflammation that can cause it. Enough •ascorbate should generate H202 to prevent hypoxia. Excess secretion of mucus in the larynx, found in some SID babies, could also result from ascorbate deficiency.

But all this does not at all prove that babies die *because* they stop breathing. Rather, the cessation of breathing appears to occur because the nonspecific stress reaction, again, creates a condition incompatible with life.[29] In an anecdote reported by a mother with an M.D. degree, the heart stopped beating; she attributed death to a cardiac problem (3.2). The pupils of the baby, who had been breastfed, still reacted well to light.[19]

2.3 The role of iron in crib death has been almost universally ignored. Some have found excess iron in SID autopsies. Ferritin is a strong oxidant; an excess promotes oxidation reactions, at least when activated by deficiency of selenium;[30] and it destroys vitamins C and E, needed to prevent those reactions. Iron weakens immune function (and cancer cells appear to thrive in an iron-rich environment).[31„35] Excess iron could increase oxysterols and the resultant arterial damage,[36] clotting[37] and risk of coronary occlusion (see Addendum 1.)

The following information and references were kindly brought to my attention by Hans Raible of Stuttgart. Iron withholding is one of our natural immunity mechanisms, apparently developed to compensate for our inability to make our own ascorbate. Bacteria, as well as cancer cells, thrive on iron, and a natural defense would be vitamin C. Since we do not have that, we manage on low iron during suckling which protects us from infection. Both Cu and Fe are important for the human baby, and there is a very dynamic sequence of changes in the first months of life. These

appear to be unique to humans, guinea pigs and other animals that do not make their own ascorbate, as Linus Pauling suggested earlier. A mother loses a great deal of zinc in the placenta—which the mother in all other species eats immediately after giving birth. Zinc supplements are advisable so that the mother's milk will not be zinc deficient.[38] This is particularly critical for boys, who require five times more zinc than girls, to adequately supply their testicles. (Very zinc-deficient mothers even give birth to all-girl families.) Deficiency of zinc in mothers could be one reason for boys' greater SID mortality than girls', and particularly among bottle fed babies, see below.

Before birth the mother loads the fetus with two essential trace elements, copper and iron. Both have to be high at birth since they are stimulants to the brain and central nervous system, and both mother and baby have to be very alert and in top gear at delivery. Iron is pushed into the baby very late in pregnancy, filling its liver to the brim. The baby gets about 0.4-0.5 gram of Fe from the mother; of this it stores 200-370 mg.[39] In an 8-pound newborn, that is about 55-100 mg/kg. (For comparison, an adult body contains 3 to 4 g of Fe, or about 50 mg/kg.) This supply will last through the suckling period; mothers' milk contains only 0.4 mg of Fe per day.

Because this *stored iron destroys vitamin C,* more must be given per kg of body weight than to an adult, particularly in the first two months. Dr. K's suggestions take account of this fact. Mothers' milk contains ascorbate, cows' milk does not, and so a baby develops scurvy if fed cows' milk. And it makes enormous sense to supplement C in goodly amounts in order to supply the baby's *full* needs. If a mother breastfeeds, she must take C so that the breast milk contains enough of it.

Cu is reduced to more normal values immediately after birth by drinking colostrum which contains huge amounts of Zn—unless the mother is deficient in it. The colostrum Zn lowers serum Cu to more normal values, so this problem is dealt with quickly. Formula is very unlikely to accomplish this.

After delivery, the baby starts excreting iron in order to lower it. During its first week of life, healthy suckled full-term babies excrete 10 times more iron than they absorb. Their feces contained 20 times more lactoferrin than feces of babies fed cows' milk. The relatively large quantity of lactoferrin in human milk is considered an important contributor to the much lower incidence of infection in breast-fed infants than in those restricted to cows' milk or milk formula.[39] In this, it is assisted by the suckling mother, who feeds it the iron chelator lactoferrin which acts as an organic disinfectant.

During the first two months, the baby is hyperferremic. By two months, healthy infants have decreased iron saturation level of plasma transferrin from 69% at birth to 34%; and by 6 months, to 25%. Values less than about 30% are helpful in preventing infection. Iron elimination, accomplished more readily in breast than bottle feeding, may also be essential to permit normal myocardial growth.[40]

During months 4-6, the baby starts getting hypoferremic; this protects it against infection, cf. the Masai story (Addendum 2). From month 7, when the baby gets solid food and can take up iron through the gut in normal fashion, it also has adult *ferritin* which limits iron uptake to the body's needs.[39]

A single injection of 10 mg/kg iron (as dextran) into babies in their first month caused within one week a seven-fold increase in septicemias and meningitis caused by *E. Coli* and other gram-negative bacteria.[40] The sera of neonates whose mothers received dextran during pregnancy had impaired bacteriostatic and opsonizing activity. Similarly, parenteral iron increases infection in adult patients.[40]

High concentrations of *E. coli* (cf. 3.4) were found at autopsy in many SID babies and not in healthy babies.[7,22] Organ damage caused by such infections should be detected at autopsy, seeming to exclude this as the immediate cause of SID. But the bacteria hinder nutrient absorption and so worsen tissue ascorbate deficiency, making sudden death more likely by either scurvy or atherosclerosis, or both (see below). Ascorbate deficiency also promotes excessive clotting.

Giving supplemental iron during lactation, or formula "with iron," can precipitate one avoidable form of SID. Suppose a baby contracts *botulism (Clostridium botulinum).* If it lives on its mother's milk alone botulism develops slowly, the baby can be brought to the hospital and treated successfully. If the baby gets formula "with iron," botulism strikes

like lightning. In 69 botulism cases, 39 had been breast- and 30 formula-fed. *The ten who died were all formula babies getting added iron.*[40]

2.4 Some try to find the cause of crib death in sleeping position. Yet babies suddenly die sleeping in a crib or in their mother's bed, in prone, supine or any other position,[41] even in a parent's arms.[42] Some may, by inference, die awake.[43] Anaphylaxis from inhaling cows' milk to which a child has become sensitized has also been blamed for SIDs;[43] breathing passages close. But that doesn't explain SID in the breast-fed.

3. Two Proposed Explanations

Two proposed explanations for Klenner's and Kalokerinos' success at preventing SID with ascorbate appear at first to be contradictory, but on careful analysis they can be united.

3.1 Klenner was certain, and Kalokerinos and Dettman concur, that SID results from the *subclinical scurvy* described in 1972 by Irwin Stone,[44] caused by an ascorbate deficiency detectable in a laboratory or by testing the baby's urine.[1,7]

3.2 But the highly regarded neurologist/ internist Moses M. Suzman of Johannesburg, South Africa, has a different idea. Over a period of more than 40 years he nearly eliminated heart attacks and other cardiac events among thousands of precardiac adult patients using 100 mg of vitamin B6 a day—in 1972 he added other supplements—without changing diet, smoking or lifestyle, and without complaint of side effects. He also appears to have reversed atherosclerosis in many hundreds of heart patients using more B6, other supplements and an optional semivegetarian diet — -together with conventional medicines and office visits, usually for not over a year.[36-45]

Dr. Suzman is "firmly convinced" that, at least in most cases, a cot death is an extreme of *infantile atherosclerosis.* Doris Jaffe's team of pathologists at the Research Hospital for Sick Children in Toronto found at least traces of intimal thickening in 97 percent of 176 who had died of any cause in their first month.[46] Fifty studies since 1904 confirmed the finding; one study of 1,000 newborns, also by the Jaffe team, found all had at least this intimal thickening [M.M. Suzman pers. comm. 1984, 1991]. Jaffe described two deaths in her cited study as SIDs;[46] Dr. Suzman, in close telephone contact with her, laid those to occlusion of coronary arteries.[46]

A division of the South African Health Department that tracks health of blacks reports *high incidence of cardiovascular deaths among black babies ages 0 to 3 who are fed formula instead of breast-fed* [P. Bruwer personal communication 1993]. How many of these are interpreted as SIDs is not yet reported.

Pathologists McCully, Serfontein and Seattle cardiologist Lester Sauvage questioned any connection of SID to cardiac failure. But even great authorities are often wrong. The key, says Suzman, is to cut arteries lengthwise rather than crosswise, as is customary. One could, he says, cut many cross-sections and miss a delicate occlusion. Dr. Sauvage stresses the small diameter of a newborn's coronary arteries—only one millimeter, less than four one-hundredths of an inch—and the difficulty of cutting lengthwise through its many twists and turns. Yet the word "longitudinal" in the title of the report shows that Jaffe did it and emphasizes her view of its importance.[46]

In May 1993 Dr. Suzman, now 89, found and promised to send me 84 color photomicrographs from another study in Toronto. They show coronary arteries that are occluded (yet free of cholesterol, indicating no inherited familial hypercholes- terolemia) in spontaneously aborted fetuses, newborns, older children and adults. They seem to prove objectively that sudden death was identical in all.

Dr. David Ritchie of New Zealand writes, "It appears that endotoxins (in vaccine) go on to cause a cardio collapse and a total circulation collapse. It's quite a common pathway in children dying of SIDS".[47] Or apnea could result from rather than cause cardiac failure, as I suggest it may in some adult heart attacks.[48]

Does other evidence support Dr. Suzman's theory? One finding seems contrary. Pregnant women were low in arterial-wall damaging,[49,50] oxysterols-generating,[51] cocarcinogenic[52,53] *homocysteine,* an independent risk factor for coronary artery disease—which may, however, require the co-participation of lipoprotein(a).[6] Homocysteine also impedes the action

of macrophages, weakening the immune system.[54] During pregnancy high levels of estrogens promote homocysteine metabolism[55] and deter lipid peroxidation.[56]

Other related evidence supports Dr. Suzman's ideas. Expectant mothers of all economic and social classes, like old people, are typically very to extremely deficient in all nutrients including vitamins C and particularly *B_6 (Pyridoxine)}*[57,58] Most such deficiencies exist in the tissue and muscle cells and erythrocytes[59,60] rather than serum. And expectant mothers, like elderly people,[61] are deficient in B_6 even though taking RDA-strength or stronger vitamin supplements.[61]

Their Pyridoxine deficiency results from not only me- thionine-rich high-animal protein Western diets and the same toxic Western environments that affect us all; high dietary sugar lowers B_6 and many other nutrients. The stress of pregnancy and the demands of the growing fetus further drain the mother of B_6.[62] This deficiency distorts mineral balance and hormone balance as well as fluid balance.[57]

This deficit shows up in such symptoms as edema (John Marion Ellis, a pioneering clinician and researcher in Mount Pleasant, Texas, reported one pregnant woman lost 15 pounds of water from her tissues after she started B_6 !), carpal tunnel syndrome (CTS), dropping glassware, leg cramps, diabetes of pregnancy, nocturnal paralysis of the arms, muscle spasms in legs and feet. None of these conditions is normal and healthy for the mother.[57]

And for the fetus these symptoms are the stark but universally ignored handwriting on the wall foretelling not only risk of heart attack in later life but also of infantile atherosclerosis—which can be inferred to start *in utero.*[62] And, according to Suzman's theory, risk of SID! Ellis rids his pregnant patients (and their fetuses and newborns) of these symptoms using B_6 supplements. The women take 100 to 300 mg daily, gauging each's needs by the severity of her CTS.[57]

Certain nutritionists wrote that pregnant women ingest plenty of B_6; one declared that the RDA of B_6 for babies is too high. But they know nothing of clinical experience with pregnant women, an egregious example of the common "compartmentalization" of research. There is no communication between the compartments.

Further supporting Suzman's theory—critically, even in nor- mal pregnancy there is a decrease in serum pyridoxal phosphate.(PLP), the form of vitamin B_6 that the body can use. This decline is very significant in the third trimester.[55] Also, fetuses and newborns up to the traditional age of weaning are deficient or entirely lacking in cystathionine beta-synthase.[63] That enzyme is required to enable conversion of ingested Pyridoxine into PLP which, as a cofactor, enables degrading of mischievous homocysteine metabolized via the transsulfuration pathway from the methionine in formula based on cows' milk.[54]

3.3 The superior immunity imparted by breast feeding only partly explains why SIDs are commonest in bottle-fed babies. Cows' milk, in addition to its several other disadvantages discussed earlier, contains several times more methionine—the dietary source of homocysteine—than human milk, which includes less than any other animal protein[62] and less, even, than any plant protein that contains methionine [M.M. Suzman interview 1992]. The heat of pasteurizing destroys 86 percent of cows' milk's scanty B_6,[62] as well as all the enzymes required for absorption and immunity.[8]

Certain other conditions found in many SID autopsies, on analysis, support Suzman's explanation. H.D. Foster, after computerized study of U.S. Geological Survey, climatic and other records in which he considered 221 geographical variables, related SID frequency to subnormal iodine and the resultant subclinical hypothyroid condition.[64] That condition depletes intracellular magnesium promoting accumulation of homocysteine,[54] and weakens the immune system.

(Many researchers now think that half[65] or even 90 percent (L. Lee personal communication 1993) of the American population are subclinically hypothyroid, a condition not detected by conventional tests.[66] So through insurance premiums, taxes, inflation and out of pocket, patients spend hundreds of millions of dollars every year treating the multifarious symptoms (many of which may have other sources). This is profitable for all concerned except the patient—but never succeeds, as the unsuspected cause is not attacked.)

The correlation of SID rate with goiter incidence in World War I veterans ($r=0.66745$, $p = 0.0001$) appeared to explain more than

two-thirds of the variance. The rate was highest in Alaska (3.41 per 1,000 live births), high (2.40 to 2.79) in the Pacific Northwest, one of the "goiter zones"[66] where soil and drinking water are unusually low in iodine, and much lower (0.80-1.19) in the Southeast.[67] The SID rate correlated positively with soils high in sodium and negatively with industrial air pollution—now a significant source of iodine, which can be absorbed through the skin as well as inhaled.[64]

Foster also found racial differences related to consumption of iodine: in California the SID rate was 0.51 among Chinese and Japanese Americans and 5.93 among American Indians. Many Oriental Americans consume iodine-enriched seaweeds, few native Indians do. And soybean, used in some baby foods, is a goitrogen and can produce hypothyroidism in susceptible infants.[64] More iodine than usual is needed during pregnancy and lactation.

3.4 The two explanations of SID can be united in a new hypothesis. G.C. Willis, an earlier medical pioneer in Toronto, found in tests with guinea pigs that (a) subclinical scurvy induced by an ascorbate-deficient diet or infection, its result; and (b) arterial damage *occurred together.*[5] This may not be as anomalous as it seems: tiny blood clots initiate arterial damage,[37] and bleeding is typical of arterial wall thinning in scurvy.

Guinea pigs share with us not only our inborn failure to generate our own ascorbate and the hydrogen peroxide it makes and the peculiar metabolism of iron discussed earlier, but also the presence of lipoprotein(a).[6] So the finding applies very closely to humans. Which of the two explanations applies more strongly might vary according to diet—e.g. consumption of animal protein and junk foods—and biochemical individuality. (Willis could have induced arterial damage—but not scurvy—as Rinehart and Greenberg did with monkeys,[68] using a diet rich in animal protein and deficient in B6.)[54]

The supplemented ascorbate given to Dr. K' s infant patients prevented scurvy and "dropping dead" by correcting the ascorbate deficiency. And although Suzman doesn't mention this, vitamin C therapy fits his model too as explained by the *Multi-Source Oxysterol Injury (MSOI) Theory* of Atherogenesis.[36] In a generally overlooked but potentially critically important mechanism, supplemented vitamin C takes the alternative pathway[69] to prevent formation of homocysteine and the excessive *oxysterols* it generates. In sufficient quantity ascorbate, a powerful antioxidant, also destroys oxysterols ingested from processed foods and contacted from the environment.[70]

Vitamin B_6 functions as an *antioxidant,* at least at high concentrations[71-73] and perhaps by some yet unknown effect on cystathionine beta-synthase, the enzyme that makes B6 bioavailable to the body. In addition, individual SID babies may have inherited low ability to degrade homocysteine.[74]

Oxysterols are not only atherogenic but also carcinogenic,[52] and may react to food constituents to produce toxic components from nonlipid sources that become mutagenic.[75] Oxysterols have been detected in *mothers' milk,* in *umbilical cord blood,*[16] and in unusually heavy quantity in human *breast fluid.*[11] Some come from powdered formula containing oxysterols.[78] Some oxysterols normally circulate and, like cholesterol itself, serve a protective function; but levels of these from processed foods can reach 1,000 times normal.[79] Others come from the homocysteine derived from methionine in formula based on cows' milk[54] and from xanthine oxidase in homogenized milk.[80]

Other oxysterols are generated by the industrial waste, chlorine in drinking water;[81] it alters and destroys essential fatty acids needed for a healthy immune system.[82] Fluoride— another of the industrial leftovers for which our bodies serve as convenient toxic waste dumps—lowers thyroid activity,[83] increasing homocysteine.[54] It also lowers magnesium and blocks absorption of other nutrients needed to avoid arrhythmias and to resist infectious disease, further promoting accumulation of homocysteine.[54]

Fluoride increases infant mortality, partly by antagonizing ascorbate[84] and partly by decreasing the rate of white blood cell migration.[85] AIDS is commoner in fluoridated Switzerland than unfluoridated Germany[85] and in certain fluoridated than in certain unfluoridated U.S. cities.[86] A careful study should find the same for SID.

The U.S. Environmental Protection Administration stated that virtually all domestic tap water including that from wells below

8,000 feet altitude—is polluted with lead and other heavy metals.[87] Most purity tests check for disease organisms and overlook heavy metals. One, mercury from "silver" dental fillings and amalgams in the pregnant woman, goes into her fetus,[88] promoting coagulation and thrombosis and inducing resistance to antibiotics.[89] Polluted air is a source of oxysterols; it also provides iodine.[64]

3.5 Many SIDs are precipitated by some usually minor, often not even noticed insult to a seemingly healthy, rapidly growing baby; or the baby is slightly ill, as discussed earlier. Any stress including burn or injury, as well as illness, increases the body's need for ascorbate. A premature baby is more susceptible to SID than a full-term child:[25] it uses more ascorbate to fight greater stress. High incidence in winter might be explained by dry indoor air carrying more disease organisms; that from midnight to 6 a.m. remains mysterious.

3.6 Often the insult, Kalokerinos found, was an inoculation.[1,7] SIDs increased alarmingly after a "routine" immunization campaign. Death was common if a baby was inoculated during or soon after an illness, while scorbutic or sub-scorbutic. Leaving for a lecture trip, Dr. K. instructed the Caucasian substitute doctor not to inoculate any sick child. But imbued with the cocksure wisdom of prevailing medicine, he disregarded the warning. Two vaccinated Aboriginal babies died—and he left town in haste fearful for his life.[7]

Connaught Labs' 1986 DTP vaccine insert reads, "Sudden infant death has occurred in infants following administration".[47] William Torch, at the University of Nevada School of Medicine, noted that in one survey *two-thirds* of 103 American children who had suddenly died had been given DTP (diphtheria/ tetanus/ pertussis) vaccine within 3 weeks of death. Many died within 1 day of the procedure.[47] "In 1979, during a vaccination campaign in Tennessee there were 8 SIDs immediately following routine DPT vaccination. Of this group, 5 children died within 1 day of vaccination".[47] A study that same year at UCLA, sponsored by the U.S. Food and Drug Administration, indicated that in the USA approximately 1,000 babies die annually as a direct result of DPT vaccination, and these are classified as SIDs.[47] One survey is reported to have found a 7.3-percent risk of SID within 3 days after inoculation.[7]

Often given at the age of only two months, inoculation with toxic materials destroys any minimal ascorbate in the tiny body and may catastrophically damage the still developing blood and nervous systems.[90] Immunizations are big "money-spinners" for pharmaceutical drug companies and their allies, accounting for the insistence with which they are promoted.[7]

Japanese health authorities realized by 1975 that early inoculations were causing crib deaths. So they postponed them until the 24th month—and SIDs virtually disappeared.[91] Also, incidence of whooping cough (pertussis) during the first two years, when it is most dangerous and even life-threatening, dropped sharply. Instead of preventing whooping cough as intended, the inoculations promoted it as well as SID [V. Scheibner pers. comm. 1993]. *That information should have led to a recommendation at least to postpone vaccinations in Western countries.*

After-effects are followed only for a short time. But effects may not be seen for 30 years and will hardly be associated with the inoculation or the supposed watchdogs.[92] Long-term neurological impairment in the most sensitive has been found and shown on television.[7] Dr. Klenner concurred: "Most children don't need artificial immunizations and those that do, cannot use them!".[10] Some say that a mother rendered artificially immune to a disease by inoculation cannot pass a natural immunity to that disease on to her suckled babies.[7] In this and other ways, immunizations may contribute to the lowering of immunity and the spread of auto-immune diseases such as arthritis, and of AIDS throughout the industrial world.[93]

There is little if any evidence that immunizations lower disease rates. In most if not all cases, incidence had already dropped sharply when inoculations began, and the rate of decline did not accelerate [Russell Jaffe lecture, Well Mind Association Seattle 1990].[7] Maladies diminished only in areas where sanitation and hygiene improved.[7] And 180 doctors in Switzerland unanimously recommended against mass immunizations;[7] the Swiss Minster of Health wrote that immunizations

continue.

Massive anecdotal evidence shows that inoculations cause as many cases of disease as they prevent and maybe far more. See above regarding whooping cough in Japan. The supposedly protective substance is packed with preservatives such as *carcinogenic formaldehyde* [N.S. Green personal communication 1993]. And in one case it contained disease organisms that caused cancer in monkeys and could start a new epidemic[93,94] Whistleblowers were demoted or transferred.[9293]

An Australian research group strongly incriminated DPT immunization as guilty of causing many SIDs. Using electronic breathing monitors called "Cotwatch," which do not touch the baby, they tracked the breathing of 70 babies after DPT inoculations (cf. 1.3). Although all survived, they experienced breathing crises on certain days: typically the first 48 hours, the 5th-6th and the 11th-14th days. In other babies, *SIDs after DPT inoculations were bunched on those days.*[29]

Dr. Viera Scheibner, the retired principal research scientist of the New South Wales Health Department, has examined 20,000 pages of scientific publications relating SID to vaccinations. Her book on the subject was published: *Vaccination—100 Years of Orthodox Research shows that Vaccines Represent a Medical Assault on the Immune System,* Victoria, Australia: Australian Print Group. It includes evidence that *large-scale SID began with mass immunizations*—which is not now hard to believe.

The picture is much like that of dental mercury. The American Dental Association claims that "silver" fillings (which are more than 50% Hg) and amalgams are perfectly safe; the Hg is "locked in" to fillings. In fact, Hg is constantly inhaled and swallowed causing many health problems. Dr. Rogers comments, "The position of the ADA is like other phases of environmental medicine where if one chooses not to be aware of the research and in addition looks only for symptoms that affect an epidemic number of people right after the event in exactly the same way, then the problem doesn't exist. Convenient ignorance".[95] *The Inquisitors refused to look through Galileo's telescope since what he claimed to see could not be there.* How shockingly little has changed.

3.7 Other factors. Because of biochemical individuality re- suiting from genetic and acquired differences, some babies require much more, even many times more vitamin C or B6 than others.[96] And Dr. Kalokerinos thinks Aborigines may have a special immunity problem.[7] The adults' high consumption of powdered milk (1.1) suggests that any formula they used was powdered; that powder is atherogenic.[78] Some SIDs appear to have been triggered by *cigarette smoke*—ingested through the umbilical cord, in breast milk, or inhaled.[7,97] Passive smoking has been related to low birth weight and respiratory tract infections,[21] both of which should be minimized if not eliminated by appropriate ascorbate supplements. Other SIDs followed aspirin,[7] which hinders absorption of B_6 and C.

Inhaled vapors of toxic substances outgassed from an often newly furnished baby room have been proposed to explain why fewer SIDs seem to strike babies sleeping in mother's bed.[98] But at least through 1982, routine viral cultures and toxicicologic procedures had not been found helpful and were no longer part of the routine examination after a SID.[21] Others suggest alternating-current electromagnetic fields that increasingly bombard us, or magnetic variations such as those from underground oil or metal deposits and watercourses may make some babies more susceptible than others. The brain is 10 to 100 times more vulnerable to stress during sleep than while awake.[99] By the laws of physics, such influences are impossible. Yet "practitioners aware of these stresses report that 30 to 50 percent of chronically sick patients exhibit some kind of geopathic stress ...," and such diseases as arthritis often show geographically localized patterns.[100] Such variations in stress might alter susceptibility to SID.

Mother rolling onto baby: no. "We are convinced that the new mother, even in sleep, is subconsciously so acutely aware of her baby that she could not lie on the infant or permit any part of her body to endanger its respiration or welfare... [An exception might be] the mother who is drugged or intoxicated." And child abuse can nearly always be ruled out.[21]

Some did find evidence suggesting relative immaturity of the brain stem in SID babies. But given adequate ascorbate, those should survive. SID babies appear to have been

"different from normal" even before birth; they grew less than average, etc. Siblings of a SID baby are more likely to be affected: one study found 21 per 1,000 live births vs. the usual 2.[21] On a nationwide television show Oprah Winfrey interviewed a mother with 3 SIDs who emphatically rejected accusations of murder.

Magnesium deficiency operating through histamine release causing an anaphylactic reaction was proposed as a cause of SID.[101] Mg serves as a cofactor in many of the functions of vitamin C, and particularly of B6. But in two studies totaling 9 SID babies, Mg levels were within the range found in babies whose deaths had been expected. The finding was the same for Ca, Zn and other micronutrients.[21]

Dr. Chris Wheeler of New Zealand blames margarine in the diet of nursing mothers.[102] Besides being atherogenic and carcinogenic,[103] margarine facilitates the loss of trans-cholesterol, calcium and fat-soluble vitamins and other associated reactions.[102] These could increase the body's need for vitamins C and B_6 and so worsen their deficiency.

Further well known influences are food processing which destroys or discards nearly all nutrients including vitamin C and the enzymes required both for absorption of nutrients and for immunity; additives and chemicals such as monosodium glutamate (MSG) elevate the need for ascorbate. Processing and cooking also drive off all the hydrogen peroxide present in raw foods," which is needed for health.

Many babies are fed fake milk (formula) which not only is atherogenic if made from powdered milk,[78] but has been warmed in a microwave. Outlawed in Russia, microwaving destroys some of the nutrients and transforms some amino acids into carcinogens.[104] And warming in a microwave destroys 98% of immunoglobulin-A antibodies and 96% of liposome activity, reducing breast milk's resistance to infectious *E. coli.*[105] Further, the blood of people who consumed microwaved food for two months showed pathological changes compared to matched controls whose food was cooked with heat,[106] boding ill for such a person's offspring.

Catalytic converters substitute for other air pollution a series of nerve gases, says Hans Nieper, a Germany authority. These inhibit vital enzymes in cell membranes[85] and must increase SIDs near heavily traveled streets and highways. Depleted oxygen in central cities[107] might push some susceptible babies over the brink. Almost all these factors in life except underground geological features came with industrial life. Before, despite humanity's inherited failure to synthesize our own ascorbate people weren't deficient enough in it, or B_6, Mg etc. to cause SID.

3.8 This cascade of information elucidates a fact that Suzman learned about the geographical incidence of SID. During his 60 years of practice he interviewed people in many cultures not only in South Africa but in Asia and throughout the world on his lecture tours. *SID occurs only in industrialized societies,* where it is a leading cause of death in the first year. It is unknown in populations consuming largely vegetarian diets, living in non-toxified environments, not drinking rat poison (fluoride) and oxysterols- and trihalomethanes-generating chlorine,[81] not taking aspirin—and not given inoculations. *This fits the hypothesis that SID was rare before mass immunizations.*

So MSOI offers for testing a tentative explanation of SID, of the two doctors' success at its prevention, and the virtual disappearance of crib death in Japan after immunizations were postponed to the 24th month. By inference the new theory also suggests other ways to help eliminate SID, such as supplementing B6, folate, zinc and magnesium for both mother and baby.

Folate cofactors remethylation of homocysteine into methionine, an antioxidant, which is not itself dangerous.[54] Microgram quantities of timed-release folate lowered homocysteine below a defined safe level[107] in 60 percent of moderately homocysteinemic subjects; of B12 in absorbable form, 15%, and 10 mg/day of B6 in 15%. A patented, highly absorbable combination of the three did it in 90%; a placebo did not lower homocysteine [W.J. Serfontein pers. comm. 1993]. The recommended folate supplement for women who may become pregnant—made 15 years ago in Britain—should then lower risk of SID of atherogenic origin as well as neural tube birth defects.

The explanation covers breast-fed as well as bottle-fed babies. Further, about 50 per-

cent more male than female babies die SIDs.[25] Why? Adult males metabolize homocysteine less efficiently than women because they synthesize lower quantities of female sex hormones—which induce homocysteine metabolism[108] and limit peroxidation of lipids.[56] Perhaps that is true too of male babies. Earlier, the large, often unsatisfied need of male babies for *zinc* for the testicles was mentioned. If Dr. Suzman's explanation of SID is to be rejected, how are we to interpret this voluminous supporting evidence?

Researchers purport to learn about SID by study of dogs and other animals;[7] but all mammals except guinea pigs and primates generate their own ascorbate. Funds collected for such work provide salaries for "scientists," administrators and fund raisers but little if any information of use in understanding human SID.

4. Another wrinkle to the SID story: Estimates of annual American SIDs were 10,000 in 1980 and 5,300[64] to 7,000, about two per thousand births, by 1990. The decline is not known to have occurred elsewhere, except in Japan. I suggest that the improvement resulted from increased use of infant vitamin drops or their equivalent added to infant formulas, and have seen no evidence that such vitamin supplements or fortification are common abroad. The labels on bottles of Johnson & Johnson's *Tri-Vi-Sol* (R) for newborns state that a day's recommended one-milliliter quantity contains 35 milligrams of vitamin C; a chewable form for older babies, 60 milligrams.

The American pediatricians I called recommend such vitamin drops "for all babies"—a curious U-turn from what they were taught in medical school and from what most other doctors and many nutritionists teach. (An acquaintance's pediatrician, though, didn't suggest them.)

Although the quantities of ascorbate so ingested are smaller than in Kalokerinos' and Klenner's regimen—and may not be enough—they should prevent SID among the less sensitive. Or perhaps they are enough; does SID spare babies given the vitamin drops? Querying a SID mother might be a terrible experience for her—to realize that she herself potentially killed her baby by failing to provide the needed nutrients.[17] I sent this document to 30 pediatricians and asked if they know of a baby given the drops in the recommended quantity who died suddenly; of the five who replied, none knew of such a death or thought babies are vitamin C-deficient.

Why do some babies not get vitamin drops? Some parents read very little and cannot afford pediatricians' fees. And some who can, may choose to follow the advice of a pediatrician who doesn't recommend them and most other doctors and nutritionists, "You can get enough of all needed nutrients from a [never defined] balanced diet [of junk foods]." Most M.D. physicians know precious little about nutrition; most nutritionists and registered dietitians (R.D.) are 60-80 years behind scientific knowledge in their field.[109]

Those giving such advice may unknowingly be guilty of manslaughter; people following it probably suffer most adult heart attacks too.[36,110] Babies who still die suddenly must not be getting the vitamin protection, or not enough such protection for their biochemical individuality—or are not suckled by a mother who takes appropriate supplements.

The manufacturer of *Tri-Vi-Sol* declined to provide sales figures. The Mead Johnson specialist wrote, accurately, "A claim of the type that you proposed might be supportable would make the product a drug by regulatory definition, and thus require all the study and review [and multi-millions of dollars] needed for approval." (Let the FDA withdraw this product from sale and watch the SID rate climb.) Earl Conroy, D.C., N.D., of Motueka, New Zealand suggests an ideal way to rid the world of SID: eliminate processed foods, cola drinks and pasteurized milk—and take the supplements [pers. comm. 1993]. Perhaps more practically, SID, as well as most coronary heart disease and strokes, might be eliminated in the future by appropriate fortification of processed foods, cola drinks and milk with ascorbate, vitamins B6 and folate and cofactors including magnesium.[110] Since young women give birth, eliminating SID might take no more than about one generation. And the Japanese experience suggests SID could be decimated *with the stroke of a pen* by postponing the start of vaccinations.

Addendum 1

A study, "Risk of sudden infant death syndrome after immunization with the diphthe-

ria-tetanus-pertussis vaccine," by Marie R. Griffin et al (1988), *New Eng. J. Med.* 319:618-623, claimed to find no relation between SID and DTP inoculations.

However, Viera Scheibner in Australia revealed that her SID population[29] included the 109 crib deaths counted in the Griffin study. *Those American SIDs, like the Australian ones, bunched on the first two, the fifth-sixth, and the 11th-14th days.*

Addendum 2

(1) One-third of adults on the street probably have levels of vitamin C so near zero as to be consistent with clinical scurvy and another third, with subclinical scurvy [Schorah, C.J. (1981), Vitamin C status in population groups, in: Counsell, J.N. & D.H. Horning, *Vitamin C (Ascorbic Acid),* Englewood, Colorado, Applied Science Publishers; Cheraskin, E. (1993), *Vitamin C: Who Needs it?,* Birmingham, Alabama, Arlington Press & Co.]. It then is not hard to see why many babies are scorbutic or close to scorbutic.

(2) Bioflavonoids accompany vitamin C in fruits and wherever else they occur together, and probably weigh at least as much as the vitamin C with it. Then "taking vitamin C without bioflavonoids is like clapping with one hand" and all earlier tests of vitamin C need to be re-evaluated [Cheraskin, op cit]. It then follows that using bioflavonoids with the supplements program described in this article should materially strengthen the results.

Addendum 3

On the protective function of iron deficiency against infection. [40] and from Hans Raible Hosts station iron-binding proteins at potential sites of invasion. E.g., egg yolk contains 1 mg of Fe; but the white doesn't contain any—to starve potential microbial invaders that get through the porous shell. A powerful iron-binding protein comprises 12% of egg-white solids. Even Shakespeare wrote of using egg whites to heal infected wounds. The chief antimicrobial component of egg white is *conalbumin,* which is highly active at pH above 6.4. It suppresses growth of gram-positive and gram-negative bacteria and fungi.

Lactoferrin, the second of three known host-defense iron- binding proteins, constitutes as much as 20% of total protein content of human milk; bovine milk contains only 1/10 as much. Lactoferrin is released on degranulation of cells in a septic area; after combining with iron in the infected region, the metal-saturated protein is ingested by macrophages. It may also assist in killing microbial cells by helping iron catalyze formation of dangerous hydroxyl radicals. Lactoferrin can bind iron as the pH declines below 4.0, whether from lactic acid or other sources. Patients deficient in lactoferrin have recurrent gram-positive and gram-negative bacterial infections. This defense protein can be transported to sites of infection by neutrophils and synthesized locally at some sites. Its chief additional function is suppression of absorption of intestinal iron from diet and bile.

Transferrin is the third of three known host-defense iron-binding proteins. It can transport iron to host cells rather than withhold the metal from invaders. It is synthesized in hepatocytes, leukocytes and possibly by other cells. Many research papers have shown that antimicrobial activity is enhanced by a rise in transferrin and depressed by its lowering. It requires pH above 6.5 for best iron-binding activity.

Conalbumin and lactoferrin normally are highly unsaturated to permit the proteins to function as iron-withholding rather than iron-transport agents. Transferrin must engage in both functions. Normal values of transferrin in man vary with age from 69% at birth to 22%. It also has broad-spectrum antimicrobial ability. Patients deficient in it are at greater risk of bacterial and mycotic infections.

A bit of history: During infection, body levels of iron are depressed and this was called *anemia.* (It is now more appropriately called *hypoferremic response* to infection and chronic disorders.) So for several decades, extra iron was fed or injected—fighting the body's own efforts to heal itself! (Cf., from the newer Darwinian medicine, artificially lowering fever, swelling or body cholesterol.) However, the body was usually able to reject assimilation if fed, or stored the iron if injected. During the inflammatory process of infection, release of the metal from macrophages is inhibited preventing its normal recycling to transferrin. The mechanism also lowers plasma zinc and increases plasma copper.

Keeping iron low is an effective defense against infection: The Masai of East Africa live mainly on milk and blood from their

cattle.[111] So they have a low hemoglobin of about 11.7 +/- 0.8 g/dl and a transferrin saturation of only (14 +/- 2.6) %. On these low values they are free of malaria. When fed iron to get their iron up to what Westerners regard as normal values, hemoglobin rose to 13.1 +/-1.2 g/dl, and transferrin rose to (29 +/- 3.1) %, i.e., to the upper limit of normal values. Then 17% of the Masai had malaria attacks— and none of the untreated controls. Evidently, the malaria protozoon thrives on the additional iron, whereas deficiency is protective and otherwise does no great harm. It is certainly better than to have sickle cell disease or thalassemia which also are protective in malaria.

In addition, Masai had <9% rate of amebiasis; daily iron supplements of 6.2 g (as sulfate) for a year raised the amebiasis rate to 83%. Somalian nomads were fed 9 g iron (as sulfate) for a year: 38% developed active infections vs. only 8% of controls, $p < 0.001$. So much for the wisdom of Western doctors.

References

1. Kalokerinos, A. (1982), *Every Second Child*, New Canaan, Ct.: Keats Publishing.
2. Kalokerinos, A. & G. Dettman (1981), "The spark of life," *Health and Healing, Journal of Alternative Medicine* 1:15-19.
3. Cathcart, Robert F. III. (1981), "The method of determining proper doses of vitamin C for the treatment of disease by titrating to bowel tolerance," *J. Orth. Psych.* 10:125-132.
4. Dettman, G.C. & Kalokerinos, A. (1977), "Ascorbate intake of Fijians," *Med. J. Aus.* 26:2-5.
5. Willis, G.C. (1957), "The reversibility of atherosclerosis," *Canad. Med. Assoc. J.* 77:106-109.
6. Pauling, L. & M. Rath (1991), "An Orthomolecular theory of human health and disease," *Jour. Orth. Med.* 6:135-138.
7. Health Care Reform Group, Australia (1992), "The Hand that Rocked the Cradle," video.
8. Lee, L. (1993), "Food enzymes," chapter in *Holistic Health Encyclopedia*, Beverly Hills, California: Future Medicine Publishers.
9. Klenner, F.R. (1949), "The treatment of poliomyelitis and other virus diseases with vitamin C," *Southern Med. & Surg.* 111:209-214.
10. Klenner, F.R. (1971), "Observations on the dose and administration of ascorbic acid when employed beyond the range of a vitamin in human pathology," *J. App. Nutr.* 111:210-214.
11. McCabe, E. (1988), *Oxygen Therapies*, Morrisville, New York: Energy Publications.
12. Wells, K.H. et al (1991), "Inactivation of human immunodeficiency virus type 1 by ozone in vitro," *Blood* 78:1882-1890.
13. Noto, V. et al (1989), "Effects of sodium ascorbate (vitamin C) and 2-methyl-l, 4-napthoquinone (vitamin K3) treatment on human tumor cell growth in vitro," *Cancer* 63:901-906.
14. New Scientist 4/20/91.
15. "Breast milk: Can it slime away disease?" *Sci. News* 12/5/92:390.
16. "Cot death, looking up?" (1992), *Economist* May 9:108-109.
17. Kahn, A. et al (1988), "Infants with an apparent life-threatening event and possible risk for SIDS," *Padiatrie und Padiologie* 23:293-306.
18. "Crib death" (1976), *The Encyclopedia of Common Diseases*, Emmaus, Pennsylvania: Rodale Press:377-382.
19. Lachs, M.S. (1974), "Cot death—Thoughts on a personal experience," *Med. J. Australia* July 27:139-140.
20. Hoffer, A. (1991), "Editorial: Freedom of choice wins more freedom," *Jour. Orth. Med.* 6:55-56.
21. Valdes-Dapena, M. (1982), "The pathologist and the sudden infant death syndrome," *Amer. Jour. Pathology* 106:118-131.
22. Bergman, A.B. et al (1972), "Studies of the sudden infant death syndrome in King County, Washington: III Epidemiology," *Pediatrics* 49:860-870.
23. Roberts, P. et al (1984), "Vitamin C and inflammation," *Med. Biol.* 62:88.
24. Selye, H. (1978), *The Stress of Life*, New York: McGraw-Hill 25.Ebalo, E.E. (1993), "Putting SIDS risk into perspective," *Well Street Report* Feb./Mar.
26. Shaw, E.B. (1979), "Apnoea and unexpected death," *Lancet* 2:954.
27. Swift, P.G. & J.L. Emery, (1973), "Clinical observations on response to nasal occlusion in *infancy," Arch. Dis. Child.* 48:947-951.
28. Guilleminault, C. et al (1975), "Apneas during sleep in infants: Possible relationship with sudden infant death syndrome," *Science* 190:677-679.
29. Karlsson L. & V. Scheibnerova (1991), "Association between non-specific stress syndrome, DPT injections and cot death," Canberra: Second Immunization Conference.
30. Salonen, J.T. et al (1992), "High stored iron levels are associated with excess risk of myocardial infarction in Eastern Finnish men," *Circulation* 86:803-811.
31. Martin, W. (1984), "Do we get too much iron?" *Med. Hypotheses* 13:119-121.
32. Keown, P. et al (1985), "Ferroproteins and the immune response," *Lancet* i:44 (ltr.)
33. Sullivan, J.L. et al (1980), "Serum iron and

acute infections," *New Eng. J. Med.* 303:285.
34. Murphy, M. et al (1989), "Iron and the sudden infant death syndrome," *Brit. Med. J.* 298:1643.
35. Selby, J.V. & G.D. Friedman (1988), "Epidemiological evidence of an association between body iron stores and risk of cancer," *International J. Cancer* 41:677-682.
36. Hattersley, J.G. (1991), "Acquired atherosclerosis: Theories of Causation, novel therapies," *Jour. Orth. Med.* 6:185-198.
37. Morin, R.J. & S.-K. Peng (1991), "The role of cholesterol oxidation products in the pathogenesis of atherosclerosis," *Annals Clin. & Lab. Sci.* 19:225-237.
38. Association for the Promotion of Preconceptual Care (n.d.), "A lay person's notes on the interpretation of mineral analysis," n.p.
39. Linder, M.C. & H.N. Munro (1973), "Iron and copper metabolism during development," *Enzyme* 15:111-138.
40. Weinberg, E.D. (1984), "Iron withholding: A defense against infection and neoplasia," *Physiological Rev.* 64:65-95.
41. Dwyer, T. et al (1991), "Prospective cohort study of prone sleeping position and sudden infant death," *Lancet* 378:1244-1247
42. Morse, M.L.(1991), "Consult with doctor about baby's sleeping position," *Seattle Post-Intelligencer* (ltr.) Aug. 25.
43. Coombs, R.R.A. & S.T. Holgate (1990), "Allergy and cot death: With special focus on allergic sensitivity to cows' milk and anaphylaxis," *Clin. Exp. Allergy* 20:359-366.
44. Stone, I. (1972), *The Healing Factor: "Vitamin C" Against Disease,* New York: Grossett & Dunlap.
45. Suzman, M.M. (1973), "Effect of Pyridoxine and low animal protein diet in coronary artery disease," *Circulation* 48:suppl. IV, IV-254, abstract.
46. Jaffe, D. et al (1971), "Coronary arteries in newborn children: Intimal variations in longitudinal sections and their relationships to clinical and experimental data," *Acta Paediat. Scand.* Supp. 219:1-27.
47. Chaitow, L. (1979), *Vaccination and Immunization: Dangers, Delusions, and Alternatives,* London: C.W. Daniel.
48. Hung, J. et al (1990), "Association of sleep apnoea with myocardial infarction in men," *The Lancet* 336:261-264.
49. Harker, L.A. et al (1974), "Homocystinemia: Vascular injury and arterial thrombosis," *New Eng. Jour. Med.* 291:537-543.
50. Harker, L.A. et al (1976), "Homocystine-induced arteriosclerosis: The role of endothelial cell injury and platelet response in its genesis," *Jour. Clin. Investig.* 58:731-741.
51. Parthasarathy, S. (1987), "Oxidation of low-density lipoprotein by thiol compounds leads to its recognition by the acetyl LDL receptor," *Biochim. Biophys. Acta* 917:337-340.
52. Fieser, L.F. (1954), "Some aspects of chemistry and biochemistry of cholesterol," *Science* 119:710-717.
53. McCully, K.S. & M.P. Vezeridis (1988), "Homocysteine thiolactone in arteriosclerosis and cancer," *Res. Comm. Chem. Path. Pharmacol.* 59:107-119.
54. McCully, K.S. (1983), "Homocysteine theory: Development and current status," *Atherosclerosis Reviews* 11:157-246.
56. Ueland, P.M. & H. Refsum (1989), "Plasma homocysteine, a risk factor for vascular disease: Plasma levels in health, disease and drug therapy," *Jour. Lab. Clin. Med.* 114:473-501.
56. Yaki, K. & S. Komura (1986), "Inhibitory effect of female hormones on lipid peroxidation," *Biochem. Int.* 13:1051-1055.
57. Ellis, J.M. (1985), *Free of Pain*, revised edition, Dallas: Southwest Publishing.
58. Delport, R. et al (1991), "Relationship between maternal and neonatal vitamin B6 metabolism: Perspectives from enzyme studies," *Nutrition* 7:260-264.
59. Azuma, J. et al (1976), "Apparent deficiency of vitamin B6 in typical individuals who commonly serve as normal controls," *Res. Comm. Chem. Path. & Pharmacol.* 14:343-348.
60. Folkers, K. et al (1977), "Studies on the basal specific activity of the glutamic oxaloacetic transaminase of erythrocytes in relationship to a deficiency of vitamin *B6," Res. Comm. Chem. Path. Pharmacol.* 17:187-189.
61. Vir, S.C. & A.H. Love (1977), "Vitamin B6 status of the aged,'V. *Vitam. Nutr. Res.* 47:364-372.
62. Gruberg,E.R.&S.A.Raymond(1981),5eyo«c? *Cholesterol: Vitamin B6, Arteriosclerosis, and Your Heart,* New York: St. Martin's Press.
63. Suzman, M.M. (1984), Nutritional and metabolic factors in the development of coronary artery disease in early life: The possible role of dietary protein and Pyridoxine, Unpub. abstr.
64. Foster, H.D. (1988), "Sudden infant death syndrome and iodine deficiency: Geographical evidence," *Jour. Orth. Med.* 3:207-211.
65. West, B. (1990), *The Cholesterol Folly,* Carmel, California: West Publishing.
66. Berkowsky, B. (1992), "Hypothyroidism: A pandemic symptom," *Health Freedom News* Sept.: 8-13.
67. Spiers, P.S. (1980), "Risk of sudden infant death syndrome by area of residence in the Unites States," *International Journal of Epidemiology* 9:45-48.
68. Rinehart, J.F. & L.D. Greenberg (1949), "Arteriosclerotic lesions in pyridoxine-deficient monkeys," *Amer. J. Pathology* 25:481-492.

69. McCully, K. S. (1971), "Homocysteine metabolism in scurvy, growth and arteriosclerosis," *Nature 231:391-392.*
70. Frei, B. (1991), "Ascorbic acid protects lipids in human plasma and low-density lipoprotein against oxidative damage," *Am. J. Clin. Nutr.* 54:1113S-1118S.
71. Witting, A.L. (1957), "The relationship of Pyridoxine and riboflavin to the nutritional value of polymerized fats," *Jour. Amer. Oil Chemists' Soc.* 34:421-424.
72. Kuzuya, F. (1991), "Vitamin B6 and arterio-sclerosis," *Daiichi Vitamin News* 6:1-7.
73. Zhou, Y.-C. & R.-L. Zheng (1991), "Phenolic compounds and an analog as superoxide anion scavengers and antioxidants," *Biochem. Pharmacol.* 42:1177-1179.
74. Genest, J.J. Jr. et al (1991), "Prevalence of familial hyperhomocyst(e)inemia in men with premature coronary artery disease," *Arteriosclerosis and Thrombosis* 11:1129-1136.
75. Peng, S.-K.&C.B.Taylor(1984), "Cholesterol autoxidation, health and arteriosclerosis," *World Rev. Nutr. Diet* 44:117-154.
76. Eberlein, W.R. (1965), "Steroids and sterols in umbilical cord blood," *J. Clin. Endocr. Metab.* 25:1101-1118.
77. Petrakis, N.L. et al (1981), "Cholesterol and cholesterol epoxides in nipple aspirates of human breast fluid," *Cancer Res.* 41:2563-2565.
78. Taylor, C.B. et al (1979), "Spontaneously occurring angiotoxic derivatives of cholesterol," *Amer. J. Clin. Nutr.* 32: 40-57.
79. Smith, L.L. (1991), "Another cholesterol hypothesis: Cholesterol as antioxidant," *Free Radic. Biol. & Med.* 11:47-61.
80. Oster,K.A.& D.J. Ross (1983), *The XO Factor*, New York, N.Y.: Park City Press.
81. Morris, R.D. et al (1992), "Chlorination, chlorination byproducts, and cancer: A meta-analysis," *Amer. Jour. Public Health* 82:955-963.
82. Bercz, J.P. (1992), *Chem. Research in Toxicology* May/June.
83. Yiamouiannis, J. (1986), *Fluoride: The Aging Factor*, Delaware, Ohio: Health Action Press.
84. "Fluoride a known vitamin C antagonist" (1965), *Prevention* June.
85. Nieper, H. (1992), Lecture, June 23.
86. Carson, R. (1989), *Alternative Prostate Remedies*, Winter Park, Florida: Reality Publications.
87. Janney, P. (1992), "Health dowsing in the 1990s" (Audio tape), Sylvia, North Carolina: Goodkind of Sound.
88. Vimy, M.J. et al (1990), "Maternal-fetal distribution of mercury (203 Hg) released from dental amalgam fillings," *Am. J. Physiol.* 258:939-945.
89. *Science News* 4/10/93.
90. Prechtl, H.F.R.(1984), "Continuity and change in early neural development," in Prechtl, H.F.R. (ed.), *Continuity of Neural Function from Prenatal to Postnatal Life*, Philadelphia: Lippincott: 1-15.
91. Cherry, J.D et al (1988), "Report of the Task Force for Postponement of Immunization,"' *Pediatrics* Supplement:93-94.
92. "The boat that never rocks" (1993), *Second Opinion* Jan.: 1-6.
93. Nichols, W. (1972), "Division of Biologics Standards: The boat that never rocked," *Science* March 17:1225-1230.
94. Coulter, H.L. (1990), *Vaccination, Social Violence and Criminality*, Washington, D.C.: Center for Empirical Medicine.
95. *Northeast Center for Environmental Medicine Health Letter*, Spring 1993.
96. Williams, R.J. (1956), *Biochemical Individuality*, New York: Pitman.
97. Cross, T. et al (1990), "Gas phase cigarette smoke (CS) induces lipid peroxidation in human plasma," Free Radic. Biol._Med. 9 (Suppl.):69 (abstr.).
98. Green, N.S. (1993), "Sudden infant death syndrome: A possible cause," *Let's Live* January 76-78.
99. Maes, W. (1990), "Baubiologie," *P.P.N.F Journal* 14:1-12.
100. Scott-Morley, A.J. (1985), *Geopathic Stress and Its Significance*, Institute of Bioenergetic Medicine, Dorset, England.
101. Caddell, J.L. (1972), "Magnesium deprivation in sudden unexpected infant death," *Lancet* 2:258-262.
102. Wheeler, C. (1992), "Margarine guilty in infant deaths?" *Soil & Health* July-August:30.
103. Rogers, S. A. (1991), *Tired or Toxic?* Syracuse, New York: Prestige Publishing.
104. Lee, L. (1992), "Health effects of microwave radiation," *Earthletter* 2:13-15.
105. Baker, E. (1991), *The UnMedical Miracle - Oxygen*, Indianola, Washington: Drelwood Communications, Inc.
106. Quan, R. et al (1992), Effects of microwave radiation on anti-infective factors in human milk, *Pediatrics* 89:667-669.
107. Microwaves: Scientific proof of dangers (1992), *Jour. Franz Weber* 19:3-10.
108. Ubbink, J.B. et al (1991), "The prevalence of homocysteinemia and hypercholesterolemia in angiographically defined coronary heart disease," *Klin. Woch.* 69:527-534.
109. Ueland, P.M. et al (1992), "Plasma homocysteine and cardiovascular disease," in *Atherosclerotic Cardiovascular Disease, Hemostasis, and Endothelial Function*, ed. R.B. Francis Jr., New York, Marcel Dekker: 183-236.

109. Pauling, L. (1986), *How to Live Longer and Feel Better*, New York: W.H Freeman.
110. Hattersley, J.G. (1993), "Don't have that heart attack!," chapter in *Holistic Health Encyclopedia*, Beverly Hills, California: Future Medicine Publishers.
111. Mann, G.B. (1972), "Atherosclerosis in the Masai," *Am. J. Epidemiology* 95:26-37.