

# Ascorbic Acid and Other Modern Analogues of the Germ Theory

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## On the Cost of Traditional Opposition to Modernization in Clinical Medicine

*The Modus of Opposition.* There is an almost universally held view that medicine is and should be a prestigious profession. Sadly, in recent decades, significant erosion of this view has occurred. The general nature of the erosion and other published laments are cited here. There is preponderant agreement among scientists and the lay public that medicine has an obligation to know what “is known” (i.e. that given modalities have been reported in the literature by competent authors to be far superior to corresponding treatments of choice, but their adoption has been opposed without reason). Work by Hoffer and Pauling led to a fundamental principle of orthomolecular medicine which involves the adjustment of levels of molecules that are normally in the body which can both prevent and cure disease with little toxicity or side effects compared to xenobiotic drugs. The theme detailed in this paper was developed by Pauling,<sup>1</sup> Klenner (p.63),<sup>2</sup> Shute (pp. 77-83, Appendices),<sup>3</sup> Coca (pp.185-9)<sup>4</sup> and others (hereinafter called Pauling et al.) who: (1) cite evidence they claim proves that virtually every disease can be treated with far greater safety and efficacy<sup>5,6</sup> i.e. with less Morbidity & Mortality (M&M) and far less cost, with rejected modalities (frequently orthomolecular) alone, or adjunctively to standard care in some cases; (2) report that mainstream medicine has either ignored or actively opposed adoption of these modalities by falsely condemning them without investigation or proper clinical trial; (3) claim that these actions have doomed most US citizens to disease-filled lives and early deaths after enormous suffering and expense from invasive,

outmoded, inefficacious “treatments of choice”; (4) state the opposition comes from self-appointed but highly influential “authorities” (often identified) who most frequently are not practicing physicians, have never studied or used, and do not understand the modalities they condemn, such as orthomolecular ascorbic acid or ascorbate (AA), or even the genetic basis for megavitamin therapy (Ch.11)<sup>1</sup> or that the strength of one’s convictions is no criterion of their validity; and (5) name true experts from mainstream whose endorsement of rejected modalities was also ignored (AA in surgery for wound strength and buffy coat, and in myocardial infarct for buffy coat, etc; high vitamin E for phlebitis, other thromboses, hemolysis). These rejected modalities are all analogues of the germ theory.

*Germ Theory Analogues.* History insists medicine has shown the same characteristic behavior, denying that blood circulated in the body, denying the germ theory, denying the role of ascorbic acid and other “analogues of the germ theory” (i.e. basic facts readily proven from the literature to be of great importance and subsequently adopted although initially vigorously opposed at enormous cost in M&M). There is evidence from the life of Westin Price, PhD, DDS, impressive scientist and coworker of Charles Mayo, MD, that some senior physicians in large city hospitals still had not accepted the germ theory as recently as 1920. We study here the evidence of Pauling et al. using the modern analogues, the first of which is AA (although commonly called “vitamin C;” we show it is not a vitamin). If it is true that mainstream medicine is chronically unable to modernize (i.e. impartially evaluate new modalities in a timely way) for which Pauling et al. make a compelling case, there must be extenuating circumstances. Certainly, the vast

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majority of mainstream practicing clinicians are highly educated and motivated, hard working people (no one works harder) who would favor better modalities even if “unprofitable” but, it appears, are losing well deserved respect through no fault of their own. Clinicians: (1) will not have had time to read the medical literature because of the “size problem” discussed below; and (2) will have heard entirely erroneous opinions from high “experts” on the matters of interest here. Due to peer pressure, time pressure and mindset in the self-regulated medical profession, the new modalities continue to be ignored. There is a sad difference in this regard between the communities of physics and clinical medicine. When something new is tentatively announced in physics, every physicist in the world is interested and wants to know about it. However, in medicine it seems that essentially no one wants to hear about the germ theory analogs.

*The Literature Size Problem.* Medical science literature has become essentially inaccessible because of its size. At the University of Washington, the Health Sciences Library receives over 40 million new pages per decade from the journals to which it subscribes. Most of this material is both excellent and relevant to clinical medicine. It isn't possible to turn this many pages in a decade. No clinician (or even researcher) could possibly find the time to read 1% (40,000 pages/year) of the flood. Thus, we are all ignorant of the greatest findings. It might be stated: there is no area of human endeavor where ignorance (of what is known) is growing as rapidly as in clinical medicine.

*What Can Be Done?* Relevant history of AA and some other related analogs are reviewed here with descriptions of the immense harm from their neglect. It has been estimated that the total annual U.S. health care cost including both private and public funds exceeds \$1 trillion, i.e. over one sixth of the national debt or \$4000 per capita. In spite of this largess, it is widely stated that these 273 million citizens suf-

fer more invasive, toxic, ineffective life-shortening therapies than any comparable group in history. The question is considered of whether the self-regulating medical establishment can be allowed to continue ignoring new modalities on the basis of ignorant bias of an influential few whose style seems to be “condemnation without investigation,” as per Pauling's meticulous exposé of many cases.<sup>1</sup> An alternative might be the creation of a committee, shielded from influence, requiring that advocates of “analogs” be allowed to demonstrate, for example, that AA will cure most viral infections (including acute hepatitis and polio), most cancer (with glycemic control) and most otherwise lethal intoxications. Severe penalties would be imposed for: (1) misleading mainstream medicine or the public regarding the true value of the “analogs”, thereby greatly increasing M&M and care cost, literally bankrupting the nation and killing the people; or (2) influencing editorial policy of medical journals against manuscripts that report benefits of “unprofitable” modalities.

#### A Preview of Four Major “Analogues”

Recall from the opening paragraph, Pauling et al. state that fundamental orthomolecular modalities such as AA, vitamin E (E), coenzyme Q<sub>10</sub> (CoQ<sub>10</sub>) and glycemic control (or unrefined diet) have all been proven to enhance health and longevity and have much lower cost and greater efficacy for most diseases than corresponding conventional care (for details, see later sections and references cited). The following “Mammals” section seeks to clarify widespread misconceptions regarding AA, a modality of central interest in this paragraph and most of the others. But, before we get into that, we wish to show that opposing the adoption of these four modalities is not “saving” people from quackish nostrums, but dooming them to standard care M&M, which most people can only escape by the disease prevention provided by these adop-

tions. People whose intake provides the RDA for AA (60 mg) and E (10 mg), the average diet's CoQ<sub>10</sub>, and 50% of calories from US refined foods<sup>7</sup>: (1) will have a body AA pool of about 1.5 g; this could last a month if unperturbed, but can fall to 0.5 g in one day of pain, fear, or other stress; at that point the white blood cells (WBC) or buffy coat level is scorbutic, resistance to infections and ability to heal are very depressed, a typical finding in heart attack and non-elective surgery patients; (2) will all be E deficient, because the US polyunsaturated fatty acid intake is several fold in excess of 1 g/mg E, increasing the susceptibility for rapid aging, cancer, heart disease, hemolysis, stroke and other thrombotic accidents; the <10% hemolysis of "normal healthy" people on the hydrogen peroxide test of RBC is due to E deficiency and disappears with 200 IU E daily; (3) Additionally the 20 mg CoQ<sub>10</sub> in the average diet becomes inadequate since endogenous synthesis declines after age 20, and brain levels halve between 45 and 70, worsening or causing many disorders and increasing risk of stroke;<sup>8</sup> and (4) risk and severity of every major disease are increased by the refined diet.<sup>7,9</sup>

### AA in Mammals

*Normal Mammals and Stress.* As Pauling et al. reported, ignorant self-appointed "authorities" have conveyed to clinician and lay readers alike false and dangerous notions such as: AA doses much above a gram are innately *dangerous and wasteful* because white blood cells (WBC) are *saturated*. When viewed in the perspective of the Pauling et al. exposé and the papers to which they lead, doesn't it appear that rejection of the analogs (AA alone!) was responsible for millions of deaths and trillions of dollars? Correction of the three falsities above is given briefly in three numbered statements following (and in detail in the rest of this and later sections): (1) the mammals discussion here and other sections below, cited explain why large

doses, even >200 g/d AA have given great benefit in all patients (rapid healing, curing most "incurable" infections including polio) except humans with genetic susceptibility to hemolysis (see below); (2) spilling AA in the urine is viewed as wasteful by the "authorities" who seem to be unaware that mammalian evolution selected plasma AA levels that provide normal healing and resistance to disease and stress and which, even in the unstressed state, are well above the renal threshold (about 1 mg %), so continually spilling AA in the urine is the price that must be paid for these qualities by both normal (AA synthesizing) mammals and others who like humans must supplement (adequately) to do so;<sup>10</sup> and (3) thus, for animals and humans using AA correctly, WBC AA (usually measured as buffy coat AA) does not "saturate" but is pumped up to a large multiple of the plasma level that, as stated in (2) above, is always higher than renal threshold.

There is one simple way to gain much understanding of the true kinetics of AA and the reasons for the confusion that dominates modern medicine regarding this small but extremely important molecule. First, we cite a widely accepted fact that, among the approximately 4,000 mammals, only four, humans, the other primates, the guinea pig and a fruit eating bat are known to have lost, through a genetic defect, the ability to synthesize AA, and must get it from exogenous supply. They are considered "abnormal" in this defect that has strongly influenced human history (eg. military campaigns, especially naval). It is believed all other mammals synthesize AA copiously from glucose in the liver and that AA has a number of functions that are very necessary for health and survival, especially in stress.<sup>11</sup> Now, we consider what happens in stressed "normal" mammals and what role AA plays. A normal mammal (goat, dog, cat) can be stressed in any of several ways (by disease, intoxication, pain, rage): the first two can occur in a normoglycemic

state such as a rat given aspirin. Sixty years ago, it was shown that, in response to aspirin, barbiturates or other intoxicants, rats would increase their synthesis of AA 100 to 1000 times and excrete urine AA far in excess of their total body stores daily. It has been inferred that the great elevations of plasma AA necessary to excrete such quantities in the urine is many times the renal threshold (possibly >100) (p.109).<sup>12</sup> If prolonged for a day or more, such plasma AA rises result in corresponding increases of AA in WBC and extreme stimulation of cell mediated immunity (CMI) ability to resist disease by destroying viruses and other pathogens and some tumors.<sup>13-26</sup>

To provide the vital needs of a special metabolic pathway called the HMP shunt (or the "pentose pathway"), AA is required at very high levels in WBC. The HMP shunt: (1) supplies ribose needed to copy DNA in cell division (when, for example, the disease antigen is sensed and cytotoxic T-cells need to multiply to destroy the pathogen); and (2) helps regenerate the toxic chemicals necessary for use in phagocytic cells to destroy pathogens they ingest (phagocytize). The rate of the HMP shunt increases about 700% as plasma AA rises from 0.7 mg% (a typical human value) to 140 mg% (100 times the renal threshold) and stimulates WBC functions correspondingly.<sup>13</sup> There can be no doubt that this marked increase in CMI including increased interferon production at high AA levels must have been developed during evolution in response to infective challenge.

*Humans and Stress.* In the human many AA functions are the same as in normal mammals, thus, one realizes the system "expects" the adrenal response to infective or trauma stress to be supported by AA synthesis from the liver which does not occur, leaving every sick or surgical patient in CMI anergy (WBC suppression). This dangerous fall of WBC AA to scorbutic levels within 12 hours after stress was reported in 31 consecutive heart attack pa-

tients.<sup>27</sup> The expected striking increase of CMI at extremely elevated plasma AA and prompt ability to cure polio, viral encephalitis, and acute hepatitis, in a few days has been reported repeatedly by numerous investigators using intravenous and oral AA (which replaces the missing liver response.<sup>12,14,16,18-20</sup>) in humans. Thus, from consideration of the stress response in normal mammals, we see that AA is not a vitamin (a nutrient needed in minute quantities) but a molecule with many functions that may be required in amounts from ten to >100 g/d to obtain the benefits gained by normal mammals (Ch.4)<sup>12</sup> and thus, humans must greatly augment body AA stores during disease or other stress. Stone<sup>10</sup> summarizes National Research Council AA RDAs for three defective mammals, human, monkey, and guinea pig (in mg/kg body weight) respectively as 1, 55, and up to 167 (depending upon guinea pig diet)! If a 60 kg human desires a health status as high as the monkey or guinea pig, it appears the RDA should be between 3 and 10 g/d. In the same source, Stone tells us that Klenner, Pauling, Dr Albert Szent-Györgyi (Nobel Prize in Medicine for AA research), and other notables all agreed with him or called for even higher RDAs.

Now, we return to the educated human who wishes to supplement AA to simulate the missing liver synthesis. In the stressed normal mammal, the WBC are continuously (possibly days or weeks) in blood that contains many times the usual level of AA. It was shown in 1961 that WBC (which normally "pump up" their internal AA levels to 50 times the existing plasma level) require about 20 hours incubation in grossly elevated AA to pump up to 50 times that new level.<sup>17</sup> This shows that WBC don't "saturate", as has been suggested by others to occur at very low AA intake. It is a remarkable fact that this amplification of WBC AA occurs, but unfortunate for humans that it takes so long to adjust. It is simple to elevate plasma AA levels to 4 mg/dL as described

by Lewin (p.151)<sup>12</sup> if a human adult takes oral AA 1 g/hr thus permitting exposure of the CMI to sustained elevations. Although very high AA levels have been shown by many investigators<sup>2,18-22</sup> to enhance CMI and provide rapid recovery, truly sustained AA elevations in plasma require constant oral AA dosing during acute disease. Robert F. Cathcart, MD, world famous for his work in orthopedic surgery at Stanford, developed the AA bowel tolerance protocol, so effective for infectious diseases, and gives much emphasis to this need.<sup>14</sup> For this reason, physicians such as Klenner and Cathcart, who were successful in treating polio, viral encephalitis, hepatitis and AIDS with intravenous ascorbate (IVC) drip, also always gave oral AA around the clock (time release AA tablets for outpatients in sleep).

#### Hemolysis: AA and Vitamin E

It is important to anticipate hemolysis and prevent it by increasing E intake with high AA. However, without mention of E, Klenner and Cathcart report treating over 30,000 patients with multigram doses of AA for over four decades without a single report of serious side effects. In a standard *in vitro* test of hemolytic tendency of RBC using hydrogen peroxide, a "normal" (no supplemental E) human's value (9%) may typically be increased by half (to 14%) one day after ingesting 1 g AA (Mengel, personal communication). Why is hemolysis not seen in normal subjects at high AA? The following hypothesis is offered to explain: these RBC, the 9% that disintegrate under oxidative stress of the test, are the oldest, ie, nearest to the 120 day nominal life span; the increased hemolysis observed after (small) AA intake is a toxic effect due to DHA (AA oxidized in the RBC; 75% of blood DHA is in RBC); but, if large doses of AA, i.e. multigram, oral or IV are given, the redox potential (found using the ratio AA/DHA in the Nernst equation) is so reducing that this ratio remains high, and DHA in RBC is too low to cause hemolysis. Thus, the safety and

efficacy of AA have been proven in many thousands of patients (who have no genetic susceptibility to hemolysis) and reported in detail by Klenner,<sup>2,18,20</sup> Hoffer,<sup>5,6</sup> and Cathcart.<sup>14,15</sup>

However, in subjects with these lytic tendencies, such as glucose-6-phosphate dehydrogenase deficiency (G-6-PD), sickle-cell anemia and beta-thalassemia, this RBC fragility must be addressed. Individuals with this defect are at increased risk of hemolysis from various drugs, infections and other stresses. Improved hematological parameters following long term supplementation of E at 800 IU/d is reported.<sup>28</sup> Use of AA should still be approached with great caution and only with RBC hemolysis monitoring. Without these measures, there has been one report of a death,<sup>29</sup> and several cases of hemolytic emergencies associated with large doses of AA.<sup>30,31</sup> It has been recognized for almost 50 years that vitamin E protects red blood cells from hemolysis.<sup>32</sup> Chen showed that supplemental vitamin E strongly diminished red blood cell hemolysis in rats given AA, with the protection increasing in a dose-dependent way with the level of E.<sup>33</sup> Thus it seems prudent to supplement E when taking multigram doses of AA.

#### Glycemic Control as a Modality

Almost 2,000 years ago in the time of Galen, it was observed that tumors grew poorly or not at all in underfed animals. As a result of a simple theory called the "glucose-ascorbate antagonism" and strongly supporting studies in both humans and animals, it now appears certain that CMI works best when blood sugar is low.<sup>23,24,34</sup> Of course, one doesn't want it too low. Humans become unconscious (not necessarily harmful) below 40 mg%, brain damage is reported to occur below 20 mg%, but CMI is reported to work well down to 10 mg %. In frequent or prolonged hypoglycemia, care must be taken regarding cortisol rise (and the associated lympholytic effects)

that differs greatly among subjects and conditions. Cancer, infections and other diseases such as CVD, have lower incidence and heal more rapidly if AA is adequate at the low blood sugar levels seen in primitive diets, i.e. 50 to 90 mg%.<sup>35,36</sup> Deficiency in trivalent chromium in US soil and diet has long been said to be a cause of impaired glucose tolerance and disease.<sup>37</sup> Some questions have been raised about the safety of Cr picolinate as a Cr supplement.<sup>23</sup>

### Stress and AA

Rapid response of the adrenals in emergency is vital to mammalian survival and its mediator, "fight or flight." It is well known that the highest AA concentration in the adult human (70 times plasma level) occurs in the adrenal cortex, where it is used as an electron donor in myriad syntheses, but it can be depleted rapidly under severe stress (such as injury, intoxication or fear). This can occur in hours in humans; but, in normal mammals, liver production of AA can increase over 100 fold synchronous with stress onset replacing the adrenal stores (maintaining readiness for the next emergency, improving stamina, rate of healing, resistance to stress and disease and bolstering other protective and repair processes). Even when not in stress, normal mammals such as the dog have plasma AA levels higher than the renal threshold and void AA in the urine 1 g/d. Monkeys given the human equivalent of 4 g AA/d were far more resistant to extreme cold exposure than monkeys given the equivalent of 300 mg daily.<sup>38</sup> In a study of 100 elderly hospital patients with adrenal insufficiency (and other disorders as primary), administration of 0.5 g AA resulted in immediate return of adrenal function.<sup>39</sup>

### Allergy and AA

*Anaphylactic shock and AA.* Since the 1930's when AA first became available,

many researchers have studied anaphylaxis and showed that AA could prevent death and even the symptoms of shock in a sensitized animal. Their results demonstrated a dose dependent protection that was reproducible for a given species and allergen type and dose. For example in guinea pigs sensitized to horse plasma 50 mg prevented shock symptoms and death whereas 20 mg only delayed death.<sup>40</sup> Extrapolation to a 70 kg human would suggest that 15 g might be required for protection against symptoms and death.

For 40 years, there have been theory and evidence that allergies are stress diseases that can be prevented and frequently cured by nutritional therapy including large intakes of AA, pyridoxine (vitamin B<sub>6</sub>) and pantothenate (vitamin B<sub>5</sub>)<sup>41,42</sup> It has long been known that urine and plasma levels of ascorbic acid are low during seasonal or other exacerbations of allergic rhinitis<sup>43</sup> and that in humans AA antagonizes the airway constriction effect of histamine.

*Allergy and Disease.* Arthur F. Coca, MD, founder and first editor of the Journal of Immunology, Medical Director of Lederle Labs for 17 years and namer of atopy demonstrated that (mainly) noninfectious disorders of virtually every body system could be caused by allergies to foods or inhalants and could be eliminated by avoidance of the allergens. His disease list included many "incurables" treated with great expense and morbidity by mainstream medicine. He developed the simple "pulse test" for allergen identification.<sup>4</sup> Although the findings of other great allergists (W. Vaughan 1927; H.J. Rinkel 1944) and internists (Walter Alvarez of Mayo) directly supported his work, it was rejected without trial by the same arrogance and ignorance ensconced in the seats of power that have suppressed the other "analogs." Three mechanisms uniting these diseases as allergic dysautonomias were explained by Ely in 1978 using one of the clues related to the biochemistry of schizophrenia uncovered by Hoffer. It has been widely

reported that the psychosis of institutionalized persons recovers in about four days on water fast (allergen avoidance) (Well Mind Assn Newsletters, Jun. 1982, Oct. 1989, Dec. 1989, Seattle, WA). In view of the incredible simplicity and extremely low cost to test Coca's claims—at least, in incurables of high pain such as migraine and of high M&M and cost such as hypertension—the question, “Why isn't it tried?” is asked by skeptics from all walks of life (including the vast majority of mainstream practicing clinicians).

### Cancer and AA

*Vale of Leven and Mayo Clinic Studies.* Cameron and Pauling reported a prolongation of survival over 4.2 times as great in 100 untreatable cancer patients given AA 10 g/day until death when compared to a control group of 1,000 patients matched for age, sex and disease.<sup>44</sup> The Mayo Clinic did two studies that were purported to test the validity of the Cameron/Pauling AA modality. Pauling has summarized the results of both studies integrated with much related material in his book (p.234)<sup>1</sup> the source of the details in this paragraph. Pauling states that the Mayo Clinic trials were not equivalent to Vale of Leven and that the results of the second Mayo trial (published in a leading journal) were misrepresented to the American people by a number of high placed authorities in mainstream medicine as proof “that vitamin C has no value against advanced cancer and recommended that no more studies of vitamin C be made”. Inter alia, Pauling also states that the Mayo patients did not receive AA continually until death, but for only a short time (median 2.5 months) and that none of them died while taking it, but they were studied for 2 more years and had not received any AA for a long time (median 10.5 months) at death but these incredible discrepancies were not revealed to the public. In the rest of Chapter 19 and elsewhere throughout the book, Pauling argues that large numbers of deaths result from the facts that both physicians and the public were con-

vinced by the widely publicized incorrect and misleading statements of the authorities that multigram AA has no value in cancer or most other diseases.

If high AA and aggressive glycemic control are not adopted, patients have greatly reduced probability of achieving tumor free remission. Virtually all conventional tumor reduction modalities involve stress, pain, or toxicity which can rapidly deplete buffy coat (WBC) AA to scorbutic levels, and cause hyperglycemia which makes it difficult to restore WBC AA.

*Cervical Cancer/Dysplasia, AA and Folate.* Cervical dysplasia progressing to cancer results in disfiguring surgery, much other morbidity, death and high medical care costs. It was reported that the mean concentration of “vitamin C” in plasma was significantly lower in 46 cases with dysplasia than in 34 controls (0.36 vs 0.75 mg/dL,  $p < 0.0001$ ).<sup>45</sup> The authors suggested a clinical trial with “vitamin C”. From a separate university, Orr et al. reported significantly lowered levels of plasma folate, vitamin A and “vitamin C” in a study of 78 patients with untreated cancer of the uterine cervix and stated the possible value of these vitamins for prevention and treatment requires investigation.<sup>46</sup> Since all gynecologists read *American Journal of Obstetrics and Gynecology*, how could these findings be ignored? The unequivocal but false declarations by high placed medical “authorities” indicted by Pauling<sup>1</sup> that AA and other vitamins are without value in cancer, would seem to explain this inexcusable neglect. Cathcart routinely eliminates cervical dysplasia with AA, 10 g/d and 10 mg folate/d as do some other physicians known to us.

### Reproductive Anomalies, AA and Glycemia

Although fetal malformations occur in 60,000 births per year in the US and are strongly correlated with maternal glycemic level in early pregnancy, it has been shown that even diabetic mothers are spared this tragedy, as well as an associated fecundity defect, by strong glycemic control.<sup>47</sup> It was suggested that the hyperglycemia-associated

birth defects are actually due to low maternal and, hence, low fetal intracellular AA, all caused by maternal hyper-glycemia.<sup>48</sup> It is well known that mitosis requires high intracellular AA for the HMP shunt, to provide ribose.<sup>13</sup> Hyperglycemia always decreases intracellular AA by competitively inhibiting the insulin-mediated active transport of AA into cells. In non-diseased humans, the highest AA concentrations in all tissues occur in the fetus and decrease with human age (p.78).<sup>12</sup> Therefore, the newborn must avidly absorb AA from the mother both as a fetus during gestation and in postpartum nursing. It has long been known that women who do not supplement AA show a dangerous fall in plasma AA from about 1.0 mg/dL to ~0.35 mg/dL at parturition, levels associated with serious diseases or death in the newborn.<sup>49</sup> The predicted<sup>48</sup> effects of glycemic level and AA were strongly supported by experiments in an animal model.<sup>50</sup> Yet, for early pregnancy, glycemic control is not stressed and adequate supplementation of AA is condemned as a cause of miscarriages today by "authorities" (p.358).<sup>1</sup> Klenner reported that, in a series of 300 pregnancies, the gravidae were given AA, 4 g/d in the first trimester, 6 g/d in the second and 10 g/d in the third.<sup>20</sup> All the infants were robust and there were none of the "normal" complications of pregnancy frequently encountered when little or no AA is supplemented. Klenner was able by this AA method to carry women through successful pregnancies who had aborted all previous attempts under their obstetricians.

### Aging and AA

Glycation has been shown to be an important aging mechanism.<sup>51</sup> In experiments on glycemic modulation of tumor tolerance in mice given supplemental AA, a marked depression of glycation of hemoglobin (40%) was observed (Ely, unpublished). This dose dependent effect was

also found in the GHb (glycated hemoglobin) data of 600 humans on whom both blood chemistries and diet questionnaires including the amounts of supplemental AA habitually taken (at their election) were available. The implications of these findings regarding AA and aging are far reaching; they were announced at a meeting.<sup>52</sup>

### Morbidity, Refined Carbohydrate Intake and AA

The primitive (unrefined) diets on which most humans evolved contained no pure sugars and no other refined (or rapidly hydrolyzable) carbohydrates (rCHO), i.e. zero "empty calories." High sugar foods such as berries, sugar cane and honey, have high nutrient contents and were seasonal or rare. It is established that: (1) habitual consumption of rCHO raises both fasting and postprandial blood glucose levels in a dose dependent way; (2) elevated glucose competitively inhibits insulin-mediated active transport of AA into WBC against the 50:1 concentration gradient; (3) lower WBC AA causes the HMP shunt to slow<sup>13</sup> decreasing resistance to infections and cancer; and (4) on a diet with half the calories from sugar some normal humans suffer serious endocrinological derangement with cortisol elevated to three times normal.<sup>9</sup> An important study by Cheraskin and Ringsdorf puts all of these facts in perspective.<sup>7</sup> They cite USDA statistics that, in 1970, the average American consumed 264 pounds of empty calories. On a dry weight basis, this was just over 50% of food intake. Using the Cornell Medical Index Health Questionnaire and a sample of 715 well educated people, they created plots of rCHO intake vs. health problems. Extrapolations of these plots show that zero health problems occur at zero rCHO intake. Consider the implication for staggering increases in M&M and dollars that have been caused by rejection of the glycemic control (via the unrefined diet) modality.<sup>23,35</sup>



### Mortality and AA Intake

Analysis of death rates from six principal causes in England and Wales, and their correlations with 13 nutrients from the National Food Survey was reported by the University of Birmingham.<sup>53</sup> "Vitamin C" had the highest negative correlation (-0.63) with the sum of all causes and was negatively correlated with the individual diseases although the correlation became quite low, as is expected, in diseases where blood sugar is high (i.e. -0.19 in diabetes). How could US "authorities" dispute the value of AA in view of this publication in the third largest circulation journal in the world?

### The Common Cold and AA

Although most of the large scale clinical trials of AA and the common cold done in the 1960s showed some significant reduction in frequency and/or duration of colds, the results were disappointing to Pauling, to this author, and to a small number of others who are able to eliminate colds completely by one or more grams AA/day. These people are those who avoid sugar and have low values of plasma glucose (fasting and postprandial). This author during his first three decades had a high sugar intake and suffered several colds yearly. By chance, in his early thirties, he stopped eating sugar and (influenced by Klenner)<sup>18</sup> started taking 1 g AA/d. He had only two colds in the next two decades, each one after a rare sugar gluttony. In 1972 Pauling stated he had studiously avoided sugar for many years prior to taking AA at the suggestion of Irwin Stone in about 1966. If the US per capita sugar consumption had been zero instead of over 100 lbs. per year, the cold trials would have completely protected almost all of those given one gram or more of AA/d because of the resulting HMP stimulation at low blood sugar, as explained above. Additionally, all white blood cells are enabled to be effective against disease by the HMP shunt which runs at a rate proportional to the intracellular AA con-

centration.<sup>13,21</sup> The cold trials should be repeated using subjects who have not been taking AA; each subject would have glycated hemoglobin (GHb) measured by a finger stick and be assigned to the 5 quintiles according to GHb. Then, members of each of these quintiles are randomly assigned to the AA treatment or placebo control groups. It is predicted that when the trial is concluded in one or two years, the reduction in incidence of colds will be found to be essentially zero in the highest GHb quintile and essentially 100% in the lowest quintile.

### Infectious Diseases and AA

In addition to the common cold, other viral and numerous bacterial diseases can be treated successfully by AA orally. Although Klenner used AA (ascorbic acid orally, sodium ascorbate in IV) to treat measles, mumps, chicken pox, adenovirus, herpes, he felt that its greatest value in viral disease was against the polio virus.<sup>18,54</sup> All 60 polio patients in the 1948 epidemic recovered within 72 hours with no residuals.<sup>18</sup> For a period of 26 years, using the same regimen, all encephalitis patients recovered completely within 72 hrs.<sup>20</sup> Klenner states that in such crises, the minimum dose is 350 mg/kg body weight to be repeated every hour for 6 -12 hours depending on clinical improvement, then every 2-4 hours until the patient has recovered. Large doses of IV AA have a striking influence on mononucleosis.<sup>20</sup> Cathcart uses AA orally >150 g/d for this disease.<sup>15</sup> Cathcart also cures influenza and induces prompt remission in herpes infections (cold sores, genital lesions and shingles) with bowel tolerance AA.<sup>15</sup> Both Klenner's and Cathcart's acute infectious hepatitis patients are cured in a few days with massive doses of AA. Patients are well and back to work in 3-7 days<sup>20</sup> and lab tests including SGOT, SGPT and bilirubins rapidly normalize.<sup>15</sup> Baetgen used AA 10 g/d to cure hepatitis.<sup>55</sup> Morishige and Murata<sup>56</sup> have demonstrated

the effectiveness of AA in preventing hepatitis from blood transfusions. Although AA may not cure chronic hepatitis, 10 g AA/d plus other nutrients can indefinitely control disease, preventing liver necrosis and progression to hepatoma, transplant (Cathcart private communication).

Treatment of bacterial infections also benefit from AA. Klenner reported diphtheria, hemolytic streptococcus and staphylococcus infections clearing within hours following AA given IV with "a 20G needle as fast as the patient's cardiovascular system would allow" (500-700 mg/kg body weight).<sup>2</sup> When bacterial infections are treated with the appropriate antibiotic and AA, the effect is synergistic and patients respond rapidly, even if the bacterium is antibiotic resistant.<sup>15</sup>

Anyone who considers the use of AA can benefit from Cathcart's writings<sup>57</sup> which include much clinical wisdom, not the least of which is his explicitly stated regret that people think of AA as a vitamin (which we have seen it is not).

The HMP shunt rate has been shown to increase by up to 700% at plasma AA 140 mg/dL (200 times normal).<sup>13</sup> Such high levels are attainable in humans only by IV ascorbate (often called IVC) combined with continued oral intake. This protocol has been used by Cathcart in all infections including AIDS patients (brought under control but not cured by AA). He starts with often well over 50 g/d to bring the disease "under control" and tapering down to as low as 10 g/d as a maintenance dose in some cases.<sup>14</sup> Notice that the (very expensive) interferon enhances CMI but it has been reported for 20 years that AA very inexpensively doubles interferon production wherever virion replication is in progress.<sup>25,26</sup> Elevated interferon not only inhibits virion replication of an existing infection, but renders the host very resistant to infection by a second, possibly more deadly virus. As long suspected,<sup>14</sup> the results of HIV studies may be "polluted" because most AIDS pa-

tients have found the value of AA and use it unknown to their physicians (who they fear would forbid it, asserting it is without value).

In summary, it appears that most bacterial infections and all acute viral infections treated by Klenner and Cathcart can be rapidly put in remission by high AA. It seems likely that Ebola, hanta and other highly lethal virus infections might also be treatable and preventable by this modality. The intellectual paralysis induced by the incompetents identified by Pauling<sup>1</sup> makes the whole world suffer deadly epidemics, even polio, and deaths in virology labs. Since 1990 even AIDS is reported to be curable by the antigen specific Transfer Factor developed against a conserved antigen on HIV by H. H. Fudenberg and coworkers,<sup>58,59</sup> another ignored analog. But why rapidly cure one million HIV+ patients for \$100 each, if \$20 billion per year is being spent on their care?

In the US where refined diet prevails and most MDs are constrained to use the ineffective treatments of choice, the shocking annual toll from infections is nearly 200,000 premature deaths (over 2 million years of life lost before age 65), over 42 million hospital days and cost over \$17 billion.<sup>67</sup>

### Surgery and AA

Klenner (p.74)<sup>20</sup> cites references from 1937 onward,<sup>60,61</sup> that it was known that post operative AA was necessary for healing wound tensile strength, resistance to infection and elimination of most post-operative deaths. Klenner found that without post-operative AA, by 6 hours, the plasma AA fell 1/4; by 12 hours was down to 1/2; and at 24 hours was 3/4 lower than at surgery. Klenner encouraged patients to take oral AA 10 g/d for weeks prior to elective surgery and suggested surgeons use AA freely in fluids. His clinical wisdom is apparent in an example demonstrating the safety and efficacy of high AA doses in an "incurable" case: he had assisted on an abdominal exploratory surgery for an apparently scorbutic patient with numerous

intestinal adhesions of her friable tissue. After repairing 20 tears, the surgeon closed the cavity as hopeless. In post-operative care Klenner gave the patient 2 g AA every 2 hours for 2 days, and then 4 times/day; the patient was ambulatory in 36 hours, was discharged well in seven days and outlived the surgeon by many years. What is the annual cost in M&M and dollars of poorly healed and infected wounds for mainstream's failure to evaluate Klenner's findings that are known by orthomolecular physicians to be valid?

### Intoxications and AA

Intoxications from many causes are successfully treated with AA. Many thousands of needless deaths occur each year from the bites of snakes, spiders, flying insects and caterpillars. Klenner obtained rapid reversal of the swelling, pain, breathing difficulties and shock, resulting from such "bites" using IVC (350 to 710 mg/kg body weight) and AA orally as followup.<sup>20</sup> The release of histamine, which is a major shock-producing substance, is minimized by AA. Similarly, Klenner normalized the shock in patients with barbiturate poisoning using 12-75 g IVC.<sup>20</sup>

The value of AA for chemical intoxications is also emphasized by Stone.<sup>10</sup> AA protects against the effects of poisonous metals (e.g., mercury, lead, arsenic), organic chemicals including bacterial and plant toxins, as well as addictive drugs. In a pilot study of 30 heroin addicts given 25 to 85 g sodium ascorbate/day (along with high doses of multivitamins, essential minerals and protein), the appetite returned, restful sleep was restored and mental alertness improved in 2 or 3 days.<sup>62</sup> Moreover, there were no withdrawal symptoms. After 4 to 6 days, the AA dosage can be reduced to maintenance levels. AA can also be a life-saving measure for drug overdosage. Why aren't these effective and inexpensive methods being studied and used?

### Other Modern Germ Theory "Analog"

*Coenzyme Q<sub>10</sub>: A Rejected Modality.* For background and striking results reported with CoQ<sub>10</sub> in aging, cancer, heart disease, and stroke read Physicians' Update.<sup>8</sup> In 1997, a survey could not find MDs (even cardiologists) at major US medical centers who were aware of CoQ<sub>10</sub> (all were content with statins and surgery) although this miraculous molecule had been used in Japan for 30 years. Another specific example of mainstream ignoring its own highest expert at a cost of possibly millions of lives and trillions of dollars is found in the 1990 warning by Karl Folkers. This frequently honored chemist who first determined the structure of CoQ<sub>10</sub> in 1958 and was Director of Research for Merck for 20 years, warned that heart disease is caused or worsened by the depression of CoQ<sub>10</sub> that is associated with statin use and that CoQ<sub>10</sub> should be supplemented in patients given statins.<sup>63</sup> A review of the literature on toxic effects of depressing CoQ<sub>10</sub> suggests, inter alia, increased susceptibility to stroke injury (a leading cause of M&M) since the only agent found to protect significantly in stroke in decades of work on three animal models and a few human strokes<sup>64</sup> is CoQ<sub>10</sub>. Internationally, as of 1995, there had been at least nine placebo controlled studies on the treatment of heart disease with CoQ<sub>10</sub>, a number were very large scale, the largest having 2,664 patients. All confirmed the remarkable safety and efficacy of CoQ<sub>10</sub>.<sup>8</sup> Recent studies claiming to show lack of efficacy of CoQ<sub>10</sub> appear flawed (low dose, short time, untreatable patients) in the light of world findings. Is this action designed to oppose acceptance of the low cost (unprofitable), non-toxic (endogenous), versatile CoQ<sub>10</sub> modality (which, with vitamin E, could replace statins, and much else), just as the Mayo cancer "trials" authorities doomed AA?

*Vitamin E: Another Rejected Modality.* Several of the many benefits of high E intake have been discussed above. For better accounts with many references of E's numerous values, as published in leading jour-

nals, from 1950 on, by world respected clinicians (Ochsner, Haeger), and its arbitrary rejection by the "authorities," see Pauling<sup>1</sup> and Shute.<sup>3</sup> It is surprising that E, second only to CoQ<sub>10</sub> among available antioxidants, and known for 50 years could be termed "worthless" by a nutritionist. It is well known that: (1) cholesterol is not a risk factor for CVD unless LDL is oxidized; and (2) this is simply prevented by E.<sup>65,66</sup> In the late 1940's, the Shutes were able to show striking beneficial effects in CVD and other patients by 300 IU E/d. They were also able to conclude that loss of E in food processing to produce the US refined diet was a most probable cause of the appearance and growth of CVD as the largest producer of M&M from 1916 on. These matters are known to every capable student of nutrition. Until recent CoQ<sub>10</sub> stroke findings, Shute's E therapies were the only or best available for CVD, stroke and rheumatic fever.

#### Note

PubMed abounds with reports of the beneficial effects of caloric restriction on a variety of diseases including cancer, the mechanism of which (the authors state) is unknown.<sup>68</sup> We have shown in humans and animals that in breast cancer<sup>23,24</sup> and birth defects<sup>47,50</sup> even modest glucose elevation competitively inhibits insulin-mediated active transport of AA into WBC, depressing HMP shunt, CMI and mitosis.

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#### Abbreviation List

AA= ascorbic acid, ascorbate  
 CMI= cell mediated immunity  
 CVD= cardiovascular disease  
 GHb= glycated hemoglobin  
 g/d= grams per day

HMP= hexose monophosphate  
 IV= intravenous  
 IVC= intravenous ascorbate  
 M&M= morbidity and mortality  
 mg%= mg/dL = mg/.1Liter  
 CoQ<sub>10</sub>= Coenzyme Q<sub>10</sub>  
 RBC= red blood cells or erythrocytes  
 rCHO= refined carbohydrate  
 WBC= white blood cells

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