Note to Patreon Patrons

I’m now clear that the Patreon interface is designed by engineers and will not be fixed in 2020. So if you read “per month” when I have specified “per chapter,” don’t worry. The way it will work is that you will be billed at the first of the month only if I release a version of the book the previous month. Since I am releasing new versions of the book with multiple new chapters each version, I have yet to release it twice in any given month. So you will be charged $1, $2 or $4 (plus tax as of July).

If you want to financially support this project more than $4, please feel free to make a one-time donation to paypal.me/swfowkes. Non-financial support can be contributed by (1) sharing the book with friends and family by email attachment or cloud-account download link (I’d be happy to send you mine), (2) blogging about the book on your favorite social-media page, (3) asking people to become patrons at www.patreon.com/swfowkes, or (4) offering editing and organizational feedback directly to me.

I have heard from one friend that Patreon trapped her in a project that she wanted out of. I see an un-patronize button that looks like opting out is an easy process. But if you opt out and have any difficulties, do not be worried; I can opt you out if the Patreon software fails. If this happens to you, send me a message through Patreon, or through email, and I’ll fix it.

The first release had four chapters, and the second had seven, and the third a dozen, so the “practical” core of the book is now in place. Vitamins D, A and C, the minerals selenium and zinc, and hypothyroidism/hypometabolism are the pillars of natural anti-viral self defense. It was my wish with Patreon to publish information as fast as I can, with the sole limitation that the content be intrinsically readable, understandable and practical. So from that limited perspective, the book has been written. But I am now dealing with supplementary materials that are the foundation of the practical side. This includes presentation of lesser-known aspects of immune therapeutics, the appendices, adding new, updated content to old chapters, and fixing bad grammar.

The “deliverable” each time is a PDF book with all completed chapters included. This means that you get a complete new book with all the information from the earlier books included. So, no assembly required.

To help you see the new content, the new text color will be colored. In version five, it was purple. In this version it is blue. So you can scan for the new content fairly easily. In future updates, the oldest colored content will revert to black. Plus, I post a black-only version for printing, after posting the color version.

The tier-$2 and tier-$4 patrons are getting a monthly Zoom session. We have already done four of these, and because of popular consent, the second and fourth were recorded and posted as a bonus item. I am happy to address questions about sections of the book that are NOT finished. Towards this end, I am building an email group of patrons so that I can send you advanced notice of the Zoom sessions in a timely manner. To best stay in touch with me, and me with you, please put “Zoom Patreon” at the beginning of the subject line so I know to add, change or delete your email status as you wish.

My email addresses are steve@projectwellbeing.com and swfowkes@gmail.com.

Please feel free to let me know your suggestions, edits, typos, and less-than-ideal explanations.

Thank you for your patronage!

——Steven Wm. Fowkes, 7 July 2020, Cupertino, California, USA

PS: Feel free to distribute this book to your friends, family and loved ones.
Note to Everybody Receiving this Book as a Gift

If you are reading this book and do not know about Patreon, it’s likely that somebody gave it to you or you downloaded it. This is a good thing. I want this information to spread far and wide. Patreon.com is the online platform where this book is being serially published. This is the fifth serial release at ten months into the project.

This information is an antidote to the vaccine-only scare tactics that the media, public health officials and governmental bureaucrats are using to impose nonsensical and unprecedentedly disastrous public-health policies on otherwise-free people in fear of their lives and loved one’s lives. Although the structure of the SARS-CoV-2 virus is structurally optimized for infecting humans, that’s not where the mortality risk lies. The average person contracts about 20 novel viruses every year, most of them quite infectious. The danger lies in the oxidative stress caused by Covid-19 disease. The people at great risk are those with low antioxidant reserves: the elderly, the infirm, those with chronic illness and those with low metabolism.

All those pre-existing health conditions that increase Covid-19 risk also cause low antioxidant reserves. Oxidative stress, antioxidant defense, redox-buffering and antioxidant reserve are explained in detail in these pages. It’s not just viruses that induce oxidative stress. Bacterial infection (sepsis) does, too.

The good news is that antioxidant reserves can be strengthened quite easily. This book will tell you how to do that. Almost all of the natural anti-viral substances covered here are sold over-the-counter as supplements. You do not actually need the government to help you. The power is in your hands.

The bad news is that 90-99% of hospitals will not strengthen your antioxidant reserves. In fact, they will do the opposite, by taking away your antioxidant supplements for the duration of your stay, or by hydrating you with a D5W IV drip, instead of saline or Ringer’s lactate.

This failure to treat Covid-19 infections with the right therapeutic strategy is why the death rate is so high in most hospitals. Elder-care facilities also fail to treat it properly. There are private-practice doctors who have been strengthening antioxidant reserves dating back 80 years! Some of these pioneer doctors have even published their amazing results. It’s all just being ignored by mainstream medicine, or attacked as if it were medical heresy, just like the Catholic Church did during the Renaissance Age in resisting the idea that the earth revolves around the sun.

Everybody knew that the Earth was the center of the universe. But they were all wrong.

Today, everybody knows that what the government and media are saying is the truth. It’s all about the deadliness of Covid-19 and nothing about oxidative stress. It’s not about hot spots, it’s just that other areas are lucky. Covid-19 kills perfectly healthy people. There is no need to consider oxidative stress and antioxidant defense in treatment. But they are also all wrong.

And you can know it now.
With this “heretical” knowledge, you will be empowered in ways far beyond knowing that the sun is the center of our solar system. The antioxidant defense system (and the redox-buffering system that is its foundation) can be easily (and almost effortlessly) strengthened by dietary supplements that are widely available in the over-the-counter market.

At this time, such decisions are still 90% your choice.

Those vested interests which are maneuvering to corner the market on future Corona-19 vaccines would like to shut down the availability of these dietary supplements. They already say that “vitamins make expensive urine,” despite the same being equally true of drugs in every respect. Their allies at Facebook have already started to censor dietary and medical advice from honest and knowledgeable doctors and biohackers (like me).

Sending this book to your friends and family is one way to circumvent such censorship.

It may be a sad thing from a bureaucrat’s or know-it-all’s point of view that they cannot compete “fairly” in an open and free market. But from my perspective, it is a saving grace. Fortunately, most of the censorship efforts by Google and Facebook are being effectively bypassed by alternative software platforms where censored content is being collected and made available. Citizen resistance to censorship may be feeble in the general population, but it is alive and well in a vocal minority.

The Internet may be wild, woolly and heavily fertilized with male bovine soil additive, but it may yet keep us alive, informed and free from misguided public-health policy. (Do not miss Appendix G on the Great Barrington Declaration, which is new for this release.)

It may not seem plausible to you that the primary risks of morbidity and mortality from Covid-19 can be categorized so simply: oxidative stress. But it really is that simple (jump to page 78).

The field of redox medicine (the biophysics foundation of medicine, see Appendix A) has all the science to prove this, even to those rare skeptics who know enough chemistry and physics to understand the arguments. But for those readers who do not consider themselves proverbial “rocket scientists,” I explain this in analogies that do not require you to learn to speak “science” or understand specialist language and acronyms.

I do use acronyms (and citations to the primary scientific literature) in this book to help defend the ideas presented here from scientific criticism. But, rest assured, I also explain them in readily understandable language somewhere in the book. And if you cannot find it, I am happy to have it presented as a question during the first-Thursday-of-the-month Zoom sessions (if you are a patron on Patreon), or the third Sunday of the month Zoom sessions (if you got the book another way). It is my opinion that the average high-school student can readily understand oxidation, reduction (the opposite of oxidation), antioxidant defense and redox buffering. I smile when you say, “Oh, I get it.”

If you have never heard of reduction and redox, do not worry. Your doctor may be in the same place.

If you have heard these terms before, but do not yet feel like you “get it,” this book is for you.

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1 I host a general, self-care Zoom meeting on the third Sunday of the month at noon California time, which accommodates people in Europe (late night, Sunday) and people in the Asia Pacific region (very early morning, Monday). This is the Zoom version of my Meetup.com/ProjectWellbeing gathering that was formerly in my den. The topics for each meeting are set by those who attend. Find the Zoom invitation at (1) Meetup.com or (2) the Project Wellbeing website, or (3) the Project Wellbeing email list (sign up), or (4) my Facebook feed. Assuming I’m not censored and my account squelched.
Underneath Oxidative Stress: Innate Immunity

While morbidity (disease and disability) and mortality (death) are the direct result of unmanaged oxidative stress, it is the viral infection that sets it all up. And this is where your innate immunity plays the pivotal role. Your innate immunity is your first line of defense against viruses.

The viral infection can take one of two courses, (1) mild and asymptomatic if your innate immune system effectively manages the infection in its earliest stages, or (2) debilitating and life-threatening if your innate immunity fails in those early stages, and your humoral (antibody-based, cytokine-based) immunity is forced to step in to “rescue” you. It is your humoral immune response, item (2), that causes the oxidative stress that can kill you with a cytokine storm. So item (1) has the first shot.

Because of this two-phase immune reaction, much of what you will read in these pages deals directly with the functionality of the innate immune system. The innate immune system is the first line of defense against all viruses, including SARS-CoV-2. The three most necessary and essential nutrients for strengthening your innate immune response are vitamins D and A, and the mineral zinc. Each of these has a chapter devoted to it.

This book will explain why these three are pivotal.

Support

If you come to value the information in this book, please consider supporting it in some way.

I’d love it if you would become a patron of the book at Patreon.com/swfowkes.

I’d love it if you’d share this book and information with somebody else. Maybe many somebodies.

I’d love it if you’d make a direct donation to my PayPal account (steve@projectwellbeing.com).

If you do not have a PayPal account, you can donate with a credit card through PayPal.me/swfowkes.

I’d love it if you’d consent to be interviewed about your Covid-19 and vitamin C experiences.

I’d love it if you would save your life and somebody else’s life by putting these options into practice.

And I’d love it if you would stop being afraid of Covid-19.

All of these many “pre-existing conditions” that are being exposed by the SARS-CoV-2 epidemic are treatable, all the way from simple nutritional deficiencies to the complex processes underlying aging. They

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2 Awareness of vitamin D and zinc is becoming mainstream in early-adopter circles despite official propaganda efforts. There is simply too much highly positive data being published about vitamin D, which is likely the most efficacious Covid-19 treatment in use today. And now that the scientific fraud campaign against hydroxychloroquine is exposed, there is a very strong, compelling clinical rationale for zinc, which hydroxychloroquine delivers through its remarkable zinc-ionophore activity.

3 Fear and anxiety may improve memory consolidation, but they both strongly suppress immune function. It is also true that anything that sabotages sleep quality has an immunosuppressive effect the next day. So worry and obsessive ideation are also on the best-not-to-do list. These are both classic side-effects of watching the evening news. Many people tell me that it is their duty to watch the news so they know what is going on. This is not true on several levels. First, “the news” only tells a tiny fraction of what is going on in the world, and it plays to salacious and gory items that invoke a stress response, to make the news addictive. So it is not honest. As it is said, “if it bleeds, it leads.” The news is also politically filtered by the cronies of media moguls. They want you to be upset about their special-interest issues. All of this does not make you well informed. Quite the contrary. It makes you biased, and susceptible to illness in the bargain.
may have been ignored by your doctor and tolerated up until now as “normal aging,” but this is merely a widely held belief supported by our cultural prejudices and contradicted by piles of scientific evidence. Beliefs may take time to change, but they can change. The first step in that change is cultivating a modicum of doubt. The second step is paying attention to all the evidence for specific anti-aging effects from improvements in lifestyle, habits, exercise, food, diet, supplements, water, air, thoughts, emotions, beliefs, and last but not least, the practice of medicine.

All the governmental and public-health policies that have led to this dangerous juncture can be reversed. And, hopefully, in the meantime, ignored or bypassed.

It’s your life. Live it your way.

——Steven Wm. Fowkes, 4 July 2020 in Cupertino, California, USA
(updated 24 August 2020 and 29 December 2020)

PS: Here’s the second verse of a wonderful lyric from Gypsy Soul⁴ that inspires me:

We all need a chance to prove that we were here, share our fears, shed some tears.
(Ooooh) And faithful friends who never need to ask the reasons why.
A chance to dream, a space to scream, and a time when we fly so high.
So when will you choose that you will live, until you die?
I’m calling in, I’m calling in, all the answers inside of me.
I’m calling in, I’m calling in, a life that sets me free.

PPS: The quotes on the previous page are from (1) Ronald Reagan, who said “The nine most terrifying words in the English language are, “I’m from the government and I’m here to help,” (12 August 1986) and (2) the character “V” from the movie V for Vendetta, which is an excellent translation of the graphic novel of the same name (1982-1989) by Alan Moore (writer) and David Lloyd (illustrator). I was originally interested in the movie because of my rumored ancestral connection to Guy Fawkes (the character archetype for the movie and the novel), but ended up enthralled by the acting skills of Hugo Weaving (The Matrix films, Lord of the Rings trilogy) acting the V character entirely from behind a mask (no Covid joke implied). If you have not seen it, look for it. It’s become quite ironic in a reality-imitating-art context for the current Covid-19 situation in light of the gain-of-function disclosures regarding Anthony Fauci and the NIH programs to manufacture more deadly coronaviruses for research purposes and ship them to China. So I have Dr. Fauci and V as archetypal opposites, both in masks—intentionally.

⁴ I’m Calling In, Gypsy Soul, here’s a link to a live performance: https://www.youtube.com/watch?v=YrVHUWDQfl8
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Author Declaration

This book does not contain medical advice. As the author, I am not a medical practitioner and am not licensed to practice medicine in any capacity whatsoever. So nobody should take any of this content as medical advice. This book only contains information. Scientific information. Medical information. Biohacking information. Practical information, yes. As such, it is intended for educational and entertainment purposes. The educational purposes are obvious to most, and especially to those who know me personally.

The entertainment purpose is for two groups of readers, (1) those with an appreciation for sarcasm, satire and irony (you know who you are), who either laugh or cry at the way human beings are being treated under the guise of public-health policy, and (2) those quackbusters (and public-health bureaucrats) out there, who have honored me—presumably inadvertently—by declaring my work to be quackery. Thank you, from the bottom of my heart. You do not know what it means to me to be a peer of those pioneers and practitioners that you have also honored by such praise.

I have spent four decades of my attention sifting information in search of a meta-level understanding of health, wellness and disease. I find gratification in acknowledgement and appreciation by others for my own quest for Truth,

Justice

and the . . .

...that special ah-ha moment when the next jigsaw puzzle piece drops into its big-picture spot.

“The American Way,” the deliberately omitted part of Superman’s catch phrase, is no longer based on truth or justice. Today’s American way is propaganda, hidden agendas, character assassination, public endangerment, regulatory intimidation, social ostracism, administrative threat and media pandering. It’s about superficial appearances rather than reality. There is no statesmanship in the legislative or administrative branches of the US government. Fiscal responsibility is zero. The health and welfare of US citizens is being purposefully undermined by an oligarchy seeking power and wealth from “disease management.”

Much of the content of this book will be in direct opposition to the goals and policies of that oligarchy. So it is my final declaration that this book’s content is fully protected by the First Amendment to the US Constitution and in no way represents me crying “Fire!” in a crowded theater.

——Steven Wm. Fowkes, 30 April, 2020 in Cupertino, California, USA

May 13th Postscript (the second serial release):

This book is being written online with serious time pressures. As a result, it may contain errors that need fixing for the next edition. I’ve already corrected several in the Allan Smith case where New Zealand doctors refused vitamin C therapy, then after grudgingly consenting, withdrew it abruptly after Allan began to recover rapidly, causing rebound scurvy and putting Allan, again, in mortal danger. Unlike Allan’s case, which was covered in the media (60 Minutes, New Zealand), this is happening all over the world with no mainstream media coverage. I have roughly 30-60 days to get this book into some semblance of useful completion for the transition from the lock-down quarantine mentality, where herd immunity is ignored, to re-engaging in social interactions and commerce, when increased infection rates will eventually reach herd immunity, and a steady-state between humans and SARS-CoV-2, like we have with influenza and natural
coronaviruses. It is important to me to have this information out for the summer months, during the anti-flu season, when we will be less susceptible and have the option to experience gentler coronaviral infections and a dramatically lower death rate. And in the fall, when the next flu season hits, more will be prepared for the next round of Covid-19.

**July 8th Postscript** (the third serial release):

This update to the book contains considerably more political content. However, I have separated the bulk of it into chapters that can be ignored by those with no taste for the ugliness (and beauty) that are essential aspects of such content. I feel this content is essential to the title of the book: self defense. On one side, you have the government, mainstream media and medical profession agencies actively promoting the idea that there is “no cure” for coronavirus, and enforcing it through censorship of those who dare speak of the cures, and police action against those that practice the cures. On the other side, you have me and hundreds of practicing physicians saying that there are cures, that they’ve been around for decades, and that the death rate from coronavirus can be dropped as close to zero as is deemed politically desirable.

That’s right. As close to zero as they want. And it works with people with pre-existing conditions.

**This may be the biggest challenge of this book.**

How do you reconcile the official position of public-health experts with the decades of clinical experience of many hundreds (thousands?) of doctors?

Who to trust? Governmental bureaucrats with no accountability? Bureaucrats who insist that double-blind studies are the only acceptable standard, yet sabotage every attempt to fund one?

Or independent doctors ethically accountable to each individual patient?

I know who I’d trust. No question.

**What about the cost?**

The at-home version costs less than $100 dollars per person. Total. This is for simple dietary supplements available online and via retail health food stores.

The in-hospital version? The cost is dramatically less than the costs currently being paid to treat Covid-19 patients ($13,000 to $40,000 per death). Maybe a tenth of the cost currently being paid. The cost of adding the oral supplements described in this book in a hospital setting, including oral vitamin C, is less than $100 per patient. The cost of the IV vitamin C is less than $1000 per patient. But these added costs are more than offset by (1) reduced amounts of medication, (2) dramatically decreased duration of hospital stay and (3) the hundred-fold drop in the clinical need for ventilation.⁵

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⁵ Since Covid-19 patients are placed on IV drip for optimizing hydration, the IV costs are already invested. All that is needed is to add sterile, pH buffered, electrolyte-balanced vitamin C solution to the IV bags that are already hanging next to their beds. This cost is currently many thousand dollars due to abrupt (20x) price increases in bottles of commercial IV-C, but would be $1000 at the pre-Covid prices that had been stable for more than a decade, and $100 if the hospitals prepared their own IV-C solution in house. But even at the full, FDA-inflated price, the average cost would be less than $10,000, dramatically less than the >$100K ICU costs associated with serious cases and extended stays.

⁶ Even at the greater-than-$500 per bottle of commercially prepared IV-C solution, costs would be less due to the high cost of hospitalization and the decreased duration of stay demonstrated by the studies cited in the book. The decrease is days compared to weeks. Maybe 4-9 days compared to 2-8 weeks. This is not only economically compelling; it undermines the public-health argument that there is not enough hospital capacity to handle the coronavirus epidemic. The Covid-19 Critical Care Working Group has reported that their medium-dose IV vitamin C protocol (MATH+) has the potential to “reduce ICU
And it’s all documented in the scientific literature. How, indeed, do you reconcile this?

**September 23rd Postscript** (the fourth serial release):

When I wrote above about the mantra being chanted by redox-medicine detractors, “Where are the randomized, placebo-controlled, cross-over trials?” in the preceding postscript, I had not yet cited the excellent meta-study of an unsubstantiated cure for “gravity challenge” that is being serially published in a variety of journals and websites (see page 228). The authors apply the same evidence-based criteria used to disqualify vitamin C and glutathione therapy for viral disease to this superficially effective cure. They write (in British English, not American English):

**Study Design:** Systematic review of randomised controlled trials.

**Data sources:** Medline, Web of Science, Embase, and the Cochrane Library databases; appropriate Internet sites and citation lists.

**Study selection:** Studies showing the effects of using a parachute during free fall.

**Main outcome measure:** Death or major trauma, defined as an injury severity score > 15.

**Results:** We were unable to identify any randomised controlled trials of parachute intervention.

**Conclusions:** As with many interventions intended to prevent ill health, the effectiveness of parachutes has not been subjected to rigorous evaluation by using randomised controlled trials. Advocates of evidence-based medicine have criticised the adoption of interventions evaluated by using only observational data. We think that everyone might benefit if the most radical protagonists of evidence-based medicine organised and participated in a double blind, randomised, placebo controlled, crossover trial of the parachute.

Now that you have stopped laughing, here’s an interesting example of the time frame between a generic nutrition discovery and its eventual study in humans on a double-blind basis. In 1975, the first paper was published that documented that sulfhydryl compounds (similar to glutathione) and reducing agents (redox-buffering substances) blocked the toxicity of acetaldehyde, the metabolite of alcohol that causes hangovers. In this study, redox-active combinations of vitamin C and reduced sulfur compounds decreased the toxicity of a LD-90 dose of acetaldehyde to LD-zero. In other words, a dose that would kill 90% of the rats killed none when vitamin C and the amino acid cysteine were on board.

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H Sprince, C M Parker, G G Smith and L J Gonzales. Protective action of ascorbic acid and sulfur compounds against acetaldehyde toxicity: Implications in alcoholism and smoking. *Agents and Actions* 5: 164-73, 1975. “Greatest protection against anesthesia and lethality was obtained at 2 mM/kg with each of the following: L-cysteine, N-acetyl-L-cysteine, thiamin HCl, sodium metabisulfite, and L-cysteic acid. A combination of L-ascorbic acid with L-cysteine, and thiamin·HCl at reduced dose levels (2.0, 1.0 and 0.3 mM/kg, respectively) gave virtually complete protection.”

Acetaldehyde is the toxic metabolite of alcohol. In the body, alcohol ingestion is followed by its metabolism. First, alcohol dehydrogenase oxidizes the alcohol to acetaldehyde, providing the body with a molecule of NADH. Then acetaldehyde dehydrogenase oxidizes the acetaldehyde to acetic acid, generating another molecule of NADH. NADH acts as either an antioxidant (a good thing) or as a fuel (also a good thing), which is why alcohol warms people up after getting chilled and why alcoholics feel “normal” for the first 30 minutes after drinking alcohol. But the temporary presence of acetaldehyde is problematic. Acetaldehyde (and its close chemical relative formaldehyde) acts as a cross-linking agent and temporarily reduces the activity of glutathione. This acetaldehyde toxicity is largely responsible for the “hangover” symptoms from...

Now fast forward 45 years. Finally, the first double-blind study in humans. In Finland.

Their methods: “Voluntary healthy participants were recruited through advertisements. Volunteers had to have experience of hangover and/or headache. The hangover study was randomized, double-blind and placebo-controlled. Nineteen males randomly swallowed placebo and L-cysteine tablets. The alcohol dose was 1.5 g/kg, which was consumed during 3 h.”

For all of us metric impaired folk, that’s about 8 ounces of 80-proof vodka.

Their results: “The primary results based on correlational analysis showed that L-cysteine prevents or alleviates hangover, nausea, headache, stress and anxiety. For hangover, nausea and headache, the results were apparent with the L-cysteine dose of 1200 mg and for stress and anxiety already with the dose of 600 mg.” This was a preprint, so maybe they will edit out “already” before it is officially published.

For everybody wanting to test this out for themselves, the Alcohol Detox formulation designed by me and manufactured and distributed by Nutrition Dynamics is available for roughly $10 per bottle of 60 capsules. My protocol is one capsule per drink, plus one capsule at the end of drinking with a full glass of water. Some people need two capsules for each dose for best results. I gave the formulation to a Texas physician, who passed it on the NutyriDyn, so I do not get any royalties from sales. Therefore, my only conflict of interest is a bit of bragging.

For the above study of 8 ounces of vodka, as eight shots over three hours, that would be nine capsules of Alcohol detox at a cost of less than $2 for replicating the experiment with my dosing recommendations of nine capsules, and $5 for replicating the experiment at the 600 mg dose used in the study (three capsules, nine times, 27 capsules total).

If you are not a teenager exercising your new legal rights to an alcohol-based rite of passage, the dose of alcohol would be smaller and the cost significantly less.

The public-health consequences of ignoring this finding have been immense. Alcoholism, drunk driving, sleep deprivation, spousal abuse, traumatized children, codependency, diabetes, heart disease and neurodegenerative diseases; these are all costs that we all pay for such bureaucratic neglect.

**January 2021 Postscript** (the fifth serial release):

The next flu season is here and Covid-19 rates are going through the roof again. And hospital staff throughout the USA are still ignoring the vast majority of the scientific and medical literature cited here.

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Not only that, but Google has declared war on science in policy. The badly considered evidence for lockdowns is now falling apart, but Google has decided that nobody should hear the evidence and is now censoring pre-eminent scientists who have clear evidence on their side.

Those three scientists are Martin Kulldorff from Harvard Medical School, Sunetra Gupta from Oxford University and Jay Bhattacharya from Stanford University Medical School. They have co-authored a document, the Great Barrington Declaration, and launched a website that “declares” that the lockdowns are bad public policy, causing unnecessary deaths and unparalleled economic and psychological harm to the world’s human population.

This all happened a month after the fourth release of this book on Patreon. And already, the forces of censorship have declared war. Google will not allow any links to the declaration, and is actively promoting links to critical responses to the declaration. Despite such efforts, thousands of scientists, physicians and medical personnel have co-signed the document. As of the writing of this paragraph, the numbers are:

- 13,000 medical and public health scientists.
- 40,000 medical practitioners
- 727,000 concerned citizens

A limited campaign by some reporters and Internet personalities to add fake signatures has been launched. These fake signatures have then been used by these and other reporters to criticize the declaration.

It’d be National Lampoon comical if it wasn’t so serious.

I’ve added Appendix G (page 234) to contain this and related content.

For example, the initial death rate from Covid-19 has been reported at 3% and 3.4% by official sources and 2-3% in more responsible citations. But the true death rate is 0.2-0.3%. The lower number is more accurate.

The explanation of how an order-of-magnitude error was made can be found in Appendix G.

This makes Covid-19 just twice as deadly as typical Asian influenzas.

It is my best estimate that use of the information in this book can drop the Covid-19 mortality from 0.2% to 0.02% (a factor of ten) if used simplistically (by a single protocol for every patient) and to 0.002% (another factor of ten) if used in a state-of-the-art protocol (optimized for each patient at every step of the infection).

I am not intending that as hyperbole. Drop death by a factor of ten or a hundred? Yes.

And reduce severity across the board. Get people out of hospitals in half the time. Cut ICU admissions by 99%.

And to address one more lingering concern, reduce long-term symptoms of all kinds, including neurological manifestations as well.

Definitely good reasons to read further.
Introduction

This book offers a biologically based approach to surviving acute viral infections.

The predecessor book (The BHT Book, now in its seventh edition) is oriented towards managing chronic viral outbreaks, first with BHT and then with nutrition and metabolism. This book focusses on acute viral disease and natural modes of prevention and treatment.

This book contains information that is not taught in medical school. So your doctor may not know anything about it, or only know parts of it. Furthermore, some doctors may not tell you about it because it deviates from the medical “standard of care” that doctors are legally mandated to follow under administrative law. The standard-of-care doctrine, however nonsensical, provides regulatory liability (discipline and loss of license) regarding making recommendations that are not “professional.”

This book also contains information that your government, public-health officials and TV newscasters will not tell you, despite some of the most important parts being well documented in the scientific literature. For example, high-dose intravenous vitamin C infusions reducing coronavirus mortality many-fold. This is the hardest thing for most people to understand: there is deep politics in public-health policy, there are huge pharmaceutical-industry profits at stake, and there are academic and professional egos fully invested in their “expert opinion” that what I am telling you is pseudoscience. They are all out to protect you from yourself, you from me, and me from myself. The old line, “If it worked, we’d know about it,” is the lie that is told generation after generation. History is replete with examples of state-of-the-art therapies and practices that were actively suppressed, and some brutally suppressed, until they quietly became the standard of care ten, twenty or forty years later.

Administrative law is the regulatory counterpart to criminal law. All kinds of citizen rights are established for criminal law that do not apply to administrative law. Under criminal law, you have a right to a “fair trial” and to face your accusers. Not so in administrative law. Under criminal law, you are considered innocent until proven guilty, also not true under administrative law. Under criminal law, you have the right to present evidence in your defense. Also not true under administrative law. So a doctor who deviates from the standard of care does not have the right to present evidence that his or her patients have benefited from that deviation. The administrative court might choose to listen to such evidence, but if they do not, you and your doctor have no legal recourse.

Fortunately, “peers” can be a tiny subset of the profession. Because of this, medical societies with special interests in functional medicine, for example, can recommend things that mainstream doctors would not support. There are medical societies built up around orthomolecular medicine, environmental medicine, chelation, dental toxicology, and more.

Consider the case of Dr. Ignaz Semmelweis, who advocated “handwashing after dissecting cadavers” to prevent infection in childbirth, and who was hounded to his death by the medical experts of the time. That was only 150 years ago. Intravenous vitamin C is a modern equivalent. And before you get too upset about this aberrant behavior, please consider that it is not aberrant. It is a tried-and-true way that human societies maintain internal cohesiveness—social ostracism for “unacceptable behaviors.” In the past, it tended to operate on the small scale, families, tribes, church groups, neighborhoods, etc., but it has expanded as our social organizations have increased in size to encompass organizations, professions, governments, races and nation states. It is my hope that this is not a depressing thought, but rather a simple understanding or recognition of how people are “wired.” Many people who “bump up” against this is real life may like to believe that this is how “others” behave. But I believe that we all do this in subtle ways, so pointing fingers is “the pot calling the kettle black.” And just as Semmelweis was vindicated years after his death, vitamin C will soon become a front-line treatment for severe viruses now that (1) it’s been 25 years since Linus Pauling died, and (2) many of his detractors have also passed in the intervening years. It is my hope that China will drive this change in 2020 and 2021, and possibly New Zealand, which has legally mandated access to intravenous vitamin C therapy.

A modern example is homocysteine testing, which was championed by Kilmer McCully, a Harvard-educated medical doctor who was Chief of Pathology and Laboratory Medicine Services for the United States Department of Veterans Affairs Medical Center, and which almost ended his career. Dr. McCully challenged the conventional view of the causes of heart disease. The
Some of what you read here is science done 90-125 years ago! And published 80 years ago! An entire appendix is devoted to this “suppressed” research (see Appendix A: The Biophysics of Life, page 153).

But do not let this institutional malfeasance depress you. This is the way the system is supposed to work. In “the age of bureaucracy” (our current phase), special interests get anti-competitive laws passed to protect themselves from innovation from within and without. And if something unexpected pops up, they immediately seek a new law to shut it down, all in the name of “the public good,” or course. This is how allopathic practitioners got rid of homeopathic practitioners 100 years ago. They commissioned an “impartial” study, The Flexner Report, conducted by their own people, which became policy for “protecting” the US public from “those dangerous homeopath.” And chiropractors. And naturopaths. And midwives.

And traditional Chinese and Ayurvedic practitioners.

Just because it’s always been this way is no reason for you to necessarily believe the Flexner Report’s blatant bigotry, overt misogyny and AMA bias. You can make up your own mind.

You can choose to act on new information regardless of the opinions of others.

For any new idea that emerges into public consciousness, there are the early adopters, mainstream adopters, late adopters and the not-in-my-lifetime non-adopters. It is wise to know which you are.

There may be significant cognitive dissonance (conflict of beliefs) if you are not an early adopter. About a sixth of the population are open to adopting a new technology when the mainstream messages are still against it. So if you have read online messages that this information is quackery, fraud, unproven and even dangerous, then your doubts may be strong. Late adopters would be unlikely to pick up this book at all.

Whatever doubts you may have, my advice is to indulge them—to not try to suppress them. Doubts are a good thing. I cultivate them regularly. Even for things considered 100% fact. And this has served me well. It may seem strange, but doubts are a necessary and essential aspect of the search for truth—although frequently ignored by many. The same thing applies to imagination being a necessary and essential aspect of science—also frequently ignored. So be true to yourself regarding your doubts. There is an emotional attachment to ideas that separates belief from information. Beliefs have momentum and change in their own time frame. Information in the absence of belief can change in seconds. If you experience cognitive dissonance when reading, just realize that an open mind only means that you consider a new perspective.

Regarding coronavirus specifically, the high concept presented here is redox defense.14 Those who have a strong redox-buffering system are much more likely to survive acute viral disease, and those with compromised redox-buffering capacity need serious medical support of their redox defense systems to have the same survival opportunities. Such support is rare in mainstream medicine. But it is available on a self-care basis. And the details of how and why are contained herein.

The perspectives in this book may be heretical in the institutional environments of the FDA, public-health agencies, academic institutions and the media. But if your mind is open, please read on.

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14 Redox is a contraction for OXidation and REDuction—but reversed for easy pronunciation. This will be discussed at great length later in the book. Redox potential is the primary biophysical foundation of the immune system. Redox potential determines the “set point” for inflammatory responses, which when dysfunctional results in cytokine storms. More on that later, too.
The information in this book is not rocket science. It’s nutrition. It’s naturopathy. It’s metabolism. It’s basic science applied to biology without political or institutional conflicts of interest.

Unlike this book’s predecessor, *The BHT Book*, this book will devote 99.44% of its content to the natural side of antiviral practices. This approach has as much or as little to do with drugs and the pharmaceutical industry as you wish. It is not dependent on drugs nor incompatible with drugs.

**High Citizenship versus Treason**

In the movie *National Treasure*, Benjamin Franklin Gates (played by Nicolas Cage) makes a toast at a formal gala hosted by the National Archives Museum:

> “To high treason. That’s what these men were committing when they signed the Declaration. Had we lost the war, they would have been hanged, beheaded, drawn and quartered, and—Oh! Oh, my personal favorite—and had their entrails cut out and burned!”

It is my opinion that the doctors, caregivers and authors who have kept redox-medicine alive for the past 80 years have demonstrated high citizenship rather than treason, although the message from public-health, academic, scientific and medical authorities is otherwise. The reason that I am on the same side of the fence as the doctors, caregivers and authors is because I have studied the arguments from both sides and come to the inescapable conclusion that the authorities are blowing smoke. I cannot say what they are smoking for sure, but their arguments are specious. They may sound scientific because of big words, and because they say them with a straight face while wearing a white lab coat with a stethoscope draped around their neck. But their carefully cultivated appearance of authority is merely that: appearance. There is no meat on their arguments.

It’s all rhinoplasty.

During a war, it’s not hard to see propaganda. Citizens who have some degree of perspective can easily see how the messages are slanted towards our national interest and away from the enemy’s interests. The pandering goes native with “Japs” and “Krauts,” and “Slant-eyes” and “Gooks,” but the public message is that we are “good,” and they are “bad.” The same thing happens in religious and racial conflicts. But when the same thing happens from purely economic motives, it’s much more difficult to see.

The “prudent diet” promoted by US public-health agencies for 60 years was pure pandering to the US food industry, and it was believed wholeheartedly (pun intended) by over 90% of the public and over 90% of physicians. Yet it has been falsified by the massive increase in cancer it caused.

Rather than give example after example of the above, let me cut to the chase.

The same kind of propaganda campaign used to slander and libel saturated fat has been used to prevent vitamin C from being an acceptable therapy. Propaganda masquerading as science. The scientists, theologians and philosophers of foregone ages did the same thing in defense of the flat-earth and earth-is-the-center-of-the-universe myths. And in the end, truth will out.\(^1\) It may take 20 or 40 years, or a century, but the truth will out.

\(^1\) Shakespeare, *The Merchant of Venice*. 1596. Launcelot: “Nay, indeed, if you had your eyes, you might fail of the knowing me: it is a wise father that knows his own child. Well, old man, I will tell you news of your son: give me your blessing: truth will come to light; murder cannot be hid long; a man’s son may, but at length truth will out.”
It is my hope that the Covid-19 episode will make it happen in five years.

Vitamin C can save your life. I know it. The doctors, caregivers and vitamin-C authors know it. And you have the opportunity to know it, too.

I’m not saying that vitamin C is the only natural anti-viral self-defense nutrient. It’s not. As you will see, \textit{But it is the collapse of the redox-buffering system that actually causes people to die.} And the weakest link in the redox-buffering chain is vitamin C.

I’ll say it again; the weakest link in the redox-buffering chain is vitamin C. See pages 78 and 79.

And the collapse of the redox-buffering system \textit{is what actually causes people to die.}

\textbf{Steve’s Short Anti-Viral Self-Defense Checklist}

1. Make sure your vitamin D₃ level is robust. This needs to be sustained. Get tested so you know.
2. Take vitamin A. And have extra vitamin A in your refrigerator to take when you catch a virus.
3. Take a selenium supplement. Organic \textit{and} inorganic. Nudge it with HIV, and push it with Ebola.
4. Cultivate a strong metabolism. Do not ignore your cold hands and feet, or low pulse rate.
5. Stop eating dairy products when viruses are nearby. Keep your sinuses and lungs unencumbered.
9. Take nattokinase for Covid-19 to keep your blood flowing and prevent clotting and fibrotic scarring.\textsuperscript{17}
10. Take vitamin C. If you catch a nasty virus, take a lot of vitamin C.
11. Take extra zinc at the first sign of a viral infection, especially with SARS-CoV-2.\textsuperscript{18}

Each of these items above has a back story below. Those items above that you already understand and practice should not be your focus. Rather, jump to the explanations for those items that seem bizarre or counterintuitive. Find out why selenium is so critical to viral virulence. Find out what I mean when I talk about metabolic balance.

\textsuperscript{16} There are plenty of trade-offs for organic food that bring this into question. However, one thing that is blatantly different between organic and non-organic produce is the glyphosate levels. It has become a popular practice among farmers in many countries to spray glyphosate (Roundup) on non-GMO crops, to facilitate desiccating (drying) them. This has caused widespread glyphosate toxicity for consumers eating a broad range of common foods. It is illegal to spray Roundup on organic crops to help desiccate them for sale. Although storage and shipping mistakes are made where glyphosate ends up in organic foods, this is relatively rare in the USA and Canada. So organic becomes a clear “best choice.”

\textsuperscript{17} This is a new (August) entry to the Short Anti-Viral Checklist. In versions 1-3 of this book, it was “Cultivate biofeedback. Focus on the quality of your sleep and your cognitive prowess during the day.” This change was made because Covid-19 has a notable effect on coagulation that is atypical and requires active intervention above and beyond that needed for influenza. If nattokinase is not well tolerated, try lumbrokinase. It’s more expensive, but might be therapeutically superior.

\textsuperscript{18} This January, 2021 entry breaks the top-ten theme for good reason. Zinc is not only being used as a monotherapy for Covid-19 in doses 10-20 times greater than the RDA, it is one of the key nutritional factors for innate immunity. Zinc is also sequestered (stored, reducing bioavailability) during inflammation. Not only do we live in “the age of inflammation” but Covid-19 routinely induces powerful inflammation when innate immunity falters. This puts zinc in the crosshairs.
Steve’s Compact, Personal-Action, Executive Summary

If you do not need the details, this chapter is for you. This list of action items is my response to a question, “What would you do for Coronavirus” posted on LinkedIn, based on (1) a strategy of prevention, prior to an infection, and (2) a strategy of therapy, during a viral infection:

**Vitamin D**
- **prevention**: raise vitamin D levels to 30-40 ng/ml, better to 40-50 ng/ml and best to 50-56 ng/ml.
- **during**: take extra D to promote cathelicidin and autophagy.
  - refuse D5W (and all refined carbohydrate foods for the duration).

**Vitamin A**
- **prevention**: vitamin A, 5-7K IU if female and capable of pregnancy, 10-15K IU if male, with no supplemental carotene.
- **during**: take high-dose vitamin A, as if treating measles in children, 50,000-100,000 IU per day for the first week.

**Selenium**
- **prevention**: selenium, 100 mcg selenite daily, and 200 mcg selenomethionine or selenocysteine weekly.
- **during**: double the dose for the duration.
  - for Ebola: no selenomethionine, triple the selenocysteine, and take 50-100 mcg selenite every 2-4 hours while awake.

**Vitamin C**
- **prevention**: vitamin C roughly 20x-50x RDA, 1-4 grams orally.
  - split into 2-4 doses (500 mg 2x daily, or 1 gram 4x daily).
- **during**: push vitamin C in proportion to the severity of infection, dose on a bowel-tolerance protocol, and add 1 gram liposomal C 2-6x daily for the duration.
- **during**: if oral is insufficient, medium-dose IV C: 6 grams 4x daily.
- **during**: if medium-dose IV-C is insufficient, high-dose IV-C: 50 grams 2x daily, and if necessary, 4x daily.

**Copper and zinc**
- **prevention**: copper- and zinc-rich foods (liver, shellfish, desiccated organ-meat capsules).
- **during**: lozenge zinc, nebulized zinc, zinc mouth spray, timed-release oral zinc.
- **during**: transdermal copper applied to thin-skinned areas on extremities.
  - (apply transdermal copper on skin over veins for IV needle sticks).

**Potassium**
- **prevention**: evaluate electrolytes and K utilization (serum-K to RBC-K normative ratio).
- **during**: if coronavirus, monitor serum K and/or urine K losses.

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19 The zinc administration may be more effective with a zinc ionophore like hydroxychloroquine or quercetin on board. This is fairly clear from the research published so far and makes sense to me as optimization strategy. My dose would be one tablet of hydroxychloroquine per week until the infection breaks.
Diet
prevention: practice dietary intermittency, flirt with ketosis, use intermittent and partial fasting.
during: never accept IV D5W (glucose), processed-carb foods, sugar, fruit juices, and cultivate high beta-oxidation and mild ketosis; then when positive, consider autophagy for 1.5-2.5 days with zero-protein diet, plus 10-50K IU/day vitamin D3.

Coagulation and clotting
prevention: assess coagulation tendencies, sed rate, etc.
during: take nattokinase at home, 2000 FU 4x daily on empty stomach, ask for low-dose, low-molecular-weight heparin if hospitalized.

And to this list I’ll add the following:

B-Complex vitamins
prevention: roughly 2-10x RDA
during: 10x RDA, especially thiamine.

Mitochondrial nutrients
prevention: intermittent carb-restriction diets, periodic protein fasting.
during: high-dose mitochondrial nutrients until urine tests positive for ketones. then low-dose for the duration. ALC, lipoic acid, B1, B2, B3, coQ10, PQQ, etc.

Multi-mineral supplement
prevention: RDA 1x daily.
during: RDA 2x daily for the duration, or as guided by cellular electrolyte/mineral testing.
during: B-complex vitamins, 10x for the duration (mentioned above).

Butylated hydroxytoluene (BHT)
prevention: none
during: 250-1000 mg transdermally if central-nervous-system infection is suspected.265

Hemochromatosis (clinical or subclinical)
prevention: regular blood donations. discard if blood is not acceptable for donation.
if liver pathology is involved, aggressive bloodletting, 1 cup (250 ml) per week.
during: if diagnosed in hospital after admission, start aggressive bloodletting after recovery is underway, as ferritin declines and TIBC falls.

Underneath the Checklist; The Anti-Viral Details
Each of the anti-viral items above has a back story below. However, the order of these summaries is not necessarily their order of importance. To know how important they are, you have to know your current health status. This is not often obvious. Selenium is much more important as an issue and as supplement in somebody who has a selenium deficiency. Metabolic rate is much more important in the elderly, those chronically ill, and those with cold hands and feet. Being alkaline dominant is much more important in infants and children, in women who experience severe PMS symptoms or are on birth control pills, in those who feel “brain fog” after eating, and in people with autoimmune and chronic-fatigue conditions.

In other words, it’s personal. This is as much a voyage of self-discovery as it is a review of potential treatments. Know thyself. It’s good advice in whatever way you can implement it.
Functional Medicine versus Vaccination

The number one political agenda being advanced by the public-health bureaucrats is that there is no treatment or cure for Covid-19 and that vaccination is the only option remaining.

This is, of course, totally wrong.

There are many highly effective treatments for viral diseases and Covid-19. The literature is replete with publications documenting them; viral treatments going back 80 years, and Covid-19 treatments going back ten months.

But the vaccine-only message is a “big lie” that’s been sold too many times for it to be dismissed out of hand. It’s been sold20 to politicians. It’s been sold to reporters and editors. It’s been sold to Bill Gates (and by Bill Gates). It’s been sold to Google and Facebook. And maybe it’s been sold to you. A billion people believe it is the truth, the whole truth, and nothing but the truth. Most of my family members believe it.

But the saddest thing is that most practitioners believe it.

Why? Because they simply do not know the fundamentals of their profession. That’s right, they are ignorant of the subtle mechanics of health. They are trained in symptoms, not mechanisms.21 They are trained in diagnosis (the pattern of “things going wrong”), not how to cure (helping to “make things right”). They do not know enough about nutrition, biochemistry, metabolism, pH, redox potential, the antioxidant defense system, the redox-buffering system, immunity, innate immunity, ketosis, autophagy, vitamin C and vitamin D to (1) make well informed therapeutic decisions, or (2) cite alternatives to vaccination.

You might expect that public-health officials would know these things, too. But you’d be disappointed.

So how is it that scientists know the mechanisms of innate immunity and physicians and public-health bureaucrats do not? If you follow that question to its many answers, you might be appalled. It is not a pretty picture. The treatment of disease on a functional level undermines profits and political influences that are deeply embedded in modern medicine and medical regulation. The way to rake in the cash is to treat symptoms and leave the mechanisms of disease operating. So that is what is done by people not knowing any better.

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20 I use the word sold rather than told because there is a huge difference. The word sold clearly implies that you have paid for the information, whereas told does not. And “paid” is definitely the case. Anybody who listens to something pays a tiny price of their time. But anybody who believes what they are told pays a substantial price by that belief. The spin doctors and media moguls know that fear breeds belief. So they exploit it. But that is not make it merely a matter of spin doctoring. It is hard wired into the subconscious mind that “danger” imprints memory. Danger means that what is going on needs to be remembered. This is fundamentally biological. It is a survival mechanism to prevent us from doing that “dangerous thing” or the behavior that put us in that “dangerous situation” ever again. So the word “sold” really tells the story with the most precision. And it tells the truth about the price that you must pay, again, to give up your belief in something false to fact. Beliefs are expensive to form and expensive to change.

21 There is a huge exception: emergency medicine. Those physicians and nurse practitioners are trained in mechanisms, and quite thoroughly. Nevertheless, they are trained in gross mechanisms, like breathing, blood flow, clotting, trauma, inflammation, anaphylaxis, resuscitation, blood pressure, reperfusion, oxygenation, hypercapnia, autonomic dysregulation, and much more. These are life-and-death mechanisms, but not in the subtle things that underlie chronic diseases. Get in a car wreck, break your leg, smash your head, these guys and gals are the go-to people. Similar things can be said about other medical specialties during acute crises. But, for example, putting in a stent before or after a heart attack does nothing to slow down or halt the pathologies leading up to the heart attack.
On one side you have vaccination for SARS-CoV-2, a potential multi-trillion-dollar industry, selling high-profit-margin vaccines that have to be administered year after year for the foreseeable future.

On the other side, you have only a few-billion-dollar industry treating the “pre-existing conditions” that promote Covid-19 morbidity and mortality, which once treated, drop medical costs and short-term deaths by a factor of ten.

Let’s get practical. Imagine this. Everybody is eagerly anticipating access to Covid-19 vaccines while an equivalent treatment is sitting on the store shelves waiting to be purchased. Both promise to provide 90%+ protection, one is something you can buy on Amazon or eBay and the other you cannot buy it anywhere.

- The one you can buy is known to be safe. The vaccine is not—yet.
- The one you can buy drastically decreases Covid-19 risks at all levels of severity, and the one you cannot buy has no such assurances—yet.
- The one you buy prevents and treats all viral strains. The vaccine only prevents a few strains.
- The one you can buy produces no side effects.22 The vaccine produces a wide range of side effects.

Is it possible that a situation so dysfunctional could be the case?

Yes.

Reading this book will give you the summaries and details of how to get the equivalent of the hoped-for vaccine without the unknown risks, or having to wait.

**How Well Does This All Work?**

The various treatments covered here have been very successful for the last 80 years in reducing viral disease severity across the board. For example, shutting down 60 of 60 cases of epidemic polio, with 100% prevention of paralysis. But for Covid-19, we do not yet know. The SARS-CoV-2 virus has been around only a year, and we only have a half-dozen clinical studies of direct therapy, all of which used only one single element of the following anti-viral options.

But two studies of vitamin D are jaw-dropping. The first one is a high-risk population of 87-year-old seniors in a French nursing home, where the quarterly (every three months) use of an 80,000 IU bolus (injection) dose of vitamin D dropped mortality by 67%, raised near-asymptomatic cases by 350% and increased the number of resident seniors not needing hospitalization or oxygen by 250%. And this with a dose that is roughly 1000 IU per day, a very small dose.

It takes roughly 5000 IU/day of supplemental vitamin D to raise the average person’s vitamin D level to 30-50 ng/ml, which is the sweet spot for viral risks, Covid-19 risks, stroke risks, cardiovascular risks and total mortality risks. And it usually takes a bit more vitamin D to raise the levels in seniors than in does in young and middle-aged individuals. So the dose in these French seniors was at best 20% of an ideal dose, yet it profoundly affected risks in one of the highest-risk subgroups of our population.

What would be a reasonable projection of the results were 5,000 IUs, 10,000 IUs or 50,000 IUs used to raise blood levels to 40-50 ng/ml or 50-56 ng/ml? With 5,000 IU/day, it might take half a year to get there.

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22 It is possible to experience sleep disruption from taking vitamin D late in the day. Vitamin D production is naturally tied to the circadian (daily) biorhythm, synchronizing with noon every day. Taking it as a supplement before noon or at noon keeps this rhythm operating naturally. So this side effect is simply a matter of disobeying label instruction and NOT a matter of any intrinsic side effect from vitamin D itself, even at doses of 50,000 or 100,000 IU per dose.
With 10,000 IU/day, it might take 2-3 months to get there. With 50,000 IU/day, it took 2-3 weeks to get there for non-seniors who started out with deficient vitamin D levels. That would be 100-125 or 125-150 nmol/L, respectively, using European vitamin D testing units. I think doubling the protection is a reasonable assumption. That would drop the death rate to 9% and the near-asymptomatic rate to 76%. That’s over 90% protection, which is comparable to that promised by vaccines. It is also in a notable high-risk population. And it’s only one of the antiviral options presented here.

This vaccine-equivalent protection is speculation, first from a study with only 66 seniors and highly unbalanced groups, second from an extrapolation from low-dose vitamin D to high-dose vitamin D. So it is only worth the quality of the speculations on which it is based. But there is a second study with much better-quality data.

The second study from which a natural anti-viral efficacy can be projected was a double-blind study of hospitalized Covid-19 patients in a Spanish hospital, also being treated with vitamin D. But this time, it was not just 1000 IU/day, a dose of only 10-20% of the ideal dose, but rather 30-60% of the ideal dose, which was 6000 IU/day for the first week and 3000 IU/day for subsequent weeks. During the study, 14 patients were admitted to the ICU for intensive care, only one of which was from the vitamin D group. Two patients died, neither of which were getting vitamin D.

Percentage wise, the vitamin D dropped the death rate from 7.7% to zero. Percentage wise, vitamin D dropped the need for ICU from 50% to 2%. So it is not only vaccines that might keep the hospitals from being crowded. Vitamin D can do it.

The pros and cons of these studies are reviewed in depth in the vitamin D chapter starting on page 26.

But the take-home message is that the efficacy of vitamin D is better than any other Covid-19 therapy.

The efficacy of natural anti-viral therapy involving other vitamins, minerals and nutrients can be added to the results from vitamin D alone. Vitamin A synergizes with vitamin D in its role in enhancing innate immunity (the immune system specializing in viruses). Vitamin A alone, in massive doses (800,000 IU) cuts the death rate of children hospitalized with severe measles by two thirds.

The mineral zinc synergizes with both vitamin D and vitamin A, and promotes health of the membranes of the lungs, sinuses, eyes, throat, and gastrointestinal and urogenital tracts. These mucous membranes are known to be targeted by SARS-CoV-2.

Low selenium levels predicts Covid-19 mortality in Germans (see page 62). It’s not yet clear that selenium is therapeutic during Covid-19, although it has been reported to minimize cytokine storms. In any case, being in the top quintile gives you half the cancer risk compared to the lowest quintile.

I hope this is enough context for you to read further.
Vitamin D3

Low vitamin D3 has a strong association with both acute and chronic viral disease.23 For SARS-CoV-2 infections specifically, vitamin D deficiency is associated with “increasing odds of death”24 and a higher likelihood of being admitted to the ICU (intensive care unit).25 Another study (illustrated at top of next page) found that those critically ill patients with the most severe Covid-19 symptoms (Group B) requiring admission to the ICU had dramatically lower vitamin D levels than those who presented as asymptomatic (Group A).26 There are well over a dozen studies showing that vitamin D status predicts Covid-19 severity, and that vitamin D supplementation profoundly decreases Covid-19 severity, at every level of severity.27

Vitamin D is the top contender for the heavyweight title for the most efficacious treatment for Covid-19 known to date. Nothing else is even close.

The authors of that last study (see illustration on next page) picked 20 ng/ml as the “deficiency” threshold. Of asymptomatic patients, 29 (32%) had deficient vitamin D levels and 62 (68%) had sufficient vitamin D levels. Compare that to the most symptomatic patients in Group B, where only two people (3%) were sufficient. The deaths show the same dichotomy, with one death versus 20 deaths.

The anti-Covid-19 heavyweight title: vitamin D.

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23 W B Grant, H Lahore, S L McDonnell, C A Baggerly, C B French, J L Aliano, H P Bhattoa. Vitamin D supplementation could prevent and treat influenza, coronavirus, and pneumonia infections. pre-publication manuscript, Nutrients March 2020. Abstract: Low vitamin D status in winter permits viral epidemics. During winter, people who do not take vitamin D supplements are likely to have low serum 25-hydroxyvitamin D [25(OH)D] concentrations. Vitamin D can reduce the risk of viral epidemics and pandemics in several ways. First, higher 25(OH)D concentrations reduce the risk of many chronic diseases, including cancers, cardiovascular disease, chronic respiratory tract infections (RTIs), diabetes mellitus, and hypertension. Patients with chronic diseases have significantly higher risk of death from RTIs than otherwise healthy people. Second, vitamin D reduces risk of RTIs through three mechanisms: maintaining tight junctions, killing enveloped viruses through induction of cathelicidin and defensins, and reducing production of proinflammatory cytokines by the innate immune system, thereby reducing the risk of a cytokine storm leading to pneumonia. Observational and supplementation trials have reported higher 25(OH)D concentrations associated with reduced risk of dengue, hepatitis, herpesvirus, hepatitis B and C viruses, human immunodeficiency virus, influenza, respiratory syncytial virus infections, and pneumonia. Results of a community field trial reported herein indicated that 25(OH)D concentrations above 50 ng/ml (125 nmol/l) vs. <20 ng/ml were associated with a 27% reduction in influenza-like illnesses. From the available evidence, we hypothesize that raising serum 25(OH)D concentrations through vitamin D supplementation could reduce the incidence, severity, and risk of death from influenza, pneumonia, and the current COVID-19 epidemic.

24 P Raharusun, S Priambada, C Budiari, E Agung and C Budi. Patterns of COVID-19 mortality and vitamin D: An Indonesian study. SSRN: https://ssrn.com/abstract=3585561 or http://dx.doi.org/10.2139/ssrn.3585561, 26 April 2020. “Univariate analysis revealed that older and male cases with pre-existing condition and below normal Vitamin D levels were associated with increasing odds of death. When controlling for age, sex, and comorbidity, Vitamin D status is strongly associated with COVID-19 mortality outcome of cases.”

25 Tomás Cuñat, Antonio Ojeda and Andrea Calvo. Diagnosis of vitamin D deficiency in patients admitted in ICU with Covid-19 disease. July 2020. “During the period from March 16-April 26, 2020, 226 patients were admitted in ICU, and the value of 25-hydroxy-vitamin D was observed in only 17 patients (7.5%). Besides that, we obtained relevant data: all patients with serum determinations of 25-hydroxyvitamin D presented a level lower than 20 ng/ml and thirteen patients (76.5%) levels < 12.5 ng/ml. We conclude that vitamin D deficiency is common in critically ill COVID-19 patients, and it continues being undiagnosed.”


The data is so blatant that it was written up as an editorial appeal to the medical profession.\(^{28}\)

When the above-20 ng/ml (green plus green) are compared to the below-20 ng/ml (red plus red), the same pattern is seen: two deaths in the above-20 ng/ml green group and 19 deaths in the below-20 red group. Clearly, vitamin D is predicting everything that matters, both the severity of disease and the odds of death.

The other studies show the same clinical and statistical pattern, with findings of 2-fold to 8-fold reductions in morbidity (disease) and mortality (death).

If this is not impressive enough for you to immediately purchase a bottle of 50,000 IU vitamin D\(_3\) capsules,\(^{29}\) let me re-frame the issue in a real-life context. This will be explained in exacting detail later, but what these studies are actually measuring is the clinical course of relatively subtle differences in vitamin D levels. When comparing below-20 to above-20, the results are impressive. Same with below-30 to above-30. But when you compare below ten to above 30 (skipping the 10-20 patients), or below 20 to above 40 (skipping the 30-40 patients), the outcomes are way beyond ten-fold improvements in Covid-19 severity and survival.

That is the hoped-for Covid-19-vaccine efficacy.

A Covid-19 solution that is as good as a best-scenario vaccine?

And it’s been sitting on store shelves the whole time.

There are many reasons why vitamin D levels are now pathologically low: fear of sun exposure, erroneous “professional” advice from dermatologists to stay out of the sun or use sunscreens, cultural prejudices towards the lightest possible skin color, indoor lifestyles (home gyms, stationary exercise bicycles, etc.), time pressures (busy, busy, busy), and governmental, NGO and medical exaggerations of vitamin D toxicity. This modern trend towards increasingly low levels of vitamin D is one of the drivers of viral diseases, both chronic and acute.\(^{30,31}\)

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\(^{28}\) https://www.medpagetoday.com/infectiousdisease/covid19/90530?fbclid=IwAR1iAwEG_2dXcQHNX5DWGCebuVjg7gs0YAlvP7TXLrl0UUPyMi3BKlj5eg8

\(^{29}\) One hundred capsules at one per week is a two-year supply, for less than $25. That’s the dose I take. Even if you want one capsule every four days, that’s more than one year of protection for $25.

\(^{30}\) Acute viral diseases would include infections from influenza, coronaviruses (SARS-1, MERS and SARS-2), and hemorrhagic fevers (ebola and Marburg). Chronic viral diseases would include infections from herpesviruses, cytomegaloviruses, Epstein-Barr viruses, hepatitis C viruses, and HIV. For an expanded list of lipid-enveloped viruses, see page 28.

Since vitamin D is also associated with other morbidities and mortalities in the same way, the low levels of vitamin D are likely driving many other pathologies, both chronic and acute. Some of these will be detailed later.

Let me take a moment to re-frame the vitamin D issue; it’s more a hormone than a vitamin. Think of it as the sunshine hormone. It drives many different aspects of metabolic activity, many of which are independent antiviral influences. It works in synergy with progesterone, testosterone, thyroid hormone, and dopamine. It also synergizes with vitamin A, vitamin K2 and calcium.

Vitamin D is a necessary and essential aspect of innate immune defense. Innate immunity is our first line of defense against viral infections. For many readers, this context for vitamin D will come as a surprise. But I have to ask, *why did it take me to bring this to your attention?*

The typical amount of supplementation it takes to get vitamin D blood levels into the antiviral range is roughly 5,000 IU (international units) per day. This may seem counterintuitive when such levels have been officially promoted (propagandized) as toxic in the past, but such doses have been in clinical and popular use for 60 years without any sign of toxicity whatsoever. In fact, the 5000 IU “antiviral” dose, and the 10-80,000 IU “therapeutic” doses are well supported by clear evidence. So the 400 IU or 800 IU recommended daily intakes will simply not cut it. They are grossly inadequate. They are ten to twenty times inadequate. Even the promotionally progressive 1000 IU formulas are insufficient unless augmented by plenty of the right kind of sunshine (see page 31). One study showed that even 3,000 IU per day was insufficient to get 20% of people to the bottom of the low-normal range.

Since you cannot “feel” your low or high vitamin D3 levels, you need to get yourself tested. Such testing can be conducted by your physician or performed on an over-the-counter basis.

It might take 7,000 or 15,000 IU for you to hit your chosen target, or it might only take 2,000 or 4,000 IU. Seasonal, genetic, metabolic, skin color and actual sun exposure affect how much supplementation is required to hit a particular range. This will be clarified a bit later in this chapter.

Among testing systems, there are two D3 ranges. In the USA, the units are ng/ml (nanograms per milliliter). In the EU, the units are nmol/L (nanomoles per liter). There is a 2.5-fold difference between those units.

There are many different opinions about the ideal and/or therapeutic levels for vitamin D.

<table>
<thead>
<tr>
<th>Pathological Level</th>
<th>Value 1</th>
<th>Value 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gross pathological deficiency</td>
<td>&lt;10 ng/ml</td>
<td>&lt;25 nmol/L</td>
</tr>
<tr>
<td>Widespread levels in the population</td>
<td>10-20 ng/ml</td>
<td>25-50 nmol/L</td>
</tr>
<tr>
<td>Canadian “insufficiency” level</td>
<td>&lt;16 ng/ml</td>
<td>&lt;40 nmol/L</td>
</tr>
</tbody>
</table>

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32 There are researchers who do not yet appreciate vitamin D’s therapeutic effects against viruses and are still focused mainly on the known “bad” effects of vitamin D deficiencies on viral disease. When >30 ng/ml is defined as vitamin D sufficiency, the conclusion is that 1200-2000 IU’s of vitamin D supplementation is the right antiviral dose. But the assumption that >30 ng/ml is “sufficient” for viral defense is pure assumption, unsupported by any semblance of science or medical data. If one does look more closely at the data, 40 ng/ml is clearly better than 30 ng/ml. So >40 is actually a better supposition. Even higher levels have been cited as better inducers of cathelicidin, an antiviral peptide that acts on viral replication at the cellular level. See the table above for target suggestions.

33 D E Roth, P Martz, R Yeo, C Prosser, M Bell and A B Jones. Are National Vitamin D Guidelines Sufficient to Maintain Adequate Blood Levels in Children? *Canadian Journal of Public Health* 96: 443–449, Nov 2005. “Vitamin D insufficiency (defined as 25-hydroxyvitamin D [25(OH)D] concentrations <40 nmol/L) may be associated with subclinical adverse effects on bone mineralization.” Conclusion: “No subject was insufficient if they had an intake >0.45 mcg/kg/day.” This is 18 mcg per kilogram as a supplement, which is roughly the RDA.
more mainstream insufficiency level: < 20 ng/ml < 50 nmol/L
still-not-enough insufficiency level: 20-30 ng/ml 50-75 nmol/L \(^{40}\)
conservative recommendations: 30-40 ng/ml 75-100 nmol/L
epidemiological minimization of total mortality: 40-50 ng/ml 100-125 nmol/L \(^{61}\)
Steve’s personal target: 50-56 ng/ml 125-140 nmol/L
aggressive recommendations: 60-80 ng/ml 125-200 nmol/L

Here is a good summation of the evidence and conclusions. \(^{34}\)

If you compare the first line to the following lines, you can easily see vitamin D’s widespread insufficiency or outright deficiency.

To provide an experiential context, it takes 2 hours per day in a bikini (90% skin exposure) to get most people in Hawaii into the 30-50 ng/ml range (above 80 nmol/L). California lifeguards at the end of the summer were in the 40-60 ng/ml range (100-150 nmol/L). And the mean vitamin D$_3$ level for dark-skinned equatorial Africans living a “traditional lifestyle” was 46 ng/ml (115 nmol/L), with the upper limit being just below 80 ng/ml (200 nmol/L). \(^{35}\)

Skin color has a significant effect. A 2003-2006 survey found White Americans with mean D$_3$ levels of 26 ng/ml, Mexican Americans with 20 ng/ml and Black Americans with 15 ng/ml.

In another study, \(^{36}\) the overall deficiency in the US population was 41.6% with <20 ng/ml (50 nmol/L) as the deficiency threshold. But Hispanics and Afro-Americans had deficiencies of 69% and 82%, respectively.

Other factors affecting vitamin D production will follow in the next section.

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\(^{34}\) P J Teben, R J Singh and R Kumar. Vitamin D-mediated hypercalcemia: mechanisms, diagnosis and treatment. *Endocrine Reviews* 37(5): 521-47, October 2016. “The upper tolerable limit, defined as the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects to almost all individuals in the general population, for vitamin D$_3$ is 1000 IU/d in infants ages 0–6 months, 1500 IU/d in infants ages 6–12 months; 2500 IU/d in children ages 1–5 years; 3000 IU/d in children ages 4–8 years, and 4000 IU/d in adolescents and adults. The short-term ingestion of up to 10 000 IU/d of vitamin D$_3$ is associated with the maintenance of 25(OH)D serum concentrations below 50 ng/mL (125 nmol/L), a concentration below which toxicity has not been observed. In a study of 40 patients with metastatic breast tumors, daily doses of 10,000 IU vitamin D$_3$ for 4 months were not associated with hypercalcemia although small increases in serum calcium and decreases in PTH were observed.”

\(^{35}\) S J Wimalawansa. Non-musculoskeletal benefits of vitamin D. *J Steroid Biochem Mol Biol.* 2018 Jan;175:60-81, Jan 2018. “1,25(OH)$_2$D improves immunity; subdues inflammation; and reduces the incidence and severity of common cancers; autoimmune diseases and infectious diseases. Almost all adequately powered; epidemiological and biological studies that use; adequate doses of vitamin D supplementation in D-deficient populations have reported favorable outcomes. These studies have concluded that optimizing 25(OH)D status improves the functionality of bodily systems; reduces comorbidities; improves the quality of life; and increases survival.” “…most studies point to significant protective effects of vitamin D in humans when the minimum 25(OH)D serum level exceeds 30ng/mL and is maintained throughout the year.”

\(^{36}\) K Y Forrest and W L Stuhldreher. Prevalence and correlates of vitamin D deficiency in US adults. *Nutr Res.* 2011 Jan;31(1):48-54, 2011. “In summary, vitamin D deficiency was common in the US population, especially among blacks and Hispanics. Given that vitamin D deficiency is linked to some of the important risk factors of leading causes of death in the United States, it is important that health professionals are aware of this connection and offer dietary and other intervention strategies to correct vitamin D deficiency, especially in minority groups.”
The literature connecting low vitamin D levels with diseases is extensive.\(^\text{37}\) Regarding viral diseases, influenza and cold was reduced in post-menopausal black women in New York state.\(^\text{38}\) The placebo group had 26 cold-or-flue events, the 800 IU vitamin D group had seven events, and the 2000 IU group had one cold-or-flu event. Although the literature on Covid-19 is less extensive, it is conclusive.\(^\text{27}\) The November 2020 meta review of vitamin D and Covid-19 was deemed so definitive that an ethical appeal to the medical profession was published.\(^\text{28}\)

See also an editorial.\(^\text{39}\)

There is considerable research suggesting that the optimal level for vitamin D for resistance to viral diseases and for heart disease mortality, stroke mortality and for total mortality are similar (see page 42).

Research connecting pre-infection levels of vitamin D in CoV-19 suggest that vitamin D’s preventive effects level off when the concentration of vitamin D reaches either (1) 30-40 ng/ml (75-100 nmol/L) or (2) 40-50 ng/ml (100-125 nmol/L). But it is quite clear that vitamin D supplementation of less than 3,000 IU per day was associated with increased risk.\(^\text{40}\)

<table>
<thead>
<tr>
<th>Supplementation Level</th>
<th>CoV-19 Positive Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>No supplementation</td>
<td>13% CoV-19 positive</td>
</tr>
<tr>
<td>&lt;1000 IU supplementation</td>
<td>20% CoV-19 positive</td>
</tr>
<tr>
<td>2000 IU supplementation</td>
<td>15% CoV-19 positive</td>
</tr>
<tr>
<td>&gt;3000 IU supplementation</td>
<td>8% CoV-19 positive</td>
</tr>
</tbody>
</table>

In that study, the degree of supplementation for all three groups was insufficient to get everybody’s vitamin D levels above 20 ng/ml (the study’s definition of vitamin D “deficiency”). See how the supplemented dose of vitamin D affected the percentage of people with less than 20 ng/ml.

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\(^{39}\) S. Wimalawansa. Minimize risks of Covid-19 infection. *Journal of Biomedical Research and Environmental Sciences*, December 2020. doi: 10.37871/jbres1174. Editorial, and Abstract: “The only cost-effective medical/nutritional intervention that reduces Covid-19 infection and its complications and deaths is vitamin D. Those who are at high risk, following exposure to a person with Covid-19 or a person with symptoms of Covid-19 infection likely to benefit from an upfront loading oral dose, such as between 100,000 and 400,000 IU as a stat dose or over a few days, could mitigate the illness. The same approach should be considered in all Covid-19 clinical trials conducted using vitamin D as an intervention. Following an exposure, PCR positivity, or having signs and/or symptoms of Covid-19, a potential rescue mean is to consume 50,000 IU vitamin D daily for 4 to 7 days, followed up with a maintenance dose of 4,000 IU/day. Such approaches are especially important as those people with chronic diseases, the elderly, and those in nursing homes, who are known to have a high prevalence of severe vitamin D deficiency, thus, are at high risk for Covid-19.”

\(^{40}\) D O Meltzer, T J Best, Hui Zhang, *et al.* Association of vitamin D deficiency and treatment with Covid-19 incidence. MedRxiv preprint doi: https://doi.org/10.1101/2020.05.08.20095893. Posted May 13, 2020. Results: “Among 4,314 patients tested for COVID-19, 499 had a vitamin D level in the year before testing. Vitamin D status at the time of COVID-19 testing was categorized as likely deficient for 127(25%) patients, likely sufficient for 291(58%) patients, and uncertain for 81(16%) patients. In multivariate analysis, testing positive for COVID-19 was associated with increasing age (RR(age<50)=1.05, p<0.02; RR(age≥50)=1.02, p<0.064), non-white race (RR=2.54, p<0.01) and being likely vitamin D deficient (deficient/treatment-not-increased: RR=1.77, p<0.02) as compared to likely vitamin D sufficient (not deficient/treatment-not-decreased), with predicted COVID-19 rates in the vitamin D deficient group of 21.6% (95%CI [14.0%-29.2%]) versus 12.2% (95%CI [8.9%-15.4%]) in the vitamin D sufficient group. Vitamin D deficiency declined with increasing vitamin D dose, especially of vitamin D1. Vitamin D dose was not significantly associated with testing positive for COVID-19.” See also: Meltzer, *et al.* Association of vitamin D status and other clinical characteristics with Covid-19 test results. *JAMA Netw Open* 3: e2019722, 2020. doi: 10.1001/jamanetworkopen.2020.19722.
no supplementation  63% deficient
  total test-group average  59% deficient
<1000 IU supplementation  54% deficient
  2000 IU supplementation  34% deficient
>3000 IU supplementation  21% deficient

It is now unambiguous that 2000 IU per day is insufficient to get to optimal vitamin D levels for the vast majority of people.

Even doses of 3,000 IU per day was not enough for one of every five people to get to only 20 ng/ml level, which we will see a bit later, is roughly half of the level of vitamin D for effectively managing viral-disease and mortality risks. This reinforces the general recommendation of 5,000 IU as a good starting point for vitamin D repletion. In an in vitro study of dengue virus replication, 4000 IU intake in humans was found to be superior to 1000 IU in decreasing dengue virus replication and immune overreaction when their antigen-presenting cells were extracted and exposed to the virus.  

More than a few researchers have made claims of specific doses for vitamin D optimization based on the dose-response curve becoming flat at the claimed dose. These claims have now been refuted by a well-designed study of the effect of vitamin D₃ on parathyroid hormone levels which shows a steadily increasing inhibitory effect at all doses tested and at all ages tested. This analysis was made from a database of more than 300,000 test results collected over a one-year period of time.

In the next section, I discuss local and personal aspects of vitamin D sufficiency, which will bring the range for supplementation into more detail. While 5000 IU may be a good starting dose, the ideal dose will vary from 3,000-15,000 IU per day, with 3,000 IU being sufficient in those with white skin, robust sun-exposure habits and a good altitude and latitude who are shooting for a lower target zone, with 15,000 IU possibly being sufficient for those with dark skin, little sun exposure, living at extreme latitude in an urban, air-polluted environment, and shooting for a higher target zone.

As you can see, 3,000 IU to 15,000 IU is a huge range. That’s why testing is so important.

What’s Your Local Vitamin D Risk?
The following factors influence sunlight, UV-B and vitamin-D conversion.

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41 J Martinez-Moreno, J C Hernandez and S Urcuqui-Inchima. Effect of high doses of vitamin D supplementation on dengue virus replication, Toll-like receptor expression, and cytokine profiles on dendritic cells. Molecular and Cellular Biochemistry 464: 169–180, preprint 22 Nov 2019. “We found that MDDCs from donors who received 4000 IU/day of vitD were less susceptible to DENV-2 infection than MDDCs from donors who received 1000 IU/day of vitD. Moreover, these cells showed decreased mRNA expression of TLR3, 7, and 9; downregulation of IL-12/IL-8 production; and increased IL-10 secretion in response to DENV-2 infection. In conclusion, the administration of 4000 IU/day of vitD decreased DENV-2 infection. Our findings support a possible role of vitD in improving the innate immune response against DENV. However, further studies are necessary to determine the role of vitD on DENV replication and its innate immune response modulation in MDDCs."


43 UV-B is ultraviolet light in the B frequency range. There are four ultraviolet ranges. UV-C is the most energetic and damaging to skin. UV-B is less energetic and catalyzes the conversion of 7-dehydrocholesterol into the precursor for vitamin D. UV-B also destroys the precursors for vitamin D, which makes sunlight-induced vitamin D self-limiting. In other words, sunlight exposure is not linear; you get more vitamin D in the first ten minutes than the second ten minutes, and even less in the third and fourth ten minutes. UV-A is slightly less energetic and does not activate cholesterol to make vitamin D₃. And finally, there is “black light,” which is at the threshold between visible and ultraviolet light. Black light is used to cause
Skin pigment. The darker your skin, the less UV-B gets through the outer layers of the skin to reach the underlying dermis where it can make vitamin D. This applies to natural pigmentation and to tanning pigmentation. Melanin absorbs a broad range of light frequencies, from the low-energy visible light to exceedingly energetic UV-C which can only damage the skin and cause skin cancers. But the important thing to remember is that melanin absorbs the UV-B which makes vitamin D.

Altitude. The atmosphere absorbs UV-B. So the thinner the atmosphere, the more UV-B you can get. Higher altitude means less atmosphere between you and the sun, and more vitamin D per minute of sun exposure. Lower altitude means more atmosphere between you and the sun and less vitamin D produced per minute of sun exposure.

Time of day. The thickness of the atmosphere also depends on the time of day. At noon, sunlight is as close as it will get to being vertical, and atmosphere the thinnest. In the early morning and late afternoon, the sun slants through the atmosphere and less UV-B gets through. At dawn and dusk, the angle is so severe that UV-B is zero, and even the blue light is low.

Latitude. The thickness of the atmosphere also depends on your latitude. The atmosphere is thinnest when the sun is directly overhead, which occurs at the equator, and in the tropics (between -23 and +23 degrees of latitude) depending on the season. The further north or south you are, the thicker the atmosphere and the less UV-B gets through.

Air pollution. Many air pollutants absorb ultraviolet frequencies. That’s one reason they call air pollution “photochemical smog.” If you live in a city with air pollution, your UV-B is diminished. How much depends on the amount of air pollution. Where I live (the San Francisco bay area), the prevailing winds off the Pacific Ocean blow the air across San Francisco, across the bay, into the Oakland hills. During the summer, the air pollution in Oakland is sometimes so high that there are days when there is no UV-B at street level. In San Francisco, the air pollution in the Mission District is moderate and in “The Avenues” next to the beach is near zero. So the urban landscape can be a significant factor. In the Los Angeles basin, the predomint onshore breezes stack the air pollution in the east and keep the air over the beach communities fairly clean. This reverses when the Santa Ana winds blow.

Season. The tilt of the Earth’s axis is 23 degrees relative to the sun. That means that we experience seasons: summer when the tilt of your hemisphere (north or south) is towards the sun and winter when the tilt is away from the sun. This means that you get much more UV-B in the summer than you do in the winter. This seasonality also affects the temperature of the weather, and the amount of clothes we wear to stay warm outdoors.

Clothing. The greater the clothing worn, the less skin surface exposed to the UV-B rays. The further north you are and the higher in altitude you are, the colder it tends to be. This also compounds the seasonal contribution to low vitamin D levels.

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fluorescence of pigments and otherwise-invisible stains that can convert ultraviolet light into visible-light frequencies. Black light can be used forensically to gather trace evidence and is used commercially to find leaks in mechanical systems.
The graph\textsuperscript{35} at the top of the previous page illustrates many of the above D-related elements. The effect of latitude and clothing can be seen in the difference between the “non-Hispanic blacks” curve and the “traditional equatorial Africans” curve. The effect of skin pigmentation can be seen in the progressive shift of the green, black and red curves to the right (Blacks, Hispanics and Whites, respectively).

The Flu Season

This seasonality of UV-B exposure and weather temperature is the basis for the “flu season.” The winter solstice (the 20\textsuperscript{th} or 21\textsuperscript{st} of December) is when the length of the day and vitamin D synthesis bottoms out. The long half-life of vitamin D means that vitamin D levels continue to fall for up to two more months, which makes February the depth of the flu season. But vitamin D synthesis has been falling steadily during the Fall months, so the flu season starts a month or two before the winter solstice. The summer solstice (the 20\textsuperscript{th} or 21\textsuperscript{st} of June) is the longest day and the beginning of the anti-flu season.

Experts disagree about the importance of different aspects of the seasonality issue. Some believe that low ultraviolet-B radiation and falling vitamin D levels are the greatest influence.\textsuperscript{37} This is not popular with public-health experts who do not like to acknowledge any role of malnutrition as being part of a public-health problem. So those experts blame the flu season on colder weather. That’s the theory plastered all over the Internet by official governmental and NGO organizations. This politically motivated policy has recently been challenged by an analysis of 2020 Covid-19 data where there was a sudden uptick in Covid-19 medical cases across Europe where the temperature data (dashed red line) had a very poor correlation and latitude (solid-blue line) had a high correlation.\textsuperscript{44}

I believe that both effects are deeply involved and that they synergize to make people colder in body temperature. Vitamin D drives body metabolism and body temperature up, so the waning vitamin D level in the fall season lets people’s body temperature decrease at the same time that weather is creating a temperature challenge. This is made worse by our living indoors in a temperature stabilized environment, which inhibits out ability to upregulate our metabolism in response to the cold. Living indoors means that we experience a greater temperature challenge when we do go outdoors, and for some of us, warming up after getting chilled is a much bigger deal in the winter.

\textsuperscript{44}Stephand Walrand. The unexpected spectacular 2020 October Covid-19 boost in European countries correlated with the latitude, but not with temperature. Preprint. https://www.researchgate.net/publication/344901911. This analysis compares the October dates of the sudden increase in the rate of Covid-19 to (a) latitude (the population-weighted centers of each country) and (b) temperature (the temperature a week preceding the date of increase). This study has several limitations. (1) The source of the statistics of governmental databases online. (2) The identification of a “date of onset” of Covid-19 increase (some chosen dates are possibly arbitrary). And (3) The lack of control of unknown variables affecting viral pandemic and endemic spreading—and reporting.
According to a 2020 report from China, the TCM (traditional Chinese medicine) classification of the coronavirus is “cold and damp.” This also appears to be the case for influenza. What this may mean is that stacking “cold” and “damp” foods on top of a cold and damp season is a risky thing to do because it gives cold-and-damp viruses their preferred conditions.

There is a western medicine analog to these Eastern Medicine concepts. I do not capitalize western medicine in this context because I am not using the term in its monolithic sense (the anti-competitive institutions of Western Medicine), but rather as medicine derived more by analytical-scientific methods rather than clinical-cultural-observational means. Emanuel Revici’s concept of anabolic-anaerobic-alkalinizing substances and conditions is parallel to the TCM concept of “cold and damp.” There is an entire section of this book devoted to explaining the meanings behind Revici’s concepts (see page 120), so I will not go into it in any depth here, except to say that (1) it is energy metabolism that produces warmth, therefore a low basal metabolic rate is “cool” or cold, (2) and “acid” is the byproduct of cellular energy production, either carbonic acid (hydrated carbon dioxide) if the energy production is aerobic (the efficient and “healthy” way to make energy), or lactic acid if it is anaerobic (the inefficient, temporary and/or unhealthy way to make energy). So low energy production translates to alkalinity and coldness.

Either way, the flu season starts when the weather changes in the fall, the days get shorter, the temperature drops, and we start wearing more clothing to stay warm.

**Vitamin D Supplementation: Preventive, Therapeutic or Both?**

The governments of the world spend billions assisting the pharmaceutical industry with drug development but spends just millions on non-drug therapeutics. And a portion of that meager governmental funding goes to politically motivated studies with flawed protocols, manipulative data analysis and dishonest conclusions. Stanford University Professor John Ioannidis has found that most (greater than 50%) of modern scientific “findings” (controlled, double blind, statistically significant, as reported) are invalidated within ten years. And this rate is higher for nutrition, drug-candidate and psychology research.

Science is not the careful, impartial, reliable process that scientists imply.

Given the lack of institutional incentives, it’s hard to say that there is a specific answer to such simple questions as “If I get a coronavirus infection, should I take more vitamin D3?”

For example, it might take 1-2 years before cancer risks are decreased by vitamin D supplementation. If this is true, does it also apply to viral diseases? Vitamin D3 is known to rapidly enhance production of cathelicidin (kath-eh-lih-SLIGH-din), an innate immune defense mechanism that fights viral replication inside of infected cells. But this has not been studied sufficiently to recommend a specific clinical protocol to optimize such a potential therapeutic effect.

Given the importance of such questions to public health and welfare, you might think that such questions would be thoroughly researched. But while epidemiological research suggests that low vitamin D is

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45 The TCM take on swine flu (H1N1 influenza): it’s “pathogenic heat, cold and damp.” Pathogenic heat is fever and inflammation. [http://www.china.org.cn/health/2009-05/13/content_17767231.htm](http://www.china.org.cn/health/2009-05/13/content_17767231.htm)

46 Re cold and damp: “As the weather turns colder, you should avoid eating foods which create cold and damp conditions within your body. These include salads, raw foods, and cold drinks, which should be kept to a minimum throughout the winter months. Instead, consume more hearty foods that will warm your body from within. Good examples of these include sweet potatoes, pumpkin, garlic, beetroot, cinnamon, turmeric, and cardamom.” [https://tcmblog.co.uk/how-to-treat-the-common-cold-and-flu-with-traditional-chinese-medicine/](https://tcmblog.co.uk/how-to-treat-the-common-cold-and-flu-with-traditional-chinese-medicine/)
definitely connected to bad viral outcomes.\textsuperscript{47} It is not yet absolutely clear that raising vitamin D during the infection fixes this risk. At least to the skeptics and deniers controlling public-health policy.

The first clear evidence of vitamin D\textsubscript{3}’s clinical effect on the course of Covid-19 has now been released as a pre-publication paper.\textsuperscript{48} It shows that vitamin D\textsubscript{3} supplementation virtually eliminates the need for intensive care.

In other words, if replicated, and implemented, it would effectively eliminate ICU crowding.

This was a 2:1 randomized study of 76 Covid-19 patients admitted to a university hospital in Spain in which 50 patients were given weekly vitamin D\textsubscript{3} and 26 patients were not. The vitamin D supplement was 21,280 IU (0.532 mg), with a double dose in the first week and a single dose in subsequent weeks. Taken daily, that’d be roughly 6,000 IU for the first seven days and 3,000 IU daily thereafter. The sweet spot for vitamin D levels mentioned previously is 3-12K/day depending on sun exposure, season, skin pigmentation and biochemical individuality, so this dose is cautiously conservative.

All the patients received the standard hospital therapeutic protocol of hydroxychloroquine (without zinc) and azithromycin. Intensive-care-unit (ICU) patients with pneumonia also received an intravenous broad-spectrum antibiotic (ceftiraxone).

Of the vitamin D group of 50 patients, 1 was admitted to the ICU and none died.

Of the control group pf 26 patients, 13 were admitted to ICU and two died.

While the randomization program did a fairly good job of randomizing people, the non-vitamin D group did end up with more men, more cardiovascular disease, way more high blood pressure and more diabetes, while the vitamin D group had older age, older men, younger women and three-fold more immunosuppression (including organ transplants). So the results may not be a striking once those risk factors are better controlled in a bigger study, or a more thorough analysis.

This study also did not collect data on body mass index, insulin resistance or leptin resistance. So the obesity influence on morbidity and mortality is not known.

The variable that gives me the greatest pause is the “D-Dimer” differences between the two groups, with the non-D group having twice the level as the D-supplemented group. D-Dimer is a marker for the breakdown of prior clots. So this could be an associated marker of the blood-pressure difference between the

\textsuperscript{47} P Raharusun, S Priambada, C Budiari, E Agung and C Budi. Patterns of COVID-19 mortality and vitamin D: An Indonesian study. SSRN: https://ssrn.com/abstract=3585561 or http://dx.doi.org/10.2139/ssrn.3585561, 26 April 2020. “Univariate analysis revealed that older and male cases with pre-existing condition and below normal Vitamin D levels were associated with increasing odds of death. When controlling for age, sex, and comorbidity, Vitamin D status is strongly associated with COVID-19 mortality outcome of cases.”

two groups. This may also relate to a higher ferritin (over 800) in the non-D group compared to just under 700 among the D-supplemented group. Ferritin is an acute-phase reactant and elevates noticeably during infection and inflammation. So it is more a measure of acute inflammation than a good marker of iron status that might predict the degree of Fenton chemistry (oxidative stress) that might be differentially challenging the two groups.

Despite criticism of the study for the above reasons, and its outright dismissal because of such differences between groups, a similar result was found in a study of severe Covid-19 outcomes in an “older” population in Singapore.49 This study examined the effects of a combination of vitamin D, magnesium and vitamin B12, the latter of which is known to be a near-universal deficiency in the elderly. The results?

Of 26 controls, 16 required oxygen, 16 required intensive care admissions.

Of the 17 treated with vitamin D, only 3 required oxygen and none required intensive care.

In other words, the oxygen requirements were 61.5% in the controls and 17.6% in the treated group, a 71% reduction. For ICU admissions, it was 61.5% in the controls and zero in the vitamin D, magnesium and B12 group, an infinite reduction.

So we have two studies, both relatively small, but both showing tremendous reductions in the need for intensive care. The Spanish hospital study showed a reduction from 50% to 2%, and the Singapore study showed a reduction of 61.5% to zero. So why is everybody in the world still using the crowded-hospitals argument to justify continuing panic-induced public policies? One simple therapeutic change in treatment would solve the problem, and one single medium-scale study of maybe 400-600 hospital Covid-19 admissions would prove it beyond any reasonable doubt for the skeptics still on the fence or the public-health bureaucrats still badmouthing vitamin D.

The cost to the hospitals? 20¢ per patient per day for two weeks. Less than $3 per patient.50

49 C W Tan, L P Ho, S Kalimuddin et al. A cohort study to evaluate the effect of combination vitamin D, magnesium and vitamin B12 (DMB) in progression to severe outcome in older Covid-19 patients. medRxiv preprint. doi: 10.1101/2020.06.01.20112334 From the abstract: “In univariate analysis, age and hypertension showed significant influence on outcome while DMB retained protective significance after adjusting for age or hypertension separately in multivariate analysis. Fewer DMB patients than controls required initiation of oxygen therapy during their hospitalization (17.6% vs 61.5%, P=0.006). DMB exposure was associated with odds ratios of 0.13 (95% CI: 0.03 – 0.59) and 0.20 (95% CI: 0.04 – 0.93) for oxygen therapy and/or intensive care support on univariate and multivariate analyses respectively. Conclusions: DMB combination in older COVID-19 patients was associated with a significant reduction in proportion of patients with clinical deterioration requiring oxygen support and/or intensive care support.”

50 This calculation is based on (1) bulk-purchasing of 50,000 IU vitamin D supplements, BioTech brand, at near-Amazon prices, one capsule per day, and (2) a time frame of two weeks as established by a study of vitamin-D deficient Covid-19 patients being given 50,000 IU of vitamin D3 per day until they reached blood levels of 50,000 ng/ml. At two weeks, 60% of the patients had reached 50 ng/ml (125 nmol/L) starting with blood levels below 20 ng/ml (everybody) and a significant minority below 10 ng/ml. There is no scientific question that 50 ng/ml is the flat part of the vitamin-D mortality-risk curve, but 40 ng/ml is quite close to being flat, and 30 ng/ml to likely be sufficient given the conspicuous results of much, much, much lower vitamin D intakes on Covid-19 outcomes. So those 40% of patients who did not reach 50 ng/ml taking 50,000 IU/day were likely above 40 ng/ml, and maybe a few merely above 30 ng/ml.
I think of vitamin D status as a general, constitutional issue. Vitamin D supports a wide range of fundamental health mechanisms, and it is these that directly affect viral disease risks—and cancer risks, and heart disease risks, and melanoma risks. If this is so, raising your vitamin D is predominantly a preventive strategy and not a therapeutic strategy. In the above study, only hospitalized patients were studied, which would be expected to have lower vitamin D status compared to those in general population who caught Covid-19 but did not go to the hospital for care.

But might vitamin D₃ supplementation in people with sufficient or high vitamin D₃ still be therapeutic? Certainly. Just because its benefits are mainly cumulative over time for cancer and other diseases does not mean that it cannot be therapeutic over the timeframes of days and weeks for a different therapeutic purpose. Vitamin D₃ does raise cathelicidin, which is an antiviral peptide that is conserved in all animal species.

I expect that the therapeutic benefits of vitamin D₃ may be quite higher if your vitamin D blood level is 10 ng/ml as opposed to 20 ng/ml or 40 ng/ml. University of California Professor Bruce Ames has proposed that age-related deficits in nutrition status are a serious “aging” problem, but one that can be corrected by supplementation. The deeper the vitamin D deficiency, the greater the adverse effect it might have on a clinical outcome and the greater the potential beneficial effect of immediate supplementation. This potential for therapeutics is clearly indicated by the Spanish hospital study results.

Then there is the question of optimization. Mother Nature does not address this issue because optimization is expensive, and she deals with cost-benefit realities. Raising vitamin D levels has a cost, as does raising vitamin C, selenium or niacinamide levels. Mother Nature judges such costs in terms of reproductive fitness. But we have a different standard. Our value as human beings is not just about our effect on the next generation, but about our selves, our happiness, our contributions, our experiences, our achievements and our values. We are thoughtful, emotional and spiritual beings, whose welfare and wellbeing goes far, far beyond our reproductive fitness and the stability of our family and social groupings. We describe ourselves as having souls, with creative, loving and value-driven natures. We “feed” ourselves in ways beyond simple nutrition, by ways that range from the self-assertive to the self-transcendent. What this means is that we and our loved ones value our survival more than Mother Nature does.

So how does this affect the cost-benefit relationship? *Cost becomes less important.*

An example: In a “wild” environment on the shores of an inland African sea or lake, the cost of a gram of vitamin C or 100 micrograms of selenium would be high. To get to a fruit containing vitamin C, you might have to climb a tree, from which you might slip and fall. To get selenium, you might have to dive in the sea to a depth of 10-20 meters to harvest mussels or clams. These are risky actions. But today, you can buy a gram of vitamin C for five cents, or maybe a dollar if it is buffered, packaged for convenience and/or delivered in liposomal form. And 100 micrograms of selenium costs anywhere from a tenth of a cent to five cents, depending on if you are taking homemade selenite or encapsulated methylselenocysteine. It is clear that the risk-benefit equation today is vastly different, now that we know how to ferment vitamin C, mine selenium, and add selenium to yeast cultures.

So what does this mean for vitamin D supplementation?

You will have to decide based on limited information, under the guidance of your personal values, how much to take and what your target is. As an avid biohacker, I’m not particularly risk averse and am comfortable with vitamin D₃ levels above 60 ng/ml. But you might be more risk averse than me and go for the 40 ng/ml.
And maybe your risk comfort is different when it’s your ailing grandmother and not you, personally? These kinds of decisions are intrinsically value dependent. That means that there is no right answer outside of the values of each person. Although science may say that 7 ng/ml and 19 ng/ml are levels of vitamin D that are pathological, science cannot yet tell us what is optimal, let alone optimal in the context of a life-threatening infection.

The last aspect of cost-benefit equations is that benefit is not one dimensional. Not at all. Benefits relate not just to the effect that a single nutrient may have in the general population but to what it has in you, which must necessarily take into account your biochemical individuality, your metabolic individuality, your constitutional individuality, and your genetic and epigenetic individuality.

And regarding values? Who do you trust?

**Therapeutic Effects**

Vitamin D acts on many different systems of the body that might be considered constitutional. So there is a fundamental question as to its relative value in prevention or treatment of viral pathology. In other words, how much do you need to take vitamin D in advance of a viral infection? And how much do you take during an infection to treat it? And lastly, how much does the therapeutic dose change if your vitamin D is already high?

One of the known mechanisms of vitamin D therapeutics is higher production of cathelicidins and defensins. These are peptides (protein fragments) that have innate immune activity against infections of many kinds, including viruses. But the details of how they work is not fully understood, but we do know that they are “activated” by cleaving inactive proteins.

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51 If you are not at the top of your personal list of value trustworthiness, you might want to take a look at that. It’s been a central point in my work with parents of children with Down’s syndrome. When they have a negative and abusive experience with a pediatrician who knows nothing about TNI (targeted nutritional intervention for Down’s syndrome), who should they trust? I ask them, who cares the most about their child, the pediatrician or themselves. Their answer is always, “Us!” But their reluctance to trust themselves is based on a perception that they know less that their pediatrician about childhood health. But I ask them, is this a matter of knowledge about this particular treatment option? Or is it a matter of what’s in their child’s best interests? This usually makes sense to them. Their pediatrician’s ignorance of the treatment option basically disqualifies their advice. This also applies to physician ignorance of vitamins, metabolism, minerals, and functional medicine. So, who is the most trustworthy person regarding your personal values? It’s a core question worth taking seriously.

52 U H N Dürr, U S Sudheendra and A Ramamoorthy. LL-37, the only human member of the cathelicidin family of antimicrobial peptides. *Biochimica et Biophysica Acta – Biomembranes*. 1758(9): 1408–25, 2006. A review. Abstract: “Antimicrobial peptides and their precursor molecules form a central part of human and mammalian innate immunity. The underlying genes have been thoroughly investigated and compared for a considerable number of species, allowing for phylogenetic characterization. On the phenotypical side, an ever-increasing number of very varied and distinctive influences of antimicrobial peptides on the innate immune system are reported. The basic biophysical understanding of mammalian antimicrobial peptides, however, is still very limited. This is especially unsatisfactory since knowledge of structural properties will greatly help in the understanding of their immunomodulatory functions. The focus of this review article will be on LL-37, the only cathelicidin-derived antimicrobial peptide found in humans. LL-37 is a 37-residue, amphipathic, helical peptide found throughout the body and has been shown to exhibit a broad spectrum of antimicrobial activity. It is expressed in epithelial cells of the testis, skin, the gastrointestinal tract, and the respiratory tract, and in leukocytes such as monocytes, neutrophils, T cells, NK cells, and B cells. It has been found to have additional defensive roles such as regulating the inflammatory response and chemo-attracting cells of the adaptive immune system to wound or infection sites, binding and neutralizing LPS, and promoting re-epithelialization and wound closure. The article aims to report the known biophysical facts, with an emphasis on structural evidence, and to set them into relation with insights gained on phylogenetically related
And likely more importantly, that they are involved in the mechanism of autophagy.\textsuperscript{53}

While autophagy will have its own chapter, let me briefly put this in a context. Autophagy means “self digestion” and it is a temporary state when the body resets itself by digesting cellular components for the raw materials from which they are made. Amino acids, nucleic acids, fatty acids, minerals, etc. This digestion takes place on a widespread basis, digesting both intrinsic cellular components as well as invading viral systems, and digesting both functional proteins as well as cellular garbage. Because the functional systems are restored to full function when autophagy ends, autophagy is best analogized as a “spring cleaning” of the cells of the body, getting rid of the debris that has accumulated since your last autophagy episode.

The digested debris that is not restored includes viral enzymes, viral RNA and viral DNA.

And it also digests viral proteins that sequester selenocysteine and human proteins that contain selenomethionine.

It’s not just the association of vitamin D\textsubscript{3} levels with viral disease. There is also a correlation between vitamin D and total mortality.\textsuperscript{54} For more on total mortality, see page 42.

It is also true that there is a strong synergy between vitamin D and vitamin A (retinoic acid).\textsuperscript{55} Both are substrates for nuclear receptors that pair up to influence cell regulation, and specifically the integrity of epithelial tissues and proper immune regulation of antigen presentation at the mucosal border. This has a stabilizing and diversification effect on the microbiome and helps minimize gut-mediated inflammation.

The stability of the “barrier function” of the gut epithelial layer also involves zinc nutriture.
Here’s a recent summary of the evidence for vitamin D deficiency being causal in Covid-19.56

“Early researchers reported three striking patterns. Firstly, the innate immune system is impaired by vitamin D deficiency, which would predispose sufferers to viral infections such as COVID-19. Vitamin D deficiency also increases the activity of the X-chromosome-linked ‘Renin-Angiotensin’ System, making vitamin D deficient individuals (especially men) more susceptible to COVID-19’s deadly “cytokine storm” (dramatic immune system overreaction). Secondly, the groups who are at highest risk for severe COVID-19 match those who are at highest risk for severe vitamin D deficiency. This includes the elderly, men, ethnic groups whose skin is naturally rich in melanin (if living outside the tropics), those who avoid sun exposure for cultural and health reasons, those who live in institutions, the obese, and/or those who suffer with hypertension, cardiovascular disease, or diabetes. And thirdly, the pattern of geographical spread of COVID-19 reflects higher population vitamin D deficiency. Both within the USA and throughout the world, COVID-19 fatality rates parallel vitamin D deficiency rates.” 56

How Cholesterol Affects Vitamin D Status

One of the puzzling findings that has come out of China is the positive correlation of cholesterol with coronavirus survival. Why would those with higher cholesterol levels be more likely to survive?

The answer may be that cholesterol inhibits the final step in the synthesis of cholesterol via the Kandutsch-Russell pathway (see illustration on next page, the lower pathway in which 24-dehydroreductase goes first).57


That final step is the conversion of 7-dehydrocholesterol to cholesterol by the 7-dehydrocholesterol reductase enzyme.\textsuperscript{58}

When cholesterol levels are high, 7-dehydrocholesterol reductase is degraded by proteosomal activity (see the minus sign on the dotted arrow pointing from cholesterol to the enzyme), which allows 7-dehydrocholesterol to accumulate.

This feeds the vitamin D pathway.

When cholesterol levels are low, 7-dehydrocholesterol dehydrogenase actively is high, which converts 7-dehydrocholesterol into cholesterol.

So cholesterol acts as a switch, inducing its own synthesis when it is low and promoting vitamin D production when it is high. In other words, high cholesterol may mitigate the risks of sun-avoidance behaviors in China and seasonal swings in vitamin D levels by increasing the precursor for vitamin D and making the capture of UV-B more efficient.

In this illustration, the cholesterol-synthesis pathways are highlighted in pale blue and the vitamin D-synthesis pathways are highlighted in faint yellow. Where they overlap, blue and yellow mix to make light green, where 7-dehydrocholesterol is shared by both pathways. The active form of vitamin D is shown in red as two separate pathways, one that is accomplished in the kidney for body-wide distribution through the blood stream, and the other is restricted to local tissues needing to regulate their own vitamin D level. Breast, prostate, colon and pancreas tissues make 1,25-hydroxy-vitamin D.

Other tissues may be found to do so in future research.

**High-Dose Vitamin D**

Endocrinologist Dr. Joseph Prendergast used very high doses of vitamin D\textsubscript{3} (50,000 IU daily) for extended periods of time (many months) for reversing diabetes. He never had even one case of vitamin D toxicity from that high dose. However, his protocol specified two important elements, (1) as little calcium supplementation as possible, and (2) magnesium supplementation.

Because vitamin D improves calcium absorption efficiency, if you take calcium with it, you might absorb too much, especially if you take the kinds of doses commonly recommended by physicians who do not understand the true causes of osteoporosis and still think that it is merely a calcium insufficiency. Even doses of 500 mg of calcium can be toxic with high vitamin D dosing, and many popular sources have recommended 750 mg, 1000 mg and 1500 mg of calcium. And because magnesium “balances” calcium and is ten times more likely to be deficient, it must be added to the protocol for maximizing safety.

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\textsuperscript{58} The two pathways are different in the sequence of enzymatic steps. The Kandutsch-Russell pathway take place as 24-dehydroreductase step first, the unnamed step, then 7-dehydroreductase step last. Whereas the Bloch cholesterol-synthesis pathway (the upper pathway) takes place as “unnamed” first, then 7-dehydroreductase second and 24-dehydroreductase third.

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Postscript on Vitamin D Increased Mortality Studies

There are a few studies suggesting increased mortality risk from high levels of vitamin D, one for an increase in overall mortality,\(^59\) another for increased stroke and cardiovascular mortality\(^60\) (see adjacent illustration). It is important to understand (1) these data are borderline statistically speaking, below the level of confidence that the findings are not artifactual, (2) that these data are for people not exposed to coronaviral infection, and (3) that these data do not disentangle the clinical advice commonly given to the elderly patients to supplement vitamin D late in life, when mortality is intrinsically higher. This selects for highest mortality risk for the highest vitamin D dosages.

It’s pretty obvious that coronavirus infection would affect “total mortality” in a significant way were it to be included. Again, this raises questions that cannot be answered definitively, yet, about the unknown therapeutic value of vitamin D supplementation during a coronaviral infection.

Another meta-study found no such increased risk at higher doses (see red Garland et al. curve).\(^61\) This and other studies have shown a flat line at the higher doses. My opinion is that the apparent increase in risk on some studies is an artifact from failure to correct for a known confounding variable.

When looking at the risk factors for cardiovascular mortality associated with the lowest levels of vitamin D, the increase in risk was more than double for women and fully triple for men.\(^60\)

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\(^59\) M L Melamed, E D Michos, W Post, B Astor. 25-hydroxyvitamin D levels and the risk of mortality in the general population. *Arch Intern Med* 168(15): 1629-37, Aug 2008. “In cross-sectional multivariate analyses, increasing age, female sex, nonwhite race/ethnicity, diabetes, current smoking, and higher body mass index were all independently associated with higher odds of 25(OH)D deficiency (lowest quartile of 25(OH)D level, <17.8 ng/mL [44.4 nmol/liter]), while greater physical activity, vitamin D supplementation, and nonwinter season were inversely associated.”


\(^61\) C F Garland, J J Ki, S B Mohr, *et al.* Meta-analysis of all-cause mortality according to serum 25-hydroxyvitamin D. *Am J Public Health* 104(8): e43-50, Aug 2014. “Our findings agree with a National Academy of Sciences report, except the cutoff point for all-cause mortality reduction in this analysis was greater than 30 ng/mL rather than greater than 20 ng/mL.”
Further Optimization of Vitamin D Status

The most popular vitamin-D test gives you a number for vitamin D3. But vitamin D3 is not active as a hormone (i.e., a nuclear-transcription factor). It’s the 1,25-Vitamin D3 that is the active form. So if you really want to go the extra mile, get your 1,25-D3 measured as well. This will allow you to (1) compensate for more or less efficient conversion of the inactive forms to the active form by the liver and kidney, and/or (2) less efficient catabolism (inactivation) of the 1,25-D3.

In other words, the ratio of active 1,25-D3 to precursor D3 in your system may not be normal. If your ratio is high, you are (1) more efficient at activating D3 or (2) less efficient at breaking it down. Either way, you need less vitamin D3 supplementation to maintain the same health effects as other people.

If your ratio is low, you may need more D3 to maintain the best health effects.

Vitamin D Addendum: Around the World with Vitamin D

A review in *The Lancet* by Roger Bouillon\(^6\) summarized findings from around the world regarding vitamin D deficiency at two different levels of insufficiency, < 10-12 ng/ml versus <20 ng/ml. Since we have seen that both of these levels of vitamin D are associated with significant pathology, both must be considered deficient and/or insufficient.

<table>
<thead>
<tr>
<th>geographical region of study</th>
<th>gross deficiency cutoff @ 25-30 nmol/L (10-12 ng/ml)</th>
<th>clinical insufficiency cutoff @ 50 nmol/L (20 ng/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hilger, <em>et al.</em> (2014)</td>
<td>Global</td>
<td>7%</td>
</tr>
<tr>
<td>Herrick, <em>et al.</em> (2019)</td>
<td>USA</td>
<td>5%</td>
</tr>
<tr>
<td>Cashman, <em>et al.</em> (2016)</td>
<td>EU countries (adults)</td>
<td>13%</td>
</tr>
<tr>
<td>Arabi, <em>et al.</em> (2010)</td>
<td>Iran, Jordan</td>
<td>50%</td>
</tr>
<tr>
<td>Durazo-Arvizu, <em>et al.</em> (2014)</td>
<td>Ghana, Seychelles</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Mogire, <em>et al.</em> (2019)</td>
<td>African continent</td>
<td>18%</td>
</tr>
<tr>
<td>Zhang, <em>et al.</em> (2013)</td>
<td>China</td>
<td>~37%</td>
</tr>
</tbody>
</table>

More worldwide results...

India, preventive\(^6\) 62% severe disease with vitamin D above 30 ng/ml
85% severe disease with vitamin D below 30 ng/ml.

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Saarland, Germany, respiratory fatalities not from Covid-19 vitamin D higher than 20 ng/ml, 9.4% deaths vitamin D between 12 and 20 ng/ml, 13.7% deaths vitamin D lower than 12 ng/ml, 21% deaths.

Bari, Italy, preventive vitamin D greater than 30 ng/ml, 5% mortality vitamin D less than 10 ng/ml, 50% mortality.

Chicago, USA, effect of vitamin D on testing positive vitamin D greater than 20 ng/ml, 12% positive vitamin D less than 20 ng/ml, 19% positive.

Israel, preventive vitamin D greater than 30 ng/ml, 14.2% infected vitamin D less than 30 ng/ml, 28.6% infected.

Iran, prevention mean vitamin D levels of controls, 30 ng/ml mean vitamin D levels of Covid-19 patients, 19 ng/ml mean vitamin D levels of Covid-19 fatalities, 8 ng/ml

Burmingham, UK, preventive, healthcare workers tested for Covid-19 and vitamin D vitamin D less than 12 ng/ml, 72% developed Covid-19 vitamin D greater than 12 ng/ml, 51% developed Covid-19.

of BAME (black, Asian and minority ethnic) healthcare workers vitamin D less than 12 ng/ml, 94% developed Covid-19 vitamin D greater than 12 ng/ml, 52% developed Covid-19.

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69 A A Faniyi, et al. Vitamin D status and seroconversion for Covid-19 in UK healthcare workers who isolated for Covid-19-like symptoms during the 2020 pandemic. *medRxiv* posted October 6, 2020. doi.org/10.1101/2020.10.05.2006706 The secondary study of black, Asian, Indian and other more pigmented health-care workers showed clearly that skin pigmentation was the factor influencing vitamin D and Covid-19 symptoms, not affluence and culture. The dramatic increase in below-12 Covid-19 symptoms was not matched by an above-12 increase. It remained unchanged. By drawing a line at 12 ng/ml, only the relative percentages of that population shifted, with more partitioning into the lowest-D category. The depletion of the higher-D healthcare workers in terms of numbers would minimally alter the percentages affected by slightly lowered vitamin D status.
Turkey, preventive of 149 Covid-19 patients with mean vitamin D of 15.2 ng/ml of 47 “moderate” Covid-19 cases, mean vitamin D of 26.3 ng/ml, 2.1% died of 102 “severe/critical” Covid-19 cases, mean vitamin D of 10.1 ng/ml, 66.7% died.

St. Petersburg, Russia, preventive moderate Covid-19 illness, mean vitamin D of 16.7 ng/ml severe Covid-19 illness, mean vitamin D of 11.9 mg/dl fatal Covid-19 illness, mean vitamin D of 10.8 ng/ml.

A recent review brings all these pieces together for the benefit of clinicians and public-health officials alike. The comprehensive integration of all these initially seemingly independent factors into an aligned big-picture view makes vitamin D an essential aspect of both preventive and therapeutic care.

On the preventive side, a very recent study of almost 200,000 people (Quest Diagnostics database) found a positive correlation between vitamin D levels and the risk of a positive Covid-19 test result. These results were statistically robust with positivity-versus-D curves that were essentially parallel for latitude, sex, skin pigmentation and age.

What this means is that lower vitamin D levels impair the clearance of replicating virus.

And high vitamin D promotes viral clearance, likely through an innate-immunity mechanism.

The cost of premier-brand vitamin D₃ prophylaxis at 7K IU per day is 4¢ per day at Amazon prices. This is for better quality vitamin D and a dose that is 2.35 times higher than the one used in the Spanish-hospital study mentioned above (see page 35).48

71 N Vyas, S J Kurian, D Nagchi, et al. Vitamin D in prevention and treatment of Covid-19: Current perspective and future prospects. Journal of the American College of Nutrition published online 1 September 2020. doi: 10.1080/07315724.2020.1806758. Abstract: “Vitamin D deficiency (VDD) partly explains geographical differences in COVID-19 susceptibility, severity, and mortality. VDD among African Americans, diabetics, hypertensive, and aged populations possibly explain the higher death rate, aggravated by cocooning. Vitamin D is pleiotropic, mediating bone metabolism, calcium homeostasis, and immune functions, whereas VDD is associated with inflammatory reactions and immune dysfunction, predisposing individuals to severe infections. Vitamin D modulates innate and adaptive immunity via the expression of genes that code antimicrobial peptides (AMPs). And the expression of cluster of differentiation (CD)14, the co-receptor for epidermal toll-like receptor (TLR)4. AMPs stimulate TLR2 in macrophages, increasing the conversion of vitamin D into its active form by cytochrome P450 27B1. Antiviral properties of vitamin D-induced AMPs can shift the polarization of the adaptive immune response from helper T cells (Th)1 to the more regulatory Th2 responses that suppress immune over-reactivity by preventing cytokine storm, which is already demonstrated during the Spanish flu episode. Vitamin D induces antiviral effects by both direct and indirect mechanisms via AMPs, immunomodulation, the interplay between major cellular and viral elements, induction of autophagy and apoptosis, variation of genetic and epigenetic factors. The crosstalk between vitamin D and intracellular signaling pathways may operate as a primary regulatory action on viral gene transcription. VDD may increase the likelihood of infection with enveloped viruses, including retrovirus, hepatitis, and dengue. Global data correlates severe VDD with COVID-19 associated coagulopathy, disrupted immune response and mortality, reduced platelet count, and prolonged prothrombin time, suggesting benefits from supplementation.”
72 H W Kaufman, J K Niles, M H Kroll, C Bi and M F Holick. SARS-CoV-2 positivity rates associated with circulating 25-hydroxyvitamin D levels.
73 BioTech-brand vitamin D₃, 50,000 IU per capsule, one capsule per week, or 7,000 IU per day. That dose should be sufficient to get the vast majority of people above 30 ng/ml (75 nmol/L) but not above 56 ng/ml (140 nmol/L). I paid $29 for 100 capsules, which included shipping and California sales tax (10%). That’s $0.29 per capsule, or 7.15 cents per day.
In a French nursing home, about two out of three residents came down with Covid-19. Of those that did contract SARS-CoV-2, 57 had been given a bolus (one-time dose) of 80,000 IU of vitamin D₃ and nine had not. The vitamin D lowered the death-rate by two thirds, more than doubled the percentage of mild Covid-19 patients (those not needing oxygen or hospitalization), and more than tripled the number of residents experiencing no limitations of their abilities from their Covid-19 infection.⁷⁴

That’s 44¢ worth of vitamin D₃ to drop the death rate in 87-year-old seniors (mean age) from 55% to 18%.

**The Vitamin D and Vitamin A Connection**

Although functional medicine doctors know that vitamin and vitamin A are synergistic with each other for intrinsic biological reasons, governmental and public-health officials do not. As an example of the latter, consider the National Institute for Health and Care Excellence, which promotes the role that vitamin A plays in immune enhancement but dismisses vitamin D for that same purpose.⁷⁵

Both vitamin D and vitamin A are nuclear activating factors whose signals activate nuclear synthesis of innate immune proteins that play a necessary and essential role in viral defense. Not only are both necessary and essential to immune defense, the vitamin D receptor complex (VDR) pairs up with the vitamin A retinol receptor (RXR) to upregulate innate immune defense. In this aspect of their function, they have a one-to-one molecular relationship. Their joint function cannot be realized by one without the other, so why would a public-health organization take such an idiotic position?

They are profoundly ignorant of biological science.

Although I have targeted the UK in this instance, this profound ignorance is shared with US, Canadian, New Zealand and Australian public-health agencies.

So far, the studies of vitamin D efficacy in Covid-19 have not been graced with co-administration of vitamin A. Despite this huge gaping deficit, vitamin D continues to be the highest efficacy treatment for Covid-19 found so far. Imagine how effective it will be found to be when it is used with more intelligence.

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⁷⁵ D C Anderson and D S Grimes. Vitamin D deficiency and Covid-19. Letter to the Editor: Royal College of Physicians Clinical Medical Journal Nov 2020. doi: https://doi.org/10.7861/clinmed.Let.20.6.9. “There are now, to our knowledge, 14 studies that indicate the specific benefit in the COVID-19 pandemic of having a blood level of vitamin D greater than 30 ng/mL (75 nmol/L), and a very significant danger of death from infection if the blood level lies below 10 ng/mL (25 nmol/L).”
Here are some additional references for vitamin D and Covid-19, breast cancer, pregnancy, gestation and childbirth, diabetes, and non-alcoholic fatty-liver disease.


77 A Rastogi, A Bhansali, N Khare, et al. Short term, high-dose vitamin D supplementation for COVID-19 disease: a randomised, placebo-controlled, study (SHADE study). Postgrad Med J, 2020. doi: 10.1136/postgradmed-j-2020-139065. Abstract: “Background Vitamin D has an immunomodulatory role but the effect of therapeutic vitamin D supplementation in SARS-CoV-2 infection is not known.” This was a randomized, placebo-controlled study looking at viral clearance (i.e., going PCR-negative) in vitamin D-deficient patients (<20 ng/ml by test) with only mild Covid-19 symptoms. The vitamin D group (16 individuals) received 60,000 IU/day of vitamin D3 (cholecalciferol) for seven days to achieve blood levels of vitamin D above 50 ng/ml (my personal target range, see page 27). Ten of the 16 patients achieved that goal by day seven, and two more by day 14. The placebo group, 24 patients, got no vitamin D. 62.5% of the vitamin D group and 20.8% of the control group were PCR negative (p<0.018) before day 21. Fibrinogen levels significantly decreased with cholecalciferol supplementation (p=0.007) unlike other inflammatory biomarkers” (ferritin, CRP, D-dimer and procalcitonin).


Vitamin A

The first thing to understand is that beta-carotene is not vitamin A. Despite what you may have heard or read on the Internet, beta carotene is minimally converted to vitamin A in human beings. Furthermore, when very high doses of vitamin A are needed therapeutically, using beta carotene instead of vitamin A backfires.

The second thing to understand about vitamin A is that it is not as toxic as public health authorities state. Unless you are pregnant, short-term high doses of vitamin A are fairly benign.89

Let me put it in perspective. The recommended amount of vitamin A for adults is between 2,000 and 3,000 international units (IU) per day (700 micrograms in women and 900 mcg in men). People are told not to take more than 10,000 IU per day (the upper tolerable limit) to prevent toxicity, yet I take 15,000 IU per day on average, and have taken 25-30K units per day for extended periods of time. Regarding acute doses, I’ve taken a million units in a single dose without any “observable” toxicity.

Another perspective: hospitalized children with severe measles are routinely given 400,000 IUs per day for the first two days (more than ¾ of a million total units) to save their lives.90,91

What a difference between the official advice and the actual facts.

The third thing to understand is that vitamin A is cumulative. It is fat soluble and builds up with repeated doses. This means that increasingly higher doses can be taken for increasingly shorter periods of time. When dealing with viral diseases, very short-term is the norm. A typical acute viral infection might last three days to three weeks.

But when dealing with viral prevention, the time course it can vary between short term (an epidemic, which might last for a few weeks to a few months) and medium term (a seasonal pandemic where the virus

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89 If you are pregnant, or are capable of getting pregnant, there is a general consensus that 5,000 to 7,000 IUs of vitamin A is safe for fetal development. But the data are not coherent. Some studies draw the line close to 10,000 IU. So if you are female and menstruating, consider being safe regarding vitamin A by (1) taking less than 7,000 IU of vitamin A or (2) take steps to eliminate the chance of pregnancy until enough time has elapsed to have vitamin A tissue levels drop back to safe levels. This might take 1-3 months after vitamin A supplementation has ceased.

90 G D Hussey and M Klein. A randomized, controlled trial of vitamin A in children with severe measles. N Engl J Med. 323(3): 160-4, July 1990. Conclusion: “Treatment with vitamin A reduces morbidity and mortality in measles, and all children with severe measles should be given vitamin A supplements, whether or not they are thought to have a nutritional deficiency.”

91 G D Hussey and M Klein. Routine high-dose vitamin A therapy for children hospitalized with measles. J Trop Pediatr. 39(6): 342-5, December 1993. Abstract: “Measles is without specific therapy and remains important globally as a cause of childhood death. In controlled studies, high-dose vitamin A therapy (Hi-VAT)—with 400,000 IU vitamin A—has been demonstrated to markedly reduce measles-associated morbidity and mortality. We performed a retrospective study of the hospital records of 1720 children < 15 years of age who were hospitalized for measles, to determine the extent to which these findings, in research settings, are applicable to the case management of measles under conditions of routine hospital practice. The outcomes were studied of children hospitalized during two non-consecutive 2 year periods (1985-6 and 1989-90). A policy of Hi-VAT for all children hospitalized with measles was started during the intervening period. As compared with the group of children on standard therapy (n = 1061), children receiving Hi-VAT (n = 651) had a shorter hospital stay (mean 10 versus 13 days; P < 0.001), a lower requirement for intensive care (4.3 versus 10.5 per cent; P < 0.001), and a lower death rate (1.6 versus 5 per cent; P < 0.001). No adverse effects of Hi-VAT therapy were observed. We conclude that a policy of high dose oral vitamin A (400,000 IU) supplementation in measles provides benefits which are equivalent to those previously observed only in controlled research trials, that it is highly cost effective, and that it should form part of the routine case management of all children hospitalized with measles.”
persists for 5-8 months) or long term (a repeating seasonal pandemic). So your strategy will likely depend on the perceived risk of exposure.

The SARS coronavirus outbreak of 2002-2003 lasted for roughly six months. According to CDC data, the average influenza, coronavirus or rhinovirus outbreak lasts for roughly 30 days.

The fourth thing to understand is that vitamin A is very strongly associated with effective immune function in the scientific and medical literature going back almost a century. This is not currently controversial, although vitamin A use in treating immune disorders is often overlooked.

### Forms of Vitamin A

The fifth thing to understand is that vitamin A comes in multiple forms. Retinol reversibly converts to retinaldehyde, which irreversibly converts to retinoic acid, the active form of vitamin A. Free (unesterified) retinol (vitamin A) is immediately is immediately bioavailable but has a short shelf life. This is typically what you get from cod liver oil and from eating liver. This kind of vitamin A must be refrigerated, and taste-tested for rancidity maybe once a week, especially if the quantity ordered is large and takes months to consume. Some of these products are antioxidant-stabilized with herbal preservatives like rosemary oil.

Esterified (oxidatively stabilized) retinol comes in at least two commercial forms, vitamin A acetate and vitamin A palmitate. Acetate and palmitate are fatty acids that combine with the vitamin A to stabilize it against oxidation, and the acetate is more easily removed by the body to “release” the vitamin A. I take the palmitate because it is released more slowly. These vitamin A esters do not need refrigeration, but it’s a good idea to refrigerate them for longer-term storage as “emergency” antivirals for a future viral infection.

Free and esterified vitamin A also comes in liquid, emulsified and “micellized” forms. These can be very quickly bioavailable, to treat, for example, a sore throat. But one word of caution: they usually contain preservatives to protect them from bacterial or fungal overgrowth. If you see methylparaben or propylparaben on the label, those are the most common preservatives for liquid formulations. Some

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95 When storing any supplement in the refrigerator, it is important to keep in mind that the atmosphere in a refrigerator is damp. So if you have a substance that is hygroscopic (moisture loving), like choline, or moisture sensitive, like vitamin C, it should be packaged inside of one or more layers of moisture-proof plastic. This is because atmospheric pressure changes with the weather and the lids of most dietary supplements leak air. So every time the pressure goes up for good weather, some moist air leaks into the bottle, and every time the pressure goes down for bad weather, some internal air leaks out of the bottle. When using a bottle of vitamin A or fish oil daily, this may not be a big problem because you are using it up quickly and you will get more moisture contamination opening a cold bottle in the warm air outside of the refrigerator. But if your goal is to store a supplement for 1-2 years in the refrigerator, the extra plastic layers mean that dry air is leaking in and out of the bottle while in the refrigerator. One more caution: for any moisture-sensitive supplement that you take out of refrigerator storage for use, let it fully (!) warm up to room temperature before you take it out of the bag. Cold items can “sweat” when the humidity is normal, and you do not want that humidity to condense inside your opened bottle.
formulas will use potassium sorbate as preservative because it is “natural” and is compatible with a label claim of “no artificial preservatives added.” Some people are sensitive to such preservatives.

Many vitamin A products are formulated with beta-carotene. This is fine for those people who do not eat large amounts of vegetables, especially leafy green vegetation, and those taking minimum levels of vitamin A. But for high-dose vitamin A supplementation, beta-carotene is more than a potential problem. It does not scale graciously. So do not rely on the “Vitamin A” on the front of the label to disclose if it is 100% vitamin A, instead of beta-carotene or a carotene-A blend. Always check the back label to see what the ingredients actually are.

Humans as Aquatic Mammals (Vitamin A, Zinc, Copper and Selenium)

The aquatic-ape theory of human evolution has slowly progressed from original speculation and immediate denigration to scientific validation and partial acceptance. It has taken 60 years to make this transition, partly because Elaine Morgan first popularized the theory under a feminist manifesto (The Descent of Woman, 1972), which offended and entrenched the ego-driven male-anthropologist defense of the “noble hunter” theory, which was dominant at the time and still popularly regarded. But now that the key argument in the noble-hunter theory has been falsified, and fossil evidence found in support of the aquatic theory, it is now acceptable for some academic researchers to write and publish on the formerly forbidden aquatic hypothesis.

I’m putting this sub-chapter here, between vitamin A and selenium, because shellfish evidence is conspicuous at many of the archeological sites of these human ancestors that lived in the lake region of central Africa and the rift-valley region of eastern Africa, where the Nubian and Somalian African techtonic plates are separating (see photo). In the schema of the noble hunter theory, the shellfish finds were considered oddities or aberrations. But in retrospect, this evidence can be seen as a jigsaw-puzzle piece that is neatly fitting together with the dozens of other human-oddity pieces that are forming a coherent picture where extended human brain development was the driving evolutionary influence, and women, childbirth and extended human infancy were the factors governing the successful adaptations towards us becoming human.96

96 Humans have a broad set of aquatic adaptations not found to any significant degree in our genetic closest cousins, chimpanzees and gorillas, or even in our more-distant simian relatives. However, these aquatic adaptations are common in other mammals with successful adaptation to living in water (whales, dolphins, porpoises, otters, walruses, dugongs, manatees, sea lions, seals, hippos). Hairlessness (and hair direction), subcutaneous fat layer, a pronounced diving reflex, bipedalism, a hooded nose and a waxy skin coating at birth are among the traits found in humans and not at all in chimps and gorillas. Most of the most conspicuous traits, like hairlessness and subcutaneous fat, are much more developed in women than men. I find the diving reflex to be the most compelling example. The record for human freediving (no assistance with weights going down, and no air bladders for assisting ascent, and no scuba gear) is now below 120 meters (420 feet)! Some seaside cultures feature female divers who have an amazing skillset relating to age. While most athletic performance peaks at 25 years of age, many female divers continue to dive until they are in their 60s, 70s and 80s, with abilities comparable to their young peers. Many more human deviations from the ape norms and terrestrial mammal norms have been identified: we do not
The brain-development pattern of human beings is unique in several ways. One way is the extreme branching of dendrites in our brains. This means that human nerves are more complicated in their interconnections, which may be why we have sophisticated language abilities and conceptual skills. Some have hypothesized that these brain changes are caused by a simple genetic shift in the time devoted to late brain development (neoteny), which is argued to extend child-like learning abilities to later in life.

Another way this manifests is in larger brains, and more energetic brains. The human brain consumes roughly 20% of our total energy despite it being only 3% of our body mass. This is statistically anomalous when compared to other species.

A third way extended human brain development manifests is in earlier birth. Increased brain size means increased head size, which makes childbirth more difficult. As a result, human birth takes place while human brain development is still taking place. In other words, the pre-natal brain development that would normally take place in other species in utero takes place in humans after they are born.

Most newborn mammals have genuine motor skills when they are born. Horses learn to walk in hours. Newborn chimpanzees quickly learn to hold onto their mother’s fur when she walks or climbs. But human babies take roughly two years to complete the neural pruning (functional brain-cell apoptosis) that other mammals complete before birth.

Yet, oddly, human infants are born with an innate swimming ability. Human babies instinctively know to hold their breath when underwater. This is totally absent in our great-ape cousins.

To come full circle, what the aquatic theory of human development suggests is that our direct ancestors were adapted to diets rich in shellfish and coastal sea foods that are high in vitamin A, zinc, copper and selenium. And if we believe the current “coastline” migration theories based on genetic variations among humans living throughout the world, this nutrition preference is still wired into our genes.

Sadly, such foods are no longer in the diets of the vast majority of humans alive today. Just as we no longer live in a vitamin C-rich environment and experience pathology from insufficient vitamin C levels, most of us no longer get the vitamin A, zinc, copper and selenium that our distant and proximal ancestors did. Each of these nutrients has significant effects on chronic and acute viral diseases, so this is just one

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97 I believe this also makes us more susceptible to senility syndromes and Alzheimer’s disease. This view is consistent with the observations that ApoE protein variants affect Alzheimer’s disease risk, with the oldest genetic variant being higher risk and the newest genetic variant being of lowest risk.

98 This is why human children with Down’s syndrome (trisomy 21) end up with mental disabilities. When they are born and start breathing air on their own, the extra 21st chromosome turns negative ions (superoxide anion) into increased hydrogen peroxide, which drives neural pruning. This does not happen before birth because of maternal oxygen handling, which controls the way oxygen is transferred through the placenta to the developing baby. But at birth, the baby starts breathing 20%-oxygen air, which contains the negative ions that over-expression of SOD-1 turns into 50% more hydrogen peroxide. That hydrogen peroxide is a catalyst for increasing apoptosis, which causes over-pruning of the central nervous system—and brain damage. Contrary to what parents are told, babies with Down’s syndrome are not born retarded. They become retarded if the SOD-overexpression is not treated. Functional nutrition treatment can now prevent roughly 80-90% of that brain damage. For information on this, see https://www.ceri.com/downhome.htm or https://projectwellbeing.com/wp-content/uploads/2011/09/DSCollection.pdf. The first is the CERI website where this information was first reported, and the second is the Project Wellbeing website, where CERI’s Down’s Syndrome Collection is a free download.
more way that we have increased our viral susceptibility on a wholesale level by drifting away from Mother Nature.

Finding a dietary source of selenium may have been especially important for the success of our species because the lake region of central Africa and the rift valley are immediately adjacent to the largest selenium-deficient region of Africa (see page 56). To the west of the rift valley, the Democratic Republic of the Congo, located on the African tectonic plate, is highly deficient in soil selenium. But to the east of the rift valley are Tanzania and Uganda, located on the Somalian plate, with moderately high and moderate selenium, respectively.

At the rift-valley junction of the two plates, where the plates are separating as they grind past each other, there are deep fissures in the crust through with groundwater percolates, collecting in deep-water lakes that lie on top of the rift fracture. This scenario may have leveled the selenium field and allowed shoreline humans on both plates to partake in selenium sufficiency through seafood.

The rift-valley region is unique in many respects. The Somalian African plate has high mountains (Mt. Kenya and Mt. Kilimanjaro). The extreme northern end of the rift valley (the Denakil desert) is below sea level. The rift valley is lined with natural hot springs and volcanos, the latter of which provides regional volcanic soils that are often extremely low in selenium. Lake Tanganyika is the longest and second deepest lake on the Earth and holds 4500 cubic miles of fresh water. Lake Malawi at the southernmost end of the rift valley is the sixth deepest lake in the world and holds 2000 cubic miles of water. How these (and past) factors may have played a role in human evolution still remains largely unexplored.

Many native human cultures in Africa today follow dietary practices cultivating daily ingestion of selenium-rich nuts (see page 56). They also show intrinsically greater immune resistance to endemic ebolavirus in their local regions.

There is also a correlation between low selenium status and high prevalence of HIV infection prevalence African countries.
Selenium, the Trace Element of the Oxygen-Sulfur Family

Selenium is a trace mineral that plays an indispensable role in animal metabolism. It is not used by plants for nutritional purposes, so the selenium content of plants is an accident of the soil in which they were grown. For a practical example, Brazil nuts are frequently cited as a natural source for selenium, but there are multiple regions in Brazil where Brazil nuts are commercially harvested and only two of the five of them have high selenium in the soil.

Among tested samples of Brazil nuts from the Amazonas (northwest Brazil) and Amapá (northeast Brazil), the mean concentration of selenium in the nuts was 66 mg/kg and 51mg/kg, respectively. Very respectable. A single Brazil nut would contain approximately 200 mcg of selenium. Very respectable indeed!

But from the Roraima (northernmost) region of Brazil, the selenium in the soil is much lower. A kilogram of Brazil nuts from there have a mean selenium concentration of only 10 mg/kg. A single Brazil nut would have only 35 micrograms of selenium. But if you ate three, you’d get to the bottom end of the 100-200 mcg dose. And six is the recommended number of nuts by many “experts.”

In the Acre region (next to Amazonas and Peru) and Mato Grasso region (west central Brazil), soil selenium is quite low. Brazil nuts from those two regions were measured at 3.0 and 2.4 mg/kg of selenium, respectively. That’s only 11 mcg and 9 mcg of selenium per nut, respectively.

You’d have to eat twenty of these particular Brazil nuts to get to a 200 mcg selenium dose.

One nut giving 200 mcg versus twenty nuts?
That’s a 20-fold difference.

Where were your Brazil nuts grown?
Even within each region of Brazil, soil selenium varies hugely.99

Selenium in Redox Defense

Regarding viral disease, oxidative stress and cytokine storms, selenium is essential for maintaining antioxidant defenses and the redox-defense system. The illustration at right is part of the deep explanation of redox defense that takes place starting on page 161. But suffice it to point out here that selenium is a co-factor for four known glutathione peroxidases (with orange-to-green and yellow-to-green capabilities), and thioredoxin reductase (with yellow to dark green activity).100

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99 From Acre and Rondônia (southern Amazon), selenium ranged from 0.03 to 31.7 mg/kg (fresh), and from Manaus-Belém (northern Amazon), from 1.25 to 512.0 mg/kg. (Chang, et al., 1995). At the highest potency, one Brazil nut would give you approximately 1830 mcg of selenium. That could mean eating only one Brazil nut per week. Or every other week.

100 A comprehensive review of selenoproteins and selenoenzymes can be found here: K M Brown and J R Arthur. Selenium, selenoproteins and human health: a review. Public Health Nutrition 4(2B): 593-99, 2001. “The decline in blood selenium concentration in the UK and other European Union countries has therefore several potential public health implications, particularly in relation to the chronic disease prevalence of the Western world such as cancer and cardiovascular disease. Ten years have elapsed since recommended dietary intakes of selenium were introduced on the basis of blood glutathione
Green is where we live.

Selenium is also part of the redox-defense system by being necessary for the synthesis of the thyroid hormones (T4, T3 and T2) which regulate our metabolic rate.\footnote{It is our metabolic rate that is responsible for the amount of NADH that is synthesized (the sky-blue layer), which is half of the foundation of the redox-buffering system. Low metabolic rate is called hypothyroidism and hypometabolism in the West, and “cold” in traditional Chinese medicine. Corona and influenza viruses thrive in “cold and damp” conditions.} It is our metabolic rate that is responsible for the amount of NADH that is synthesized (the sky-blue layer), which is half of the foundation of the redox-buffering system. Low metabolic rate is called hypothyroidism and hypometabolism in the West, and “cold” in traditional Chinese medicine. Corona and influenza viruses thrive in “cold and damp” conditions.

Why selenium? If you look at the positions of oxygen, sulfur and selenium on the periodic table, oxygen is both a blessing and a curse. It is necessary for generating the huge amount of energy that warm-blooded animals need and it is essential energy for the large and complex central nervous systems of humans. But it is also toxic if it is not controlled efficiently.

Below oxygen is sulfur, which is the primary remedy for oxygen’s toxic forms (reactive oxygen species, ROS) and the free radicals produced from oxygen and radiation. Glutathione is the sulfur-based antioxidant and reducing agent that directly buffers the oxidative stresses of imperfect oxygen utilization. And, finally, below oxygen is selenium, which is used in very tiny amounts to defend and recycle the sulfur-based antioxidant systems. It’s a chain of decreasing oxidative stress and increasing antioxidant capabilities.

**Selenium, China, Africa and Ebola**

Selenium is a very potent catabolic-aerobic-acidifying element and antiviral influence (see page 160). It is strongly associated in the literature with the clinical course of viral outbreaks. Selenium plays the role of suppressing viral virulence when robust and enhancing viral virulence when deficient. Selenium status also affects host species crossover events where animal viruses alter genomic traits to become infective in humans.\footnote{This genetic instability of viruses is greatest in selenium-deficient regions [Beck 2010].} Because China has the largest selenium-deficient region of the world that is heavily populated with both humans and animals, let me start this geographical explanation with the following maps. On the left is China (from Huang, et al., 2017), with selenium ranked from lowest (less than 0.1 parts per million, darker peroxidase activity. Since then 30 new selenoproteins have been identified, of which 15 have been purified to allow characterisation of their biological function. The long term health implications in relation to declining selenium intakes have not yet been thoroughly examined, yet the implicit importance of selenium to human health is recognised universally.\footnote{And further, “It is well recognised that dietary selenium is important for a healthy immune response. There is also evidence that Se has a protective effect against some forms of cancer; that it may enhance male fertility; decrease cardiovascular disease mortality, and regulate the inflammatory mediators in asthma.”} And, finally, below oxygen is selenium, which is part of the redox defense system by being essential in the synthesis of the thyroid hormones (T4, T3 and T2) which regulate our metabolic rate.

Selenium is not only the coenzyme for iodinases which convert thyronine into T1, T2, T3 and T4, but also deiodinases which convert low-activity T4 into high-activity T3, and T4 into zero-activity reverse-T3 (rT3). This function enables tissue-level modulation of metabolic rate.

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\footnote{M A Beck, J Hardy and O A Levander. Host nutritional status: the neglected virulence factor. *Trends in Microbiology* 12(9), 417-23, 1 September 2004. (1) “In this review, we show that host nutritional status can influence not only the host response to the pathogen, but can also influence the genetic make-up of the viral genome.” (2) “Using two very different viruses (coxackievirus and influenza virus) as model systems we have shown that a host deficiency in either selenium (Se) or vitamin E, or an excess of iron, results in a change in the viral genome. In other words, specific, stable and reproducible viral mutations occur in the genome when nutritionally compromised animals are infected with these viruses; these mutations result in increased virulence of both” viruses. (3) “Hosts with normal nutritional status are susceptible to the newly virulent virus. This work represents a new area of research into the interaction of host nutrition and emerging infectious disease.”}
blue gray) to highest (greater than 0.5 ppm, in red). You can see that the northern, central and western regions of China are quite low. On the right is the USA (from USGS), with the lowest selenium regions (also < 0.1 ppm) in pale blue and the highest regions (> 0.75) in dark blue. Note that there are three dark-blue rankings in the USA map that are equal to or greater than the red ranking in China. In other words, the top USA regions are roughly half again as high as the highest China regions, and they cover vastly more territory that China’s limited red territory.

Despite the color confusion of the different keys (red versus blue), the gross selenium deficiency in China can be seen. The highest levels of selenium in China are above 500 parts per billion, and only in the extreme southern provinces, whereas in the USA, many areas are above 750 parts per billion, and the above 500 ppb areas spread across the northwestern plains states, which are the “breadbasket” (grain-growing region) of the USA. Selenium-rich wheat from the Dakotas and Iowa is eaten by residents of selenium-poor regions, providing those residents of the US with higher viral resistance than can be had in northern, central and western China.

US selenium status also affects other countries. English selenium status is falling because of decreased use of USA wheat in breadmaking. Wheat from the US’s breadbasket region (east of the Rockies, west of the Mississippi) has ten times as much selenium as wheat grown in the United Kingdom [Johnson, et al., 2010]. US wheat grown in eastern Washington and Oregon does not have this high selenium content.

Selenium is not just another antiviral nutrient. When it was assessed by Emanuel Revici almost a century ago, it was one of the four strongest catabolic-aerobic-acidifying elements on the periodic table (see page 160). As it turns out, poor selenium status is also strongly associated with the ebolavirus and ebola lethality in Africa.

The maps starting on the next page show the association. On the right are the ebola outbreaks from 1976 to the present (CDC) shows four different ebola strains, their distributions and the approximate number of cases. To the left is the selenium map of Africa (Hurst, et al., 2013) regarding selenium dietary intakes. The first thing I ask you to notice is the association of strains with their point of origin.

The most lethal Zaire ebolavirus (red dots on the CDC map) originated in the Democratic Republic of the Congo. This is the middle of the largest selenium-deficient region in Africa, that also includes the Central African Republic to the north and Zambia, Malawi, Zimbabwe, Mozambique and Botswana to the south. Next is the Sudan ebola strain (in blue), the outbreaks of which are immediately adjacent to the Democratic Republic of the Congo. The third (one green dot) is the Taï Forest strain, for which there is no known
selenium connection (or online source data). And the last is the Bundibugyo strain, which is named after the town in Western Uganda on the border with the Democratic Republic of the Congo.

Until recently, ebola outbreaks were confined to tribal communities in central Africa. This has largely compartmentalized ebola due to the insular nature of such communities. In 2014, multiple ebola outbreaks broke out in western Africa, in multiple countries, and in urban areas serviced by long-distance transportation systems. This has raised the specter of ebola outbreaks in non-African countries and the spread of ebola into the world population.

African Selenium Concentrators: Gabon Nuts, Dika Nuts, etc.

In Gabon, Africa, there is a tree nut similar to Brazil nuts that concentrates selenium from the soil. The rural communities use Gabon nuts traditionally, making a kind of Gabon-nut-butter which they consume in small amounts on a daily basis. This is not practiced in the urban Gabon communities where the ebola epidemic raged. What’s been found is a surprisingly high immunity to ebola in rural Gabon.103

While some researchers speculate that this Gabonese immunity to ebola is caused by fruit-bat saliva on tree fruits, others have pointed to the Gabon nut as a source of therapeutic selenium. In eastern Sierra Leone, a

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103 Pierre Becquart, Nadia Wauquier, Tanel Mahlakõiv, et al. High prevalence of both humoral and cellular immunity to Zaire ebolavirus among rural populations in Gabon. *PLoS ONE* 5(2): e9126, 9 Feb 2010. “To better understand Zaire ebolavirus (ZEBOV) circulation and transmission to humans, we conducted a large serological survey of rural populations in Gabon, a country characterized by both epidemic and non-epidemic regions. The survey lasted three years and covered 4,349 individuals from 220 randomly selected villages, representing 10.7% of all villages in Gabon. Using a sensitive and specific ELISA method, we found a ZEBOV-specific IgG seroprevalence of 15.3% overall, the highest ever reported. The seroprevalence rate was significantly higher in forested areas (19.4%).…”
hot spot for ebola infection, many asymptomatic cases were identified.\textsuperscript{104} Even back in 1976, when ebolavirus was first characterized, asymptomatic cases of ebola infection were seen. So this is not new information so much as it is formerly underappreciated information.

(+) add discussion of other nuts and traditional uses.

Evidence that selenium plays a key role in ebola pathology has been presented.\textsuperscript{105} The role that selenium may play in disulfide redox chemistry may be at the heart of it. The redox chemistry of the spike protein has been described by Harvard scientist Boguslaw Lipinski.\textsuperscript{106} The hydrophobic spike protein of ebola has similarities to the spike protein of coronavirus.

(+) Lipinski to be expanded with more details

(+) add content re thioredoxin and thioredoxin reductase.

**Selenium, Vitamin C and Acute-Induced Scurvy**

Unlike public-health officials, who do not talk publicly about selenium, vitamin C, BHT, viral virulence and host resistance, you can read about them here. Most of the information presented here is readily accessible to minimize your risks. This not only relates to the use of selenium, but also to the use of vitamin C and collagen therapy, for minimizing hemorrhagic outcomes (internal loss of blood and body

\textsuperscript{104} E T Richardson, J D Kelly, M B Barrie, et al. Minimally Symptomatic Infection in an Ebola ‘Hotspot’: A Cross-Sectional Serosurvey. *PLoS Negl Trop Dis* 10(11): e5087, 15 Nov 2016. (1) A blood sample was collected from 187 study participants, 132 negative controls (individuals with a low likelihood of previous exposure to Ebola virus), and 30 positive controls (Ebola virus disease survivors).” (2) “We identified 14 seropositive individuals not known to have had Ebola virus disease. Two of the 14 seropositive individuals reported only fever during quarantine while the remaining 12 denied any signs or symptoms during quarantine.” (3) The findings provide further evidence that Ebola, like many other viral infections, presents with a spectrum of clinical manifestations, including minimally symptomatic infection. These data also suggest that a significant portion of Ebola transmission events may have gone undetected during the outbreak.”

\textsuperscript{105} E W Taylor and C S Ramanathan. Theoretical Evidence that the Ebola Virus Zaire Strain May Be Selenium-Dependent: A Factor in Pathogenesis and Viral Outbreaks? *J Orthomol Med* 10(3-4): 131-8, 1995. (1) “A surprising range of *in vitro* and *in vivo* antiviral activities has been reported for various simple Se compounds, including inhibition of hepatitis B in humans, influenza virus in culture, and retroviruses like the mouse mammary tumor virus and bovine leukemia virus (reviewed by Schrauzer and Sacher and by Taylor et al.). Recent work has also demonstrated the *in vitro* activity of Se compounds against the human immunodeficiency virus, HIV-1.” (2) “Along similar lines, it is of considerable interest that Ziegler has pointed out a correlation between high rates of endemic Kaposi’s sarcoma (KS) in African subsistence farmers and geographic regions in Africa where the soils are of volcanic origin. These include regions surrounding the entire East African Rift Valley and the Nigeria-Cameroon border. It is widely documented that low Se levels in plants and Se deficiency syndromes of livestock are common in areas with soils of volcanic origin: the Rift Valley is a typical example.” The Rift Valley is hypothesized to be the aquatic environment in which ancestors of man evolved, which suggests that the archæological findings of shellfish shells may indicate that the selenium of aquatic foods may have compensated for local terrestrial insufficiencies. See page 17.

\textsuperscript{106} B Lipinski - Can selenite be an ultimate inhibitor of ebola and other viral infections? *British Journal of Medicine & Medical Research* 6(3): 319-24, 2015. **Abstract:** It is known that the virulence of Ebola and other RNA enveloped viruses involves in the first step their attachment to host cell membranes. Following this initial step the virus enters the target cell cytoplasm by forming hydrophobic spikes that make holes in the membrane lipid bilayer. Formation of such spikes is catalyzed by the reduced form of viral protein disulfide isomerase (PDI\textsubscript{red}) thus initiating chain of disulfide exchange reactions. Consequently, hydrophobic protein epitopes become exposed, which in the absence of proper chaperones form hydrophobic ‘spikes’ capable of penetrating the host cell membranes. In this communication evidence is discussed showing that the chain of disulfide exchange events can be inhibited by a small redox molecule – sodium selenite. It is suggested that this inexpensive and readily available food supplement can be an ultimate inhibitor of Ebola and other enveloped viral infections.
fluids). It has been proposed [Taylor and Ramanathan 1995] involving the acute induction of selenium deficiency by massive synthesis of viral proteins incorporating selenocysteine residues. Acute selenium deficiency is associated with the formation of clots and obstruction of vascular and capillary systems. This suggests that selenium is not just a preventive but could also be a critical therapeutic agent for selenium-scavenging viruses. HIV has been identified as a selenium-mediated virus. See also their follow-up paper [Ramanathan and Taylor 1997].

108 In addition to the collagen mechanism, a direct effect on clotting has been proposed [Taylor and Ramanathan 1995] involving the acute induction of selenium deficiency by massive synthesis of viral proteins incorporating selenocysteine residues. Acute selenium deficiency is associated with the formation of clots and obstruction of vascular and capillary systems. This suggests that selenium is not just a preventive but could also be a critical therapeutic agent for selenium-scavenging viruses. HIV has been identified as a selenium-mediated virus. See also their follow-up paper [Ramanathan and Taylor 1997].
HIV infection to AIDS, it is possible that political pressures will force public policy to ignore the selenium connection and even interfere with effective therapy by quarantine of ebola cases and forbidding patient access to these modalities.

It is also the case that the US public health authorities still have a serious attitude about vitamin C from the insights of Linus Pauling, Ewan Cameron and Matthias Rath about (1) the common cold, (2) the non-cholesterol cause of heart disease, (3) the beneficial effects of vitamin C on cancer, and (4) the beneficial effects of vitamin C on habituation to opiates.

Although denying people reasonable choice in medical treatment is illegal by US law and by international treaty (the Helsinki Accords and the UN Universal Declaration of Human Rights), this has not stopped public health officials so far.

The recent emergence of ebola virus from rural African communities into urban settings, and the spread of cases outside of Africa, has made lipid-enveloped viral disease a more immediate threat in the minds of many. This “climate of fear” gives rise to popular support for tyrannical measures.

It is important to understand that the acute hemorrhagic viral diseases require two, simultaneous therapeutic strategies. To decrease viral virulence and increase host viral resistance with the antiviral self-defense strategies outlined in this book is only part of the therapeutic solution. Changing viral virulence takes time, and the therapeutic timeframe can be quite short with hemorrhagic fevers. The acute effects of such viruses on selenium status, blood clotting, vitamin C and collagen infrastructure can kill by fluid loss and internal bleeding in less than a day. If you are lucky, it might take a few days, or maybe a week. Therefore, it is critical to combine the antiviral therapies of this book with acute therapies like intravenous vitamin C administration and selenium supplementation, to immediately mitigate the collateral effects of hemorrhagic viruses on tissue and vascular integrity, so that people can live long enough to have their resistance increase and/or the hemorrhagic virus attenuate.

According to public-health officials, viruses and bacteria do not attenuate. They assert that virulence is hard-wired into the genes of viruses (and other infectious organisms), despite clear scientific evidence to the contrary, and compelling epidemiological evidence to the contrary. This faulty view can lead to drastic unintentional consequences if public-health officials make bad triage decisions, mismanage resources based on erroneous “scientific” knowledge, and impose quarantines that deny choice of efficacious therapy for treatments that work due to a mistaken belief that they do not work.

If you have the foresight to stockpile vitamin C and IV bags, do not leave such essential supplies in your doctor’s offices where they could be “confiscated in the public interest” during a declared “emergency” or

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109 Those HIV positive patients with lower selenium status (plasma Se < 85 mcg/liter) progressed to AIDS more rapidly and had higher mortality [see Baum et al. 1997 and Campa et al. 1999]. In Africa regions where selenium is plentiful, HIV infection appears to be well tolerated.

(+) insert case against HIV as the cause of AIDS? See www.duesberg.com in the meantime.

(+) discuss “official” character assassination of Peter Duesberg?

110 E W Taylor and C S Ramanathan (1995) argue that the DNA of ebola and other hemorrhagic viruses is known to carry abnormally large numbers of UGA codons, which code for the amino acid selenocysteine. Therefore, the faster the virus replicates, the faster selenium is depleted, and the greater the effect on clotting and bleed out.

111 If you do not know how to formulate IV drips or do needle sticks, you need medical personnel to administer high-dose vitamin C therapy. If you learn how to do IVs yourself, there is no need for access to medical personnel. If you also maintain a fresh supply of liposomal vitamin C, an oral alternative to IV ascorbate, there is less need for medical personnel up front.
a quarantine situation. Keep such supplies closely held so that you may use them more wisely than well-meaning-but-ignorant public-health authorities would.

You can always take such supplies with you to your doctor’s office for administration of vitamin C therapy for your acute viral infection, assuming that it has not been administratively outlawed.

And if vitamin C therapy is prohibited, you can make other arrangements without their knowledge or interference.

President Obama has already asked for authority to detain anybody with respiratory symptoms, regardless of whether there is any evidence of ebola. If such policy gets applied to you, your quarantine in a government-run facility will likely prevent therapy with vitamin C.

Who has a clue as to what President Trump might have done, or President Biden might yet do?\(^{112}\)

In some places, merely coughing in public can get you quarantined.

Quarantine and denial of treatment? It can’t happen here? This is exactly what happened to Allan Smith and his family in New Zealand regarding Allan’s case of swine flu. Swine flu is a lipid-enveloped viral disease, and in Alan’s case, it rapidly progressed to respiratory failure, induced coma and the full measure of life-support machinery. The hospital dismissed the family’s request to use high-dose vitamin C therapy, insisting that it did not work, could not work and would not be allowed in the hospital. Because the hospital’s plan was to “pull the plug” and “allow him to die,” the family challenged to ICU staff to cooperate with high-dose IV vitamin C therapy. Reluctantly, they agreed with the understanding that they were going to pull the plug in three days. In a day, his lungs began to clear. On the second day, a CT scan revealed that his lungs were filled with air. On day four, they pulled his tracheal tube. On day seven, they took him off his life-support machinery and he began to breathe on his own for the first time in many months.

You might think this was a turning point for the family relationship with the ICU. Sadly, no. The next day, a new “consultant” for the ICU team ordered Alan’s vitamin C stopped. \textit{Without informing the family.}

There was an immediate downturn in Allan’s formerly rapid improvement. The family noticed. They started asking questions and found out what had been done. Allan’s wife Sonia was abusively belittled by the consultant, and Allan’s son’s confrontation with the new consultant ended acrimoniously. But enough furor was raised to have the vitamin C resumed. So what did the consultant order? One gram of vitamin C twice a day.

This is the kind of intransigence that ego-driven scientists and doctors manifest—absolute certainty in the face of blatant evidence to the contrary.

In a non-medical context, this would be criminal conduct.

This kind of medical malfeasance is the standard of care in the USA today. It might have remained the standard of care in New Zealand were it not for the bad publicity generated when the story was broken by \textit{60 Minutes} New Zealand. The furor was so intense that a new law was passed bypassing medical objections to vitamin C and mandating that it be made available to patients in New Zealand.

\(^{112}\) President Biden has even deeper connections to pharmaceutical interests, public-health vaccination policy and Monsanto (recently purchased by Bayer). Look for further disclosures from legal actions against Bayer from Monsanto’s decades-long allegedly illegal manipulation of evidence in support of glyphosate and GMO foods. This scandal is history repeating itself, with Monsanto in the position of tobacco companies regarding harm from cigarette smoking, and burying evidence of what they did and what they knew, and when they knew it.
Those New Zealand doctors were merely following medical policy set in the USA. Please do not dismiss the likelihood that official ebola-quarantine policy in the USA will prohibit vitamin C treatments. I’d count on it.

**Selenium and Coronavirus**

A letter to the editor published in the *American Journal of Clinical Nutrition* documents the association of hair selenium status in cities with CoV-19 clinical outcomes in hospitals in those same cities. At the lowest selenium levels (0.1 mcg/mg), the “cure rate” was 21%. At the highest selenium levels (1.0 mcg/mg), the cure rate was 61%. The relationship between hair-selenium status of a city and its cure rate was suggested to be roughly linear, although a pseudo-logarithmic relationship seems equally plausible (see lower graph).

The graph at above shows the cure rate on the Y-axis and the hair-selenium concentration for each city on the X-axis, with circles positioned to representing each city’s data and the size of the circles representing the total number of patients in that city. The blue line is the fitted linear relationship of all the circles, and the blue area is the 95% “confidence interval” for that fitted relationship.

The hospitalization mortality rate for coronavirus infection in February of 2020 was halved by high selenium status (near 40% instead of near 80%).

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114 It is important to keep in mind that these figures are not for the general population but rather for those most seriously compromised people who were hospitalized in intensive care units.
A recent analysis by a group of German researchers shows how strong the correlation is between selenium and survival from Covid-19.\textsuperscript{115} The correlation was significant and consistent for both hospitalizations and for deaths, for both elemental selenium levels and selenoprotein-P levels.\textsuperscript{116}

In the graph at right, you can see that the selenium status of the deceased patients (red) was significantly more compromised than that of the surviving, discharged patients (blue). By comparison to a European population norm (green, the EPIC cross-sectional analysis of 1900 people), both groups of hospitalized Covid-19 patients were seriously selenium deficient, the surviving patients less so than the deceased patients.

More than 40% of the Covid-hospitalized population were severely deficient in serum selenium (below the 2.5\textsuperscript{th} percentile) and almost 40% were severely deficient in selenoprotein P. This makes selenium deficiency a serious biomarker for adverse Covid-19 outcomes.

The relationship of selenoprotein P and serum selenium to each other and to survival and death were quite similar, and quite linear. Either measurement serves well to assess selenium status regarding Covid-19 risks.

The German researchers went one step further in their data analysis, looking at the time data for selenium measurements and Covid-19 survival (see above illustrations). They may have stumbled upon a unique pattern suggesting that selenium status tracks the clinical course of the infection. Confirming their earlier finding that selenium and selenoprotein P tracked with each other, the same time-pattern was observed for

\textsuperscript{115} A Moghaddam, R A Heller, Q Sun, et al. Selenium deficiency is associated with mortality risk from Covid-19. \textit{Nutrients} 12: 2098, 2020. “The mortality risk from a severe disease like sepsis or polytrauma is inversely related to Se status. We hypothesized that this relation also applies to COVID-19. Serum samples (n = 166) from COVID-19 patients (n = 33) were collected consecutively and analyzed for total Se by X-ray fluorescence and selenoprotein P (SELENOP) by a validated ELISA. Both biomarkers showed the expected strong correlation (r = 0.7758, p < 0.001), pointing to an insufficient Se availability for optimal selenoprotein expression.” While this may be true, poor zinc status also adversely influences protein synthesis. So the possible confounding effect of zinc status is not reflected in these findings. “We conclude that Se status analysis in COVID patients provides diagnostic information. However, causality remains unknown due to the observational nature of this study. Nevertheless, the findings strengthen the notion of a relevant role of Se for COVID convalescence and support the discussion on adjuvant Se supplemenation in severely diseased and Se-deficient patients.”

\textsuperscript{116} Selenoprotein P is one of 26 known selenium-containing proteins that have been discovered so far. It serves as a storage and distribution protein for selenium nutrition. It may serve other purposes that have yet to be discovered.
both selenium and selenoprotein P. This pattern was increasing selenium and selenoprotein P levels with increased survival over time, and unchanging selenium and decreasing selenoprotein P for decreased survival over time.

There is considerable room for speculation in the above findings. One speculation is that the selenium increases or decreases based on the magnitude of oxidative stress for which selenium might be mobilized. Increasing oxidative stress as a survival risk factor is observed for most pre-existing conditions. This suggests that selenium might be “drawn down” by the most severe levels of oxidative stress.

Another possibility is that there might be a conditional selenium-depletion effect from Covid-19 that is parallel to that caused by HIV and ebolavirus. Although this has not been suggested yet for any known coronavirus, is a cited phenomenon for HIV and hemorrhagic viruses.

A third speculation is that rising selenium levels reflects greater protein turnover and increased likelihood of autophagy, which would be releasing selenium from selenomethionine catabolism and releasing or reusing selenium from selenocysteine residues of proteins broken down during autophagy. If this mechanism is active, selenium nutriture improves slightly over time. If this mechanism is not functioning, selenium reserves remain constant or become slightly depleted. This opens the door to the possibility of therapeutic repletion to raise selenium levels may be able to lower mortality risks for Covid-19.

Given the dysfunctional history of the NIH towards any selenium-based viral research, I can hope that other countries will follow-up on this excellent opportunity. It might also be useful to investigate whether or not zinc status or the clinical use of a zinc ionophore (hydroxychloroquine) might improve protein synthesis, which might include selenoproteins in general or selenoprotein P specifically.

The way in which the surviving and deceased patients overlap regarding selenium status in merely an indicator that selenium is merely one of many pre-existing conditions that influence susceptibility to viral disease. However, among those pre-existing conditions, it may be the least expensive to correct or optimize. If we consider 50 micrograms of elemental selenium as the recommended dose and 100-200 mcg as the optimal dose, the cost is 1-5 cents per day for selenite supplementation and 5-10 cents per day for selenocysteine or selenomethionine supplementation.
Selenium and Polio Vaccination

The role of selenium supplementation in the immune responses to polio vaccination was investigated. Three groups of people, each composed of eleven males and females, were given either placebo, 50 mcg or 100 mcg of sodium selenite per day for 6 weeks prior to receiving an oral, attenuated live polio virus vaccine, with supplementation continuing for an additional nine weeks. Several aspects of immune enhancement by selenium supplementation were noted: (1) increased interferon-gamma production, (2) earlier T-cell peak proliferation, (3) increased T-helper cells, (4) improved antioxidant defenses and (5) faster clearance of polio virus from their systems.

This study also noted decreased viral mutations in the polio genome as tracked by PCR testing, confirming in polio the parallel findings regarding selenium inhibiting mutations in lipid-enveloped viruses.

Polio is a protein-capsid virus and not a lipid-enveloped virus.

Selenium Supplements

Selenium is available as a dietary supplement in multiple forms.

Inorganic forms include sodium selenite (Na2SeO3) and sodium selenate (Na2SeO4). These would naturally be consumed in drinking water from springs in regions of selenium-rich bedrock, or from wells where selenium is found in the aquifer. These are hypoallergenic forms of selenium.

Organic selenium forms include selenomethionine and selenocysteine. These are selenium-containing amino acids where selenium has replaced the sulfur atom of methionine or cysteine. These are found in plant foods grown in selenium-containing soils, all animal-flesh foods, and selenium-fortified yeast cultures.

There are other selenium-containing substances that are found in foods that are not available in supplemental form. Selenoneine, the selenium-for-sulfur analog of ergothioneine, is found in bluefin tuna and other seafoods. It seems to be the major form of organic selenium in human blood cells in a fish-eating population on remote Japanese islands and the fish-eating Inuit populations of northern

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118 Yumiko Yamashita, Takeshi Yabu and Michiaki Yamashita. Discovery of the strong antioxidant selenoneine in tuna and selenium redox metabolism. *World J Biol Chem* 1(5): 144–50, 26 May 2010. “This selenium compound has strong antioxidant capacity and binds to heme proteins, such as hemoglobin and myoglobin, to protect them from iron auto-oxidation, and it reacts with radicals and methylmercury (MeHg).”

119 M Yamashita, Y Yamashita, T Ando, J Wakamiya and S Akiba. Identification and determination of selenoneine, 2-selenyl-Nα,Nα,Nα-trimethyl-L-histidine, as the major organic selenium in blood cells in a fish-eating population on remote Japanese Islands. *Biol Trace Elem Res* 156(1-3): 36-44, Dec 2013. Abstract: Selenoneine is the major selenium compound in fish muscles, and fish appears to be an important source of selenium in the fish-eating population. Selenoneine has strong antioxidant activity and a detoxifying function against methylmercury (MeHg) toxicity. Dietary intake, bioaccumulation, and metabolism of selenoneine have not been characterized in humans. A nutritional survey was conducted in remote islands of the Kagoshima Prefecture in Japan. To evaluate the potential risks and benefits of fish consumption for health, we measured concentrations of selenoneine, total selenium, MeHg, inorganic mercury, and polyunsaturated fatty acid (LC-PUFA) in the blood of a fish-eating human population. The erythrocyte, leukocyte, and platelet residues following removal of serum (cellular fraction) contained 0.510 μg Se/g, 0.212 μg selenoneine Se/g, and 0.262 μg Se-containing proteins Se/g, whereas the serum contained 0.174 μg total Se/g. Selenoneine was highly concentrated in the cellular fraction in a manner that was dependent on subjects' frequency of fish consumption. Concentrations of selenoneine were closely correlated with...
Quebec province. Selenoneine appears to have potent antioxidant and methyl-mercury detoxification activity.

But regarding commercial supplements, the choices are limited, with strong advantages and disadvantages. Inorganic selenite and selenate are acutely toxic and not cumulatively toxic, whereas selenomethionine and selenocysteine are cumulatively toxic and not acutely toxic. This is because selenite and selenate are quickly excreted, and the seleno-amino acids are retained in the blood and incorporated into proteins.

Selenite is quickly reduced and utilized fairly efficiently, whereas selenate is only slowly reduced. This makes selenite the more active supplement choice. Those with redox-buffering problems might be challenged by this rapidity. I also caution that little is known about the exact mechanisms by which selenium species are reduced and converted into, for example, selenium phosphate. It is known that hydrogen sulfide is overtly toxic when its concentration exceeds the levels used by the body for cell-signaling purposes. It is possible that a similar phenomenon might apply to hydrogen selenide ions.

Selenomethionine cumulative toxicity is highest because it is mistaken for regular methionine and ends up in proteins where it is not intended, sometimes with adverse influences on that protein’s function. Whereas selenocysteine is the organic form of selenium that is capable of being directly incorporated into selenoproteins, so on that level is a superior form of organic selenium.

While selenite and selenate are intrinsically hypoallergenic, seleno-amino acids are most economically manufactured by fermentations, which can be sold as fortified yeasts or yeast extracts. These can be highly allergenic. If you are yeast intolerant and you want organic selenium, make sure it says yeast-free on the label.

The dose of selenite causing acute toxicity varies from maybe 600 mcg to maybe 2500 mcg, depending on body weight and individual metabolic factors that are not well understood. Because of this, doses of only 100-200 mcg are recommended. For higher dosages, repeat dosing is recommended because selenite is not stored.

In 2008, selenium toxicity symptoms were observed in a group of patients taking a dietary supplement which listed 200 mcg of sodium selenite on the label but contained over 200 times that amount (40,800 mg).

concentrations of MeHg in the cellular fraction. Selenoneine is the major chemical form of selenium in the blood cells of this fish-eating human population and may be an important biomarker for selenium redox status.

120 M Little, A Achouba, P Dumas, N Ouellet, P Ayotte and M Lemire. Determinants of selenoneine concentration in red blood cells of Inuit from Nunavik (Northern Québec, Canada). Environ International 127: 243-52, June 2019. (1) “Selenoneine, a novel Se compound, is found in high concentrations in marine foods (and particularly beluga mattaaq) and the red blood cells (RBCs) of populations that consume them.” (2) “Simple associations between RBC selenoneine and other Se and mercury (Hg) biomarkers were assessed using Spearman correlations and linear regressions.” (3) Selenoneine comprised a large proportion of whole blood Se and RBC Se in this population. Age and sex-adjusted geometric mean RBC selenoneine concentration was 118 μg/L (range: 1-3226 μg/L) and was much higher (p = 0.001) among women (150.3 μg/L) than men (87.6 μg/L) across all regions of Nunavik after controlling for age, region, and diet.” (4) “RBC selenoneine was highly correlated with RBC Se (rs = 0.96, p < 0.001) and whole blood Se (rs = 0.89, p < 0.001), but only weakly correlated with plasma Se (rs = 0.13, p < 0.001).” (5) “consumption of market meats (g/day; β = -0.07) was negatively associated with RBC selenoneine.”

121 Some selenomethionine is converted into selenocysteine by the CBS enzyme (cystathionine beta-synthase). This reaction combines homocysteine (methionine without its methyl group) with serine (a non-essential amino acid) to make cystathionine, which is then broken apart on the other side of the sulfur atom by cystathionine gamma-lyase to yield cysteine and alpha-ketobutyrate (and ammonia).

122 I have heard two anecdotal reports of low-body-weight women who developed hypoglycemia reactions to 600 mcg selenite doses. This is considerably lower than most suggested toxicity ranges.
mcg, as measured). Symptoms of selenium toxicity were evident within two weeks of dosing from two patients who were taking twice the label recommended dosage.\(^\text{123}\)

This case of selenium poisoning affected more than 200 people spread out over ten states. The overtly toxic dose was 200-400 times higher than the 100-200 mcg typically recommended.

<table>
<thead>
<tr>
<th>Dose</th>
<th>Description</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>55 mcg/day</td>
<td>The USA recommended daily allowance</td>
<td>742 times less than 40,800 mcg</td>
</tr>
<tr>
<td>100 mcg/day</td>
<td>Steve’s regular daily dose</td>
<td>408 times less than 40,800 mcg</td>
</tr>
<tr>
<td>200 mcg/day</td>
<td>Steve’s current coronavirus-prevention dose</td>
<td>204 times less than 40,800 mcg</td>
</tr>
<tr>
<td>400 mcg/day</td>
<td>Steve’s active coronavirus-infection dose</td>
<td>102 times less than 40,800 mcg</td>
</tr>
</tbody>
</table>

The dose of selenomethionine and selenocysteine that cause chronic toxicity in humans is not known. But in monkeys, for 30 days of straight dosing, 150 mcg/kg of selenomethionine was barely tolerated. If we build in a safety factor of ten and a body weight of 50 kg, this would give us a rough top-end dose at roughly 750 mcg/day. The official “tolerable upper limit” is currently listed at 400 mcg/day, so that brackets the potential dose at one, two or three of the standard 200 mcg selenium capsules or tablets.

Because selenomethionine and selenocysteine are cumulatively toxic, doses are different than selenite.

<table>
<thead>
<tr>
<th>Dose</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>28 mcg/day</td>
<td>One capsule per week, Steve’s addition to the above selenite.</td>
</tr>
<tr>
<td>55 mcg/day</td>
<td>Two capsules per week at 200 mcg per capsule. The official recommended dose.</td>
</tr>
<tr>
<td>100 mcg/day</td>
<td>One capsule every other day. The possibly ideal daily dose.(^\text{124})</td>
</tr>
<tr>
<td>200 mcg/day</td>
<td>One capsule per day. The label-recommended dose by the supplement industry.</td>
</tr>
<tr>
<td>600 mcg/day</td>
<td>One capsule per meal: Steve’s protocol for fighting an acute ebola infection.</td>
</tr>
</tbody>
</table>

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\(^{123}\) J K MacFarquhar, et al. Acute selenium toxicity associated with a dietary supplement. *Arch Intern Med* 170(3): 256-61, 8 Feb 2010. “Structured telephone interviews were administered to all persons identified as having consumed the product. Of 227 consumers, 201 (89%) met the case definition for selenium poisoning. The remaining 26 consumers who did not meet the case definition reported no or mild symptoms. Illness onset extended from January 5 through June 11, 2008 (Figure 2). The median patient age was 54 years (range, 4–92 years) and 121 (60%) were female. One-hundred forty-nine (74%) were white, and 15 (7%) were black. At the time of the initial interview, 118 (59%) of the patients had sought care at private physicians’ offices, clinics, or emergency departments; 83 (41%) had not brought their symptoms to medical attention. One patient was hospitalized; none died.”

\(^{124}\) M P Rayman, K H Winther, R Pastor-Barriuso, F Cold, M Thvilum, S Stranges, E Cullar and S Cold. Effect of long-term selenium supplementation on mortality: Results from a multiple-dose, randomised controlled trial. *Free Radical Biology and Medicine* 127: 46-54, 1 November 2018. Conclusions: “A 300 µg/d dose of selenium taken for 5 years in a country with moderately-low selenium status increased all-cause mortality 10 years later. While our study was not initially designed to evaluate mortality and the sample size was limited, our findings indicate that total selenium intake over 300 µg/d and high-dose selenium supplements should be avoided.” I hesitate to call this finding definitive because: (1) the selenium was administered as yeast, a common inflammatory inducer of IgG, IgM and IgA antibodies, (2) the study used Kaplan-Meier data-analysis other than mortality rate, which obscures the trends for the first 5 years compared to the second and third five-year periods, (3) there was no assessment of the ongoing retention of selenium once the dosing ceased at five years, when the selenium accumulation would be expected to begin to drop as protein turnover continued without supplementation. But the data are fully consistent with a dose-threshold response between 200 mcg and 300 mcg per day for total mortality, cancer mortality and cardiovascular mortality. This suggests that selenium supplementation not be raised above the 100-200 mcg range on a sustained basis and that any “therapeutic supplementation” be reduced to baseline as soon as the therapeutic goal has been achieved. It also suggests that lower doses may be wise when on a diet naturally higher in selenium.
Optimization of Selenium?

From the review by C. D. Thompson,\textsuperscript{125} we have the following table which compares serum selenium levels with selenoenzyme activities:

<table>
<thead>
<tr>
<th></th>
<th>selenium concentration needed (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>prevention of Keshan disease</td>
<td>&gt;0.25</td>
</tr>
<tr>
<td>optimal activity of iodothyronine 5’ deiodinases</td>
<td>&gt;0.82</td>
</tr>
<tr>
<td>maximization of plasma glutathione peroxidase &amp; selenoprotein P</td>
<td>&gt;1.00–1.20</td>
</tr>
<tr>
<td>protection against some cancers</td>
<td>&gt;1.50</td>
</tr>
</tbody>
</table>

The two-fold variation in selenium optimization for different biological uses of selenium suggests that there may be a biological hierarchy for selenium resources. The Keshan’s disease amount represents a minimalist use of selenium, with iodinase activity being the highest priority, and glutathione peroxidase close behind.

When considering selenium dosage, it may be important to know secondary clinical effects on selenium status. During a hemorrhagic virus infection, high amounts of selenocysteine are being diverted into viral proteins at a rapid rate. Alternatively, selenium reserves may be challenged to a lesser extent by HIV virus and by arsenic toxicity, which opposes selenium nutriment.\textsuperscript{126} In areas like Bangladesh, arsenic toxicity is endemic,\textsuperscript{127} adding a selenium deficiency pathology to the existing arsenic toxicity. It may also be important to know the heavy-metal burden that can also interact with selenium. Certain heavy metals, especially mercury, have a strong affinity for sulfur and selenium.

Selenium: Necessary and Essential to Redox Buffering

Selenium plays an essential role in the redox-buffering and antioxidant-defense systems.


\textsuperscript{126} S Sah, A Vandenberg and J Smit. Treating chronic arsenic toxicity with high selenium lentil diets. \textit{Toxicol Appl Pharmacol} 272(1): 256-62, 1 October 2013. “Arsenic (As) toxicity causes serious health problems in humans, especially in the Indo-Gangetic plains and mountainous areas of China. Selenium (Se), an essential micronutrient is a potential mitigator of As toxicity due to its antioxidant and antagonistic properties. Selenium is seriously deficient in soils world-wide but is present at high, yet non-toxic levels in the great plains of North America. We evaluate the potential of dietary Se in counteracting chronic As toxicity in rats through serum biochemistry, blood glutathione levels, immunotoxicity (antibody response), liver peroxidative stress, thyroid response and As levels in tissues and excreta. To achieve this, we compare diets based on high-Se Saskatchewan (SK) lentils versus low-Se lentils from United States.”

\textsuperscript{127} R M Krohn, R Raqib, E Akhtar, A Vandenberg and J E Smits. A high-selenium lentil dietary intervention in Bangladesh to counteract arsenic toxicity: study protocol for a randomized controlled trial. \textit{Trials} 27;17(1): 218, April 2016. “The micronutrient selenium is a known antagonist to arsenic, promoting the excretion of arsenic from the body. Studies are in progress examining the potential of using selenium supplement pills to counteract arsenic toxicity. We are planning a clinical trial to test whether high-selenium lentils, as a whole food solution, can improve the health of arsenic-exposed Bangladeshi villagers.”
There are five different isoforms of glutathione peroxidase that allow different levels of antioxidant protection in different cells and tissues. There are three different thioredoxin reductases, one that operates in mitochondria, one in cytosol, and one in testes. Then there are a dozen letter-labeled selenoproteins that have been identified, half of which are now known to play a role in redox chemistries (selenoproteins F, M, O, P, V and W). This makes selenium nutriture a conspicuous aspect of redox competence.

Selenium is also used in reversing oxidative damage to proteins. Methionine residues are sensitive to oxygen radicals and form sulfoxides. The selenium enzyme methionine-R-sulfoxide reductase removes the sulfoxide from the surface of proteins damaged in this manner (see illustration at right).\(^\text{128}\)

But in some respects, selenium is important for its own utilization. The enzyme selenophosphate synthase is involved in the synthesis of all selenoproteins, redox-active or not.

Together, the selenium and sulfur systems coordinate with vitamin C to provide a disseminated redox-buffering pool upon which our lives depend. Any and all acute oxidative insults can cause that redox-defense system to become weakened, with pathological consequences. The cytokine storm from Covid-19, acute poisoning from mercury or rotenone, and acute radiation poisoning are examples of this kind of “collapse from without.” But equally problematic are “collapses from within” based on

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\(^{128}\) D E Fomenko, S V Novoselov, S K Natarajan, et al. MsrB1 (methionine-R-sulfoxide reductase 1) knock-out mice: Roles of MsrB1 in redox regulation and identification of a novel selenoprotein form. *J Biol Chem* 284(9): 5986-93, 27 Feb 2009. doi: 10.1074/jbc.M805770200. Abstract: “Protein oxidation has been linked to accelerated aging and is a contributing factor to many diseases. Methionine residues are particularly susceptible to oxidation, but the resulting mixture of methionine R-sulfoxide (Met-RO) and methionine S-sulfoxide (Met-SO) can be repaired by thioredoxin-dependent enzymes MsrB and MsrA, respectively. Here, we describe a knock-out mouse deficient in selenoprotein MsrB1, the main mammalian MsrB located in the cytosol and nucleus. In these mice, in addition to the deletion of 14-kDa MsrB1, a 5-kDa selenoprotein form was specifically removed. Further studies revealed that the 5-kDa protein occurred in both mouse tissues and human HEK 293 cells; was down-regulated by MsrB1 small interfering RNA, selenium deficiency, and selenocysteine tRNA mutations; and was immunoprecipitated and recognized by MsrB1 antibodies. Specific labeling with 75Se and mass spectrometry analyses revealed that the 5-kDa selenoprotein corresponded to the C-terminal sequence of MsrB1. The MsrB1 knock-out mice lacked both 5- and 14-kDa MsrB1 forms and showed reduced MsrB activity, with the strongest effect seen in liver and kidney. In addition, MsrA activity was decreased by MsrB1 deficiency. Liver and kidney of the MsrB1 knock-out mice also showed increased levels of malondialdehyde, protein carbonyls, protein methionine sulfoxide, and oxidized glutathione as well as reduced levels of free and protein thiol, whereas these parameters were little changed in other organs examined. Overall, this study established an important contribution of MsrB1 to the redox control in mouse liver and kidney and identified a novel form of this protein.”
selenium deficiency, insufficiency of vitamin C or glutathione, loss of mitochondrial production of NADH and HADPH (reducing equivalents), or a simple deficiency of a key B-complex vitamin.

The redox pools are so fundamental to biology that the entire humoral (antibody-based) immune system is built upon their foundations. Loss of reducing equivalents from local oxidative stress activates the immune system and restoration of redox control is necessary for shutting down the immune system when the job is done. The reducing equivalents provided by glutathione and ascorbate improve immune function and decrease immune dysfunction (increased risk of cytokine storms). Interleukin-6, one of the central cytokines involved in cytokine storms, is negatively associated with selenium status.\(^\text{129}\)

Selenium is deficient throughout much the world and where it is deficient, viral diseases are more deadly. How much of this is increased host resistance and how much is increased viral virulence is not known.

In conclusion, selenium is a necessary and essential nutrient for maintaining a deep pool of glutathione and ascorbate during conditions of high oxidative stress caused by a virulent viral infection.

For more information on the concepts of redox buffering and antioxidant defense, see Appendix A.

For information on the potential benefits of thioredoxin and the selenium drug ebselen (a glutathione peroxidase mimic) for Covid-19 infection, see Appendix D (page 194).

**Vitamin E**

Vitamin E is the body’s primary fat-soluble antioxidant. This is of primary importance because it is the membranes of the body that contain (a) the contents of a cell, and (b) the contents of subcellular organelles like the nucleus and mitochondria. So the compartmentalization of metabolic processes is membrane dependent. Membranes are made from fatty acids, cholesterol and phospholipids that reject vitamin C and glutathione. So vitamin E plays the role of carrying the reducing power of glutathione and ascorbate into membranes.

Without vitamin E, the lipids in membranes can oxidize, forming peroxides, hydroperoxides, epoxides, aldehydes and other byproducts that have widely harmful effects. Fatty peroxides and cholesterol oxides are mutagens, carcinogens and teratogens with strong inflammatory effects. “Rancid fat” is a popular term for describing the peroxidation of fatty acids, but less-well-known “rancid cholesterol” is equally of concern.

Vitamin E comes in two major categories, tocopherols (toe-coff-er-all)s and tocotrienols (toe-koe-tri-een-all)s, each of which come in four isomers, alpha, beta, delta and gamma. So this means eight different forms for “natural” vitamin E. A good case can be made that we need all of them.

Synthetic vitamin E (racemic alpha-tocopherol) is the most popular form of vitamin E. This is not because it is a good choice for health reasons, but because it is less expensive and has much higher numbers on the

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\(^{129}\) C K Tseng, C T Ho, H S Hsu, C H Lin, C I Li, T C Li, C S Liu, C C Lin and W Y Lin. Selenium is inversely associated with interleukin-6 in the elderly. *J Nutr Health Aging* 17(3): 280-4, Mar 2003. “The prevalence of selenium deficiency was 35.6% in men and 43.2% in women, respectively. After adjusting for potential confounders using multiple logistic regression analysis, interleukin-6 quartiles were significantly associated with selenium deficiency. Compared to the interleukin-6 quartile I, the adjusted odds ratios of having selenium deficiency for interleukin-6 quartile II, III, IV were 1.00(0.50–2.01), 1.24 (0.62–2.50), and 2.35(1.15–4.83), respectively. The increasing odds ratios for selenium deficiency in higher interleukin-6 quartiles revealed dose-response effects (p < 0.05). Moreover, multiple linear regression analysis showed that serum selenium was significantly inversely associated with interleukin-6 after adjusting for potential confounders.”
label. Vitamin E is rated in international units (IUs), which strongly favor alpha-tocopherol over the other tocopherols and tocotrienols. So consumers prefer many hundreds of units of alpha tocopherol over dozens of units of mixed tocopherols and tocotrienols in the same-sized capsule.

The evidence for vitamin E in Covid-19 is not yet clear. But for respiratory conditions and influenza, it is striking, and conditional. For example, vitamin E decreases the risk of pneumonia by 69% in those Finnish males who are minimally exposed to smoking tobacco and who exercised, yet increased risk by 68% in those with maximum exposure to tobacco smoke and who did not exercise.\(^{130}\)

The high concept for vitamin E is that it entirely depends upon vitamin C and glutathione for its protective effects. In situations where vitamin C and glutathione are compromised, vitamin E not only does not have protective effects, but increased risk of pneumonia and tuberculosis.\(^{131}\) Such a biphasic response is not unprecedented; increased risk is also seen with beta-carotene supplementation by heavy smokers.

This is why vitamin E is relegated to a subchapter instead of having a chapter of its own. Vitamin E in not a primary influence of viral risks. It depends on other antioxidants and reducing agents that either make it or break it. Nevertheless, vitamin E is a link in a chain of redox buffering that is important for the lipids of the body.

Each of the different forms of vitamin E have a slightly different antioxidant potential. As is the case with other antioxidants, effective management of free radicals depends on an ability (1) to react with free radicals of widely differing energies, (2) to effectively distribute (delocalize) the energy of the free-radical to make it less reactive, and (3) to pass the de-energized free radical down a chain of antioxidants to vitamin C or glutathione. This principle applies to tocopherols and tocotrienols.

The differing structures of tocopherols (upper) and tocotrienols (lower) are shown. The OH group on the left side top of the molecule is the active site, which can sacrifice an electron to a free radical, pairing up the electrons of the former free radical and becoming a low-energy tocopherol radical or tocotrienol radical in its place. The thin, dashed, gray line shows the delocalization zone of the vitamin E molecules. Four of the eight outlined atoms share the unpaired electron, which decreases its reactivity (danger) dramatically. This OH feature and the delocalization zone are common to all forms of vitamin E.

The first differences to notice are in the methyl groups immediately adjacent to the active OH group, here shown in red and blue. These methyl groups stick out next to the OH group and restrict how the OH group can be approached spatially. Alpha-tocopherol and \(\alpha\)-tocotrienol have a methyl group on both sides of the OH group, which is maximum protection. Beta-tocopherol and \(\beta\)-tocotrienol have just one protective

\(^{130}\) Harri Hemilä. Vitamin E and the risk of pneumonia: using the I2 statistic to quantify heterogeneity within a controlled trial. *British Journal of Nutrition* 116(9): 1530-36, 14 November 2016. “These findings refute there being a uniform effect of vitamin E supplementation on the risk of pneumonia.” These findings are parallel those where beta-carotene was found to increase cancer in smokers, but not non-smokers. See also: Harri Hemilä. Vitamin E administration may decrease the incidence of pneumonia in elderly males. *Clinical Interventions in Aging* 2016(11): 1379-85, 12 August 2016. “Among 2,216 participants who smoked 5–19 cigarettes per day at baseline and exercised at leisure time, vitamin E supplementation reduced the incidence of pneumonia by 69% (95% confidence interval [CI]: 43%–83%; 57 pneumonia cases). In this subgroup, vitamin E prevented pneumonia in 12.9% of participants by the age of 74 years. Among 5,253 participants who smoked \(\geq\)20 cigarettes per day at baseline or did not exercise, the incidence of pneumonia was 14% lower in the vitamin E participants (95% CI: -38% to +21%; 139 cases). One-third of the participants quit smoking for a period, of whom 27 got pneumonia. The incidence of pneumonia was 72% (95% CI: 31%–89%) lower in the vitamin E group, and this benefit was also seen among those males who smoked \(\geq\)20 cigarettes per day at baseline or did not exercise.”

\(^{131}\) Harri Hemilä and Jaakko Kapiro. Vitamin E supplementation may transiently increase tuberculosis risk in males who smoke heavily and have high dietary vitamin C intake. *British Journal of Nutrition* 100: 896-902, 1 October 2008.
methyl group. So do delta-tocopherols and δ-tocotrienols, but the one group is on the other side of the OH group. And finally, gamma-tocopherols and γ-tocotrienols have no methyl-group protection. This gives each form of vitamin E a different free-radical activity and specialty.

The second difference to notice is the vitamin-E tail. This tail is different between tocopherols and tocotrienols. The tocopherol tail is saturated (no double bonds) and the tocotrienol tail is unsaturated (with three double bonds). This is embedded in the chemical name: tri = three, and en = double bond, and ol = alcohol or OH group. These different tail elements slightly change the solubility of the tocopherols and tocotrienols towards different kinds of lipids, the tocopherols favoring aliphatic lipids like saturated fatty acids in cell membranes and the tocotrienols favoring association with polyunsaturated fats and cholesterol derivatives. These eight variations on the vitamin-E theme give vitamin E a broad range of activities towards free radicals that are needed for optimal protection.

The third things to notice are the asymmetric carbon atoms in the tail and the attachment of the tail. These carbon atoms have “handedness,” or right-handedness and left-handedness. With tocotrienols, this handedness applies only to the tail attachment. But with tocopherol, two of the tail carbon atoms come in right and left-handed forms. With natural vitamin E, all three of these centers are R-isomer (right handed), whereas synthetic vitamin E is “racemic,” meaning a roughly 50:50 mixture of R-isomer and S-isomer (half right and half left, respectively).

Synthetic E is also all alpha, with two methyl groups protecting the active OH group.

Since there is an appendix devoted to BHT discussion, let me take a minute to discuss the similarities of vitamin E to BHT (butylated hydroxytoluene).

You may be able to see profound similarities. Instead of methyl groups protecting the active OH group, tertiary butyl groups protect it to an amazingly greater degree. And instead of the oxygen atom opposite the active OH group, there is a methyl group on BHT that does not require the second ring to protect it from unwanted reactivity.

The net result is that BHT acts like vitamin E in protecting membranes from oxidizing free radicals.
Zinc and Copper

Zinc is an abundant trace element with vitally important roles in both innate and humoral immune function, antioxidant defense, and protein synthesis (an essential part of the healing response). It is required in higher amounts when tissues are actively growing (like during childhood and adolescence), which makes it indispensable in adults for the mucous membranes of the sinuses, mouth, throat, lungs, esophagus, stomach, intestines, colon and urogenital tracts, which continue to grow and divide at a higher rate throughout life.

Zinc “fingers” are responsible for sealing the layers of epithelial cells where they touch up against each other. This makes it particularly essential for the integrity of the gut and blood-brain barrier.

Recommendations for zinc for viral diseases affecting the respiratory tract are common. Copper, on the other hand, is virtually ignored in the same context. Viral diseases sequester copper and lower its bioavailability just as they do zinc bioavailability. Why not recommend copper and zinc when we know that (1) copper is part of the cellular antioxidant enzyme superoxide dismutase, (2) copper is essential for the extracellular superoxide dismutase, (3) copper is also essential for collagen maturation in the extracellular matrix, which determines tissue integrity, (4) copper is essential for electron transport in mitochondria (cytochrome C oxidase), (5) copper is positively correlated with thyroid hormone levels, and (6) copper and zinc are competitive with each other.

The answer to this is item 6: zinc and copper antagonism. If you take copper with zinc, it diminishes the absorption of zinc. And it is the increased influence of zinc on epithelial tissues that is the immediate therapeutic goal. What then to do about copper if you cannot take it with zinc and get the best zinc effect? Take copper transdermally where it will not compete for zinc in the gut.

Sequestration

So what we have is two trace elements that are both essential, both necessary for totally divergent aspects of immunity and healing, both of which are actively sequestered during an inflammatory state, and both of which are in some degree of sequestration in the general population. So what does this mean for treating viral diseases in general and Covid-19 in particular?

First, the baseline sequestration of zinc that a particular person experiences just prior to a viral infection limits their innate immune response. Zinc is necessary for vitamins A and D to activate protein-based cell-defense mechanisms. Zinc is also necessary to have a sense of (taste and) smell.

Transdermal Delivery

When zinc is taken as a lozenge, a throat spray, nebulized or suppository, some of it is being transdermally absorbed. This is the preferable route of absorption because it bypasses the liver first-pass effect. All blood from the intestines flows through the liver before flowing to the rest of the body. The blood from the sinuses, mouth, throat, lungs, skin and colon flow into general circulation before only a portion going through the liver. Sequestration of zinc, copper and iron take place in the liver. So when sequestration is unnecessary (like a viral infection or an allergy reaction), it makes sense to obstruct sequestration and maintain higher bioavailability of zinc, copper and iron. That means avoiding supplementing through the intestines, and instead supplementing through non-intestinal epithelial tissues. And since zinc and copper are antagonistic with respect to each other, it means sequestration is best circumvented by zinc to one
epithelial tissue and copper to another. So the respiratory tract gets priority for the zinc, and some other
tissue gets copper by default.

There is a pre-publication report of four cases of suspected Covid-19 cases where zinc was the primary
therapeutic modality where massive doses were used, and in one case, required.132 Case one started
treatment immediately after mild fever and muscle back pain symptoms developed from exposure to a
household member with direct exposure to a Covid-confirmed case. He took three 23 mg zinc citrate
lozenges, with nine more over the next 24 hours. Improvement began on day 2 and by day three his fever
had gone. He took eight lozenges per day for the next ten days.

Case 2 was the partner of case one. Seven days after exposure to the same household member, she
developed a mild fever, diarrhea and fatigue and took only one or two zinc lozenges per day. On day ten,
her symptoms dramatically worsened and she started taking one lozenge every hour and noticed that her
symptoms improved. The next day she again dropped back to two lozenges and her cough returned. She
increased her regimen to five per day and gradually improved over the next ten days.

This suggests that large
doses are needed for zinc
monotherapy.

Case three was a 41-year-
old woman healthcare
worker who did not start
zinc until late on day 9.

She tested positive for Covid-19 and was running a 101.5°F fever with severe body aches, a worsening
cough, a low 93-94% oxygen saturation, and eventually shortness of breath. She was started on high-dose
200 mg hydroxychloroquine on day 6 and got worse on day seven and eight. On day nine, in the evening,
she started zinc, 23 mg every four hours. She started improving the next day. She continued the zinc for
the next ten days until she was well.

This is an excellent example of a biohacking experiment that can be done by anybody in the privacy of
their home. Pulse oximeters and thermometers are quite affordable and can track the blood oxygen
saturation and body temperature of anybody concerned about identifying Covid-19 infections at the earliest
opportunity. In this case, body temperature preceded the drop in oxygen saturation by one day. But that
may not be the case all the time.

Case four went on for many weeks. She was a 26-year-old woman infected by a health-care worker.
Week one involved fever, cough and severe body aches. Week two brought on shortness of breath and

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132 Eric Finzi, MD, PhD. Treatment of SARS-CoV-2 with high-dose oral zinc salts: A report on four patients. *International
Journal of Infectious Diseases* 4311: 3 June 2020. doi: 10.1016/j.ijid.2020.06.006 In case 3, body temperature rose abruptly
on day four followed by a drop in oxygen saturation on day five. Her body temperature dropped from 101°F (38.3°C) on day
ten to 98°F (36.7°C) on day twelve, indicating a near simultaneous resolution of fever and hypoxia.
severe fatigue. Her fever broke by week three, but her cough, fatigue and body aches continued, and she slept more than 14 hours per day. After three weeks, she started ten 15 mg zinc lozenges per day for 14 days. The day after starting the zinc, her cough, body aches and fatigue started to improve. She felt fully recovered in two weeks.

Copper

Copper delivery through the external skin is now feasible with copper creams and lotions using copper in peptide form. You can even buy copper peptides and add them to your favorite skin-care products. When you do this, they become blue. And when you apply them, the blue color slowly disappears into your skin as the copper is absorbed. If the copper stains your skin, that copper is not being absorbed.

Copper is especially important for viral infections because (1) it is intrinsically antiviral by being classified as catabolic-aerobic-acidifying according to Emanuel Revici’s model (see Appendix A), and (2) it is mildly inhibited when taking large quantities of vitamin C, which is critical for virulent viral infections.

Lack of Specificity for Metallothionein and Ceruloplasmin

Unlike ferritin, which is quite specific for iron, metallothionein and ceruloplasmin are not particularly specific for zinc and copper, respectively. What this means is that ceruloplasmin can bind copper and ceruloplasmin can bind zinc (and iron). I do not know what this may mean regarding loading effects of copper on zinc bioavailability and zinc loading on copper bioavailability. Maybe this is not something to be concerned about. But it might be wise to be aware of the zinc-copper dynamic so as to consider how zinc supplementation might affect (1) zinc-copper absorption from the gut, (2) zinc-copper competitiveness on metallothionein, (3) zinc-copper competitiveness on ceruloplasmin, (4) zinc-copper balance in zinc-responsive tissues (like sinus, throat, lung and vascular epithelia, and immune cells), and (5) zinc-copper balance in copper-dependent systems (SOD, collagen maturation and melanin production from sun exposure).

Simultaneous optimization of both zinc and copper may require separation of the doses (like zinc with breakfast, copper with lunch and zinc with dinner) or when oral zinc needs to be continuous, like during an acute viral infection, either abstaining from copper for a limited period of time, or administering copper via a different route, like transdermal copper or copper suppositories.\(^{133}\)

Much is not known about the subtleties of the copper-zinc dynamic.

Serum Zinc Status as a Risk Factor

A new study has identified low serum zinc levels as a risk factor for Covid severity.\(^{134}\) The researchers noted that most of their severely ill Covid patients showed zinc deficiency, so they studied it.

As the graph shows, they saw a strong correlation.

\(^{133}\) J Osredkar and N Sustar. Review Article: Copper and Zinc, Biological Role and Significance of Copper/Zinc Imbalance. *J Clin Toxicol* S(3): 1, 2011. : One of the most common trace-metal imbalances is elevated copper and depressed zinc. The ratio of copper to zinc is clinically more important than the concentration of either of these trace metals.”

However, by measuring only serum zinc, they did not assess the contribution of inflammation to zinc deficiency. Zinc, copper and iron are sequestered (stored in proteins) with immune system activation by cytokine induction of metallothionein, ceruloplasmin and ferritin, respectively. While the researchers did measure ferritin and found it higher in the bad-outcome (severe) group, they did not measure metallothionein or ceruloplasmin, both of which can store zinc at the expense of serum zinc levels. They also did not measure serum iron, which could have shown the potential association of sequestration with low serum iron and/or low transferrin iron.

So the question remains as to whether the finding of this study are entirely due to sequestration, or whether they reflect a true body insufficiency or deficiency of zinc. This also ties to the potential therapeutic benefits of zinc ionophores and zinc supplementation regarding (1) the necessary dose of zinc needed to overcome deficiency or overcome sequestration, or (2) the route of administration of zinc, where oral zinc facilitates sequestration and sublingual and transdermal zinc postpones sequestration.

**Ionophores**

Many drugs are ionophores, expressing some kind of zinc or copper affinity. The role that such affinities play in drug effects is not well studied, but there is a fair amount of evidence that such affinities affect side effects. For example, ciprofloxacin (Cipro), levofloxacin (Levaquin) and other fluoroquinolones have a copper affinity that causes rapid loss of collagen maintenance in the joint capsules of the body. This leads to potentially catastrophic ligament and tendon damage.

Some of the coronavirus drug candidates are also reported to be ionophores for zinc. The most well known is probably hydroxychloroquine (and chloroquine), although many antibiotics and natural polyphenolic bioflavonoids likely have this property. It is possible that some of their therapeutic effects may be mediated by pulling copper out of ceruloplasmin or zinc out of metallothionein.

This is one more way the zinc-copper dynamic is complicated.

Vladimir Zelenko, a physician in New York, claims a 100 percent success in treating 700 coronavirus patients using a combination of hydroxychloroquine sulfate, azithromycin and zinc sulfate. The treatment takes less than a week and is easily affordable. Dr. Zelenko reports that shortness of breath resolves *in four to six hours.*

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Vein-Preservation Protocol

Repeated needlesticks in veins can cause scaring.\textsuperscript{136} In addition, needlestick technique can be difficult if the collagen quality is not high (i.e., the veins “roll” when puncture is attempted).\textsuperscript{137} The combination of oral vitamin C and bioflavonoids, and transdermal copper applied to the skin above the veins, can minimize both problems. The following illustration shows the dependence of collagen-crosslinking (tissue integrity or “toughness”) on copper.

Inside the cell collagen pre-procollagen is hydroxylated (oxidized to make it more water soluble and slippery) by iron-ascorbate Fenton chemistry. But in the extracellular space it is copper-ascorbate Fenton chemistry that converts some lysine and hydroxylysine amine groups (NH\textsubscript{2}) into aldehydes (the “=O” on allysine), which then condense with unoxidized lysine and hydroxyline groups on adjacent tropocollagen surfaces to crosslink (interconnect) tropocollagen fibers end to end and side to side to make bundles.

The tropocollagen fibers in the collagen bundles are offset so that the end-to-end crosslinks are staggered within the bundle.

And, finally, the bundles are assembled into a mesh network within which the tissue cells are embedded.

\textsuperscript{136} The clinical import of this is most relevant to extended intravenous therapies. Chelation of lead out of bones, for example, may involve EDTA IVs administered weekly or monthly over years. Most IV use of vitamin C only goes on for maybe a few days to a couple of weeks, but the current state-of-the-art protocols specify four IVs per day to maintain the constancy of high tissue saturation of ascorbate anions. In hospitals, this is almost always administered via an IV drip bag where one needle stick accommodates dozens of infusions of vitamin C. But in outpatient clinics, each visit requires a separate needle stick. In one reported case of an extremely dedicated patient, more than 50 infusions of 50 grams of vitamin C were needed to resolve her intractable condition. The disease symptoms did not begin to abate until after 30 infusions. So there are rare situations where vein healing is of primary clinical concern.

\textsuperscript{137} I consulted on 93-year-old Lorraine Whiting’s case where she had just recovered from Alzheimer’s disease three months earlier and was then found to have been drinking lead-contaminated well water. Chelation therapy was begun. Her veins were rolling to such a degree that each needlestick was a trauma for Lorraine, the nurse and the physician. But topical copper cream solved that problem in less than a week. By alternating arms and applying copper to the arm not being stuck with a needle, the entire problem was handled in clinic at a cost of pennies per week.
This is called the extracellular matrix.

Both vitamin C and copper are required to convert lysine or hydroxylysine into their aldehyde analogs for crosslinking.

Too much crosslinking and the tissues get stiff and are not sufficiently flexible. Too little crosslinking and the tissues are floppy, loose and easily damaged by mild impacts. One of the classic signs of weak collagen is ease of bruising. But one cannot necessarily tell if the ease of bruising is caused by vitamin C insufficiency, or copper insufficiency. The “odds” favor vitamin C insufficiency because humans do not make C and because we consume the tiniest amounts of vitamin C. Furthermore, copper insufficiency is widely considered unusual, whereas zinc insufficiency is considered fairly widespread. That’s also one reason why zinc is such a universally recommended supplement for upper respiratory viral infections.

Since topical products are not designed for damaged tissue, copper creams and lotions cannot be applied to wounds, sores or needle punctures. So only apply it to adjacent skin areas that are fully intact.

For serial IV needlesticks, apply copper to the arm that is not being used for that-day’s IV needle stick and then switch sides for the next-day’s or next week’s needle stick. Rolling veins can firm up in 24 hours, but it might take multiple days to plateau.

Let’s now move from copper to vitamin C.
Vitamin C

This is likely the most difficult subject to cover in this entire book. And it’s not just because vitamin C’s role in metabolism is especially complicated. It is not. It’s because more human idiocy has been diverted into the myths about vitamin C than all the other topics in this book put together.

So I’m going to split this topic into two parts, this one, which will be a relatively simple guidance about what to do, and Appendix C where the science and politics will be explained with references to the scientific and medical literature, and governmental and NGO policies (and politicking). The first thing to clarify is that vitamin C (aka, ascorbic acid, ascorbate) is not a vitamin for the vast majority of animals on this planet. In other words, the role that vitamin C plays in human viral disease is special, and almost unique. In humans, vitamin C is a “bottleneck” in the redox modulation (and stabilization) of immune response. Because we can only recycle existing ascorbate, it is the “weak link” in the chain of immune command.\(^{138}\)

In our fellow mammals, vitamin C synthesis takes place at roughly 10-20 grams per day, scaled for our body weight. And in those fellow mammals, synthesis doubles when they get seriously sick with viral or bacterial infections. Why? Because vitamin C stabilizes the redox potential of the body outside of cells like glutathione stabilizes the redox potential inside of cells. And redox potential governs the way the immune system works. So it’s a mammal survival mechanism to have at least 10 grams of vitamin C daily, and to have more when the immune system is challenged.

Let’s talk numbers. We have 10-20 grams as the “mammal norm” and 50-100 mg as the “human” recommended daily intake. If we take the lowest mammal number and divide it by the highest human number, our human RDA is, at best, 1% of what our fellow mammals get.\(^{139}\) And during an infection, it drops to less than a half percent unless we double our intake.

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\(^{138}\) See Redox Defense System on page 34, Appendix A on page 48 and Appendix C.

\(^{139}\) Some readers may find it ironic that the RDA for vitamin C for laboratory primates and Guinea pigs is severalfold higher than that for humans. How could this be? Don’t we have the same basic relationship to vitamin C as they do? The answer to that last question is yes. However, it is illegal for a scientist to “abuse” laboratory animals, and not illegal for the government to abuse human beings. Lab-animal treatment is forced to a higher ethical standard. Partly, this is by animal-welfare groups. But it is also by scientists who do not want deficiency pathologies interfering with their fundamental research. But because human nutritional standards are just recommendations and not administrations, it’s not properly considered abuse. The scientists setting the RDAs are not giving them to any person, but they are giving RDAs to lab animals. So there can be no abuse in the former and can be abuse in the latter. As it turns out, the “standard” for setting RDAs for humans is not human welfare, which would be the case if the standards for humans were the same as for animals. To put it another way, for animals, the requirement is to prevent all signs of deficiency. For humans, the standard is to prevent one kind of deficiency. Why does this double standard persist? Animal welfare people do not care about how humans treat humans as much as they care about the way humans treat animals. And for good reason, humans have a choice, animals do not. But I think that scientists also do not care enough to do anything. In fact, sick humans make better subjects when you are not doing primary research but rather conducting drug trials for FDA approval. I also think a good case can be made that (1) nutritional welfare statistics for citizens and (2) government nutrition programs for children are judged relative to RDA numbers. Yikes! Can you imagine a school lunch program being proud of meeting 19% of this and 26% of that. Animal-welfare standards would show how bad it really is. Fact may be the standard for science, but perception is the rule for politics. For animals, even one kind of deficiency sign left unaddressed is a fact that defines animal abuse. For humans, any number of deficiency states are OK as long as (a) the primary one can be argued to be minimized by the RDA, and (b) there are drugs to treat the rest of the deficiency symptoms. It is telling that gorillas in zoos are not required to meet the nutritional guidelines applicable to laboratory chimpanzees and gorillas. And by adopting human food standards, zookeepers now have gorillas with heart disease. [http://internetwks.com/owen/gorilla3.htm].
One percent is why vitamin C is a problem for human resistance to infection.

It’s been many dozens of millions of years since we have lost the ability to make vitamin C and we have developed multiple antioxidant compensations, and very good compensations, but these are inadequate when faced with virulent infections.\(^{141}\)

And all of these compensations get weaker when we get older or suffer from chronic diseases or pre-existing conditions. The government and media will not tell you specifically that the primary risk factor for dying from coronavirus infections is collapse of the redox-buffering system, but it is true. It underlies (1) acute oxidative stress, (2) blood coagulation and clotting, (3) vascular inflammation, (4) lung fibrosis, (5) decreased zinc and copper bioavailability, (6) decreased integrity of the gut and blood-brain barrier, and (7) cytokine storms. If you want to understand why the NIH, CDC and WHO are not telling you about this, go to Appendix C (page 175).

Appendix A and Appendix C will also go into the technical basis of how the redox-buffering system operates to support the antioxidant defense system, but all that needs to be said here is that cellular energy production lies at the heart of it. Roughly 90% of the cellular energy production takes place in mitochondria, which are the cellular organelles that are the foundation of the redox-defense system, which is the foundation for the antioxidant defense system. When we fight infections, we use oxidation as a weapon against the invasion. We “activate” oxygen by using vitamin C, iron and copper. This is why mammals make more vitamin C when infected, why we need more vitamin C when we are sick, and why vitamin C gets depleted during infections if we do not supplement it.

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\(^{140}\) Uric acid is a major antioxidant in the human antioxidant-defense system. But like vitamin C it is a steady-state antioxidant that does not increase in proportion to the severity of oxidative stress during infection. So uric acid merely compensates for the low levels of vitamin C that occur in everyday life.

\(^{141}\) T C Hornung and H-K Biesalski. Glut-1 explains the evolutionary advantage of the loss of endogenous vitamin C-synthesis: The electron transfer hypothesis. *Evolution, Medicine, and Public Health* 1: 221-31, 28 August 2019. This paper offers a summary of four previous hypotheses, and the advancement of their proposed fifth hypothesis, that there is a survival value for losing the ability to synthesize ascorbate because oxidized ascorbic acid transports into human red blood cells via the glucose (Glut-1) transporter. They argue that the transfer of electrons (reducing equivalents) into red blood cells during times of restricted food availability is enhanced, yet oxidized vitamin C does not carry electrons to transfer. It carries no reducing equivalents. In other words, the electrons to reduce the Glut-1-transported oxidized C have to come from internally generated electrons. So this argument is specious. Oxidized vitamin C is efficiently absorbed by Glut-1 receptors on white blood cells, which reduce it to C for essential immune purposes. But, again, it is internal generation of reducing power within the white blood cell which accomplishes this feat. Again, the oxidized vitamin C carries no electrons into the white blood cell. The boom line of this Glut-1 adaptation is: the greater the oxidation of C in the blood, the greater the ascorbate inside of white blood cells, and the greater the immune defense that can be mounted. As I see it, their argument depends on two concurrent genetic adaptations, (1) the loss of vitamin C synthesis, and (2) the modification of the Glut-1 transporter. It also depends on the Glut-1 adaptation being first, which may not be the case, and might even be unlikely in the extreme. Having vitamin C being “pumped” into white blood cells in proportion to the degree of oxidation of vitamin C makes perfect sense to me. As part of their argument, they tested pig glucose transport and found that it does NOT transport oxidized ascorbate. But what they need to demonstrate is that either (1) the Glut-1 adaptation to oxidized ascorbate, or (2) the loss of ascorbate biosynthesis has survival value without the other. If anybody knows of further research in this area, please advise.
It is also why high-dose vitamin C is needed in the most severe infections. By “high-dose C,” I’m not referring to merely matching the 10-20 grams that mammals make, but doses well above that.142

Our political systems do not want us to know about vitamin C. Our scientists and mainstream medical professionals are cooperating with this political agenda. So is institutional academia. And what we now have as a result is an “epidemic” of cardiovascular disease in zoo gorillas.143 Like humans, gorillas and chimpanzees do not make their own vitamin C. As economically challenged zoos have increasingly substituted supermarket fruits and human vegetables for the more expensive fresh fruits and native African vegetables (for example, Aframomum melegueta), the incidence of cardiovascular disease (fibrosing cardiomyopathy) has increased. The scientists may write that the “cause of this is unknown,” but that is not actually the case. It is just an embarrassment that the veterinary scientists consulted by the zoos are too immersed in current-but-erroneous scientific beliefs about human cardiovascular disease to consider vitamin C insufficiency. The vitamin C “RDA” for gorillas and chimpanzees is many times higher than those for humans, but it is still below what those animals get from their native diet. And as I have pointed out in numerous places in this book, vitamin C insufficiency results in inadequate collagen maintenance. The cause is most definitely known.

While it is certainly possible that other collagen factors like copper and silicon may also be involved, it is likely that vitamin C plays the largest role. Supermarket fruit is picked before it is ripe, so that it can survive extended shipping and distribution timeframes (days to weeks). Unripe fruit also withstands the mechanical abuses and bruising in ways that ripe fruit dies not, which is of primary importance regarding consumer purchasing. Even though unripe fruit often has increased levels of vitamin C compared to ripe fruit, the levels of vitamin C start to decline when it is picked. So it is the delay between picking and eating that puts the gorillas (and humans) at risk for vitamin C insufficiency. And old fruit is the most affordable option for the zoos.

So these gorillas in zoos are suffering from similar vitamin C insufficiencies that humans are, and they are developing similar inflammatory consequences. The sad news is that this problem could be fixed by vitamin C supplementation that would cost less than 5 cents a day. Ignorance is expensive!

And it is cruel to gorillas to inflict our ignorance on them.
(+ ) check the vitamin C level of Aframomum melegueta or its close cousins.
(+ ) check the selenium content of Aframomum melegueta seeds.
(+ ) extract counteracts cadmium toxicity, possible selenium mechanism.

http://eprints.abuad.edu.ng/id/eprint/245 Afe Babalola University, Oyinloye, et al. Ameliorative potential of Aframomum melegueta extract in cadmium-induced hepatic damage and oxidative stress in male Wistar rats. 6(7): 1-6, 2016. ISSN 2231-3354.

(+ ) Seeds used as spice locally. “Ginger-like spice.”

Aframomum daniellii “Basted melegueta” Zingiberaceae, Fruit, 70.0 mcg Se per 100g of fruit (the only selenium assessment) no seed-selenium data.

142 High-dose vitamin C is minimally 20-40 grams per day, roughly equal to what mammals produce when infected, and 40-80 grams per day, twice that level. For acute hemorrhagic fevers, the most lethal viruses known, it might be double that again.

143 Cheryl Lyn Dybas and Ilya Raskin. Out of Africa: A Tale of Gorillas, Heart Disease... and a Swamp Plant BioScience Volume 57(5): 392-97, 1 May 2007. Abstract: “Captive western lowland gorillas (Gorilla gorilla gorilla) are susceptible to a heart condition known as fibrosing cardiomyopathy. Although the cause of the disease is unknown, the captive gorillas’ diet may be a contributing factor. Aframomum melegueta, an herbaceous perennial plant that gorillas in the wild consume with gusto, contains substances with powerful anti-inflammatory properties that may protect gorillas' health.”
Mountain gorillas live in Rwanda (27-45 Se), Uganda (68-81 Se) and the Democratic Republic of the Congo (27-45 Se), on green, volcanic mountains (volcanic soils are usually low selenium).

Lowland gorillas live in the forests of central and western Africa in Equatorial Guinea (68-81 Se), Angola (46-54 Se), Cameroon (68-71 Se), the Central African Republic (27-45 Se), Congo (68-81 Se), Gabon (82-260 Se) and the Democratic Republic of the Congo (27-45 Se).

A similar heart pathology is now affecting humans suffering from Covid-19 infections in Kenya, Germany, and Italy.

**Why the Professional Close-Mindedness?**

Medical doctors are not adequately trained to understand nutrition, metabolism and redox aspects of disease. Back 50 years ago, medical students received one lecture on nutrition during their eight years of schooling. 30 years ago, they were lucky to get one day of nutrition education. Recently, “progressive” nutrition education may involve a week of education, but it is taught by old-school professors who have the RDA-vitamin mentality. That mentality is “vitamins only enrich the sewer.”

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144 https://www.nation.co.ke/kenya/news/virus-leaves-recovered-patients-with-ailing-hearts-1909782 This may be more serious in Kenya because it is one of the lowest-selenium countries in Africa.

145 V O Puntmann, M L Carej, I Wieters, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (Covid-19). JAMA Cardiol, 27 July 2020. From the abstract: Cardiac magnetic imaging of 100 patients “revealed cardiac involvement in 78 patients (78%) and ongoing myocardial inflammation in 60 patients (60%), independent of preexisting conditions, severity and overall course of the acute illness, and time from the original diagnosis.”

146 R M Inciardi, L Lupi, G Zaccone, et al. Cardiac involvement in a patient with Coronavirus Disease 2019 (Covid-19). JAMA Cardiol 5(7): 819-24, 27 March 2020. “We describe a patient without a history of cardiovascular disease admitted to the hospital with COVID-19 and severe LV dysfunction and acute myopericarditis. From the discussion: “Our main findings are that cardiac involvement may occur with COVID-19 even without respiratory tract signs and symptoms of infection.” This case report was within the northern Italian “hot zone” for higher-than-average mortality.

147 This story is related by my father. I do not doubt his version of events, but when I related this to Dr. Thomas Levy, a MD and JD, he responded, “What lecture?”

148 Decades ago, Stanford University’s “progressive” nutrition coursework in their school of medicine was taught by Wallace Sampson, a notorious “quackbuster” who had long consulted for Quackwatch, an ad hoc consumer advocacy group, which mounted expert-witness testimony against nutritional, complementary and alternative medical practitioners throughout the USA for decades. As was discovered in court and publicized by a variety of online pundits, many of Quackwatch’s top expert witnesses were found (1) to have egregiously falsified their court-submitted credentials, and (2) to have engaged in false testimony. These were not just run-of-the-mill experts, riding on the coattails of serious scientists. They were the founders and supposedly chief officers and experts of the organization, which was actually run out of one of the founder’s basement or garage. Not that Sampson should be judged by the fraudulent company he kept, but he did testify under legal proceedings and make public assertions that vitamin C “caused” kidney stones, a claim that had no clinical evidence at the time he started making his claim, and which was substantially falsified long before he died. This was not the only way Sampson slanted evidence in his favor. He argued that nutrition beyond the RDA was fraudulent and attacked such medical practices as acupuncture, chelation therapy, homeopathy and functional medicine. He attacked as fraud such conditions as chronic fatigue syndrome. His sole focus on claims of fraud caused the Stanford course he taught to change its name from “Holistic Medicine” to a course in “analyzing false claims.” This is one of the major ethical failures of his career, that he did not apply the same criteria to “false claims” than he did to his own claims. In other words, for a claim to be true, evidence had to be overwhelmingly obvious to a skeptic (him), but for a claim to be false, all that was needed was the opinion of a skeptic (him). This has become the regulatory standard for the FDA as well, despite this being declared to be in violation of the First Amendment rights of free speech by the US Circuit Court. Multiple times. This false world view, that things are fraud until they are proven not to be fraud is, itself, fraud. This “assumption of fraud” has never been true, and it can never be true. A therapy either works or does not work, regardless of when it is found to work, or not work. Two examples discussed on other pages, (1) failing to sterilize one’s hands after dissecting a cadaver was a risk to women and their infants regardless
It may seem odd in this day and age that you can hear this idiocy said with a straight face by medical professionals. There are many thousands of published papers proving otherwise, and dozens of conditions that are known to require high-dose vitamins, for which these doctors have supposedly been trained. Nevertheless, the view is that those medical circumstances are extremely rare and that 99 out of 100 patients need only the RDA level of vitamins, and that they get this in food.

This thinking is blatantly false to fact. Needs are not a number on a piece of paper, they are determined by a clinical benefit to be obtained. If there is a benefit to be had, failing to provide it is a denial of need and not the absence of need.

With vitamin C, the same RDA-only thinking persists. Despite the fact that vitamin C requirements are determined by both metabolic and redox conditions, the thinking is that vitamin C is like all the other vitamins where only a small dose is needed to saturate the coenzyme sites on certain enzymes.

Vitamin C does not work this way. But because the medical stereotype of vitamins is this way, vitamin C is misunderstood.

And it’s not just vitamin C that gets thrown out with the bathwater. When bacteria with high thiaminase activity colonize the gut, the need for vitamin B1 goes from ten to a thousand times the RDA. And when aging interferes with the synthesis of intrinsic factor, the dose of B12 needed goes up by a factor of between ten and a hundred. We know beyond a shadow of a doubt that when vitamins are malabsorbed, destroyed, or inhibited by an antimetabolite, needs change drastically.

With vitamin C, the “destruction” of vitamin C is part of its catalytic role. As a coenzyme, the vitamin C molecule is only used once at the enzyme site. Following the successful enzymatic reaction, it is discarded by the enzyme complex to be replaced by a fresh vitamin C molecule to catalyze the next reaction. In enzyme terminology, this is a “substrate” role rather than a co-enzyme role. So the reliability of the enzymatic process depends entirely on the recycling of “used” vitamin C into fresh vitamin C, a process that occurs elsewhere in metabolism.

This dynamic process of vitamin C use in one place and recycling at another place is an essential aspect of understanding how vitamin C works and how it needs to be supplemented as a substrate to perform its vitamin function. This is atypical among vitamins. Ignoring this by treating vitamin C as “just another vitamin” is dysfunctional to the same degree as ignoring the need to change the oil in a car, or drink extra water and electrolytes when you are working hard and sweating profusely on a hot, summer day.

In the adjacent illustration, the “reduction of a reactive oxygen species” is the enzymatic function and the two different recycling paths have different outcomes. Under low oxidative load, the inner path dominates

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149 A substrate is a substance that is “acted upon” by an enzyme. In other words, the substrate goes in and something else comes out. Enzyme B converts substance A to substance C. Substance A is the substrate for enzyme B and substance C is the product of enzyme B. A coenzyme “acts upon” its substrate. The coenzyme is a part of enzyme B that actually catalyzes B’s function. A coenzyme would be like the blade in a hacksaw, or the spark igniter of your gas stove. Typically, vitamins like thiamine, riboflavin and pyridoxine (B1, B2 and B6) or metal ions like iron, copper, zinc, chromium, manganese or selenium get transformed into a reactive chemical structure (the coenzyme) that lowers the “activation energy” required to make a chemical transformation. Consider a pair of wire-cutting pliers as an enzyme. You cannot cut a wire with your bare hands, but with the pliers, your hand strength can easily cut the wire. A hammer is like an enzyme, and a nail is its substrate.
(see solid-black arrows). Under extreme load, the outer path dominates (see dashed and red arrows). The outer path allows vitamin C to be diverted from the recycling process and irreversibly destroyed.

When this is not understood, we can make bad decisions.

This substrate role of vitamin C is analogous to the way NADH and NADPH work as well. The therapeutic uses of high-dose niacin and niacinamide are also underutilized, for example, in treating schizophrenia and aging. Low NADH (co-enzymated vitamin B3) is one reason why elders are at high risk from Covid-19 infection.

Vitamin C, Covid-19 and Acute Respiratory Distress Syndrome

Rarely is vitamin C status reported in typical medical and scientific reports. But in one research letter, it was noted that 17 of 18 patients with Covid-19-associated ARDS had undetectable (<1.5 mg/L) vitamin C in their blood. And the one patient who had detectable vitamin C was 2.4 mg/L, roughly half of what is found in the non-supplementing general population.

How to Supplement Vitamin C

Vitamin C has two different sweet spots for supplementation.

There is low-dose sweet spot where oral intake does a great job at sustaining vitamin C at the typical concentration that we have when we are well, when we are eating a diet rich in vitamin C, and when we are unstressed.

Then there is a high-dose sweet spot where vitamin C levels can be sustained above the level that is normal when we are healthy but is therapeutic when we are not healthy. This can be accomplished with either oral supplementation or intravenous administration.

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150 Luis Chiscano-Camón et al. Vitamin C levels in patients with SARS-CoV-2-associated acute respiratory distress syndrome. Critical Care 24: article 522, 26 August 2020. https://doi.org/10.1186/s13054-020-03249-y This study’s methodology is barely able to detect scorbutic levels of vitamin C.
Just as other mammals make increased amounts of ascorbate when they are ill, we respond positively to increased C when we are sick. One of the fascinating observations made by physicians who have successfully integrated vitamin C therapies into their practices is that vitamin C “needs” predict the clinical course of an infection. When vitamin C absorption from the intestinal tract is increasing, the disease is also increasing. And when the intestinal absorption of C finally “breaks” and starts coming down, recovery is taking place.

First, an idea of what I mean by low, medium and high dose vitamin C.

<table>
<thead>
<tr>
<th>vitamin C dose range</th>
<th>oral use</th>
<th>intravenous use</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDI dose</td>
<td>0.06 gram</td>
<td>not applicable</td>
</tr>
<tr>
<td>low dose</td>
<td>0.5-1 gram</td>
<td>3-10 grams</td>
</tr>
<tr>
<td>medium dose</td>
<td>2-5 grams</td>
<td>11-24 grams</td>
</tr>
<tr>
<td>high dose</td>
<td>6-25 grams</td>
<td>25-200 grams</td>
</tr>
</tbody>
</table>

This is an important point because both scientists and reporters tend to say “high dose” when it’s not, just because it seems high relative to their low expectations. They just do not have the personal experience of how high vitamin C needs can go. For those who know ascorbate’s pharmacokinetics, and the amazing degree to which is recycled in healthy animals, high doses are those beyond physiological. High doses are those that are high enough to overcome whatever current level of oxidative stress is present and restore normal vitamin C-dependent functions. Those doctors who use high-dose vitamin C clinically increase the dose in proportion to the severity of the illness and the patient’s response to the starting dose.

There are many IV vitamin C protocols that specifically exclude patients with deficiencies of glucose-6-phosphate dehydrogenase, but practically, this is more of a precaution than a requirement. Oral vitamin C, even at high doses, seems incapable of overloading the red-blood-cell NADPH-based defense of its antioxidant defense system, which is particularly glutathione dependent. In fact, Dr. Paul Marik suggests that vitamin C is not actually contraindicated at all when low to moderate doses are given and that it actually helps the oxidative stress caused by restricted anaerobic NADPH generation.\(^{151}\) He cites clinical experience that IV vitamin C in the 1-10 gram range given four times per day results in “dramatic reduction of methemoglobinemia and hemolysis in G6PD patients.”

**Oral Vitamin C**

Low-dose vitamin C is measured in hundreds of milligrams for oral use (about three to ten times the RDA). At this level of oral vitamin C, the human system is not fully saturated and only small amounts of vitamin C are excreted in urine (typically less that 1 mg/dL). Vitamin C is sufficiently plentiful for handling.

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routine vitamin C needs for low-level collagen maintenance and low-level immune activity. Most vitamin-C-related diseases are prevented. Vitamin C test strips\textsuperscript{152} are efficient at testing these dose ranges.

But a few degenerative diseases resulting from insufficient vitamin C may not be prevented by such doses, or treated by such doses after years and decades of insufficiency.

And at this level of supplementation, virulent viral infections can drop the vitamin C levels in deep tissues low enough to have symptoms of scurvy (the classic disease of chronic vitamin C deficiency).

At medium doses of oral vitamin C, the body tissues become saturated and urinary excretion becomes moderate, significant enough to react with sodium selenite to form a red color.\textsuperscript{153} This is the point that vitamin C starts acting pharmacologically in adding significant redox equivalents to the redox-buffering system. This is where I chose to live, at 2-4 grams per day. This is roughly the half-way point (logarithmically) in the human-mammal vitamin C gap.

When dealing with a severe oxidative stress, like an acute viral infection, high doses of oral vitamin C may be needed. Or a resort to intravenous C.

Much of this need for intravenous vitamin C is the result of an acute viral infection getting out of control in people with insufficient vitamin C levels at the start of infection, which rapidly drop to deficiency for an extended period of time before vitamin C levels are corrected. The severity of oxidative stress and the extended period of immune dysregulation synergize to create system-level pathologies in multiple organ systems, which cascade into homeostatic failure. What starts out as a simple infection becomes a catastrophic condition requiring drastic measures when it is not addressed functionally at its earliest stages.

I predict that when this is eventually studied honestly, medium doses of vitamin C will be found to be fully sufficient in managing the severity of covid-19 infections for the vast majority of children, adults and seniors (1) if vitamin C levels are well above RDA at the start of the infection, and (2) if medium-doses of oral vitamin C are started within hours of the presentation of early symptoms or immediately following a positive viral test.

Given the entrenched political ideology that currently exists in this matter, it may take years before such honest research is conducted. However, each of you is free (1) to use this technology anonymously, (2) to tell your story about your use of vitamin C in treating your own cases, or your family and friends’ cases, and (3) to help swing the pendulum of public opinion to the point that a political reversal becomes a matter of re-election. This did happen in New Zealand, where professional anti-vitamin C beliefs were (and still are) as entrenched as they are in the USA, Canada and the UK today, so it could happen elsewhere.

As clinical evidence of this opinion, medium dose IV vitamin C is dramatically reducing mortality in US and Chinese hospitalized Covid-19 cases using the Shanghai protocol (6 grams of IV-C, 4 times per day). Even people with multiple pre-existing conditions are responding to this protocol. The Marik protocol of the Covid-19 Critical-Care Group uses half this level of vitamin C.

As it turns out, 12-24 grams of vitamin C is in the realm of what oral vitamin C can achieve in seriously ill people following a bowel-tolerance protocol.

\textsuperscript{152}http://store.riordanclinic.org/product/VPLCStrips.html for urine testing or for food testing.

\textsuperscript{153}Selenite-based vitamin-C test strips were removed from the market by FDA legal action.
Bowel-Tolerance Vitamin C Protocol

The bowel-tolerance C protocol, promoted by the late vitamin-C pioneer Dr. Robert F. Cathcart, allows the maximum oral vitamin C intake possible. Since vitamin C absorption from the digestive tract changes as the severity of the infection changes, this is a moving target. When you take more than your intestines can absorb, your bowel becomes over-hydrated and you experience loose stool (if subtle) or diarrhea (if excessive). The downside of flirting with diarrhea is pretty obvious. But when your life depends on maxing your vitamin C, such inconveniences and suffering are a bargain price for saving your or your loved one’s life.

The protocol is simple. Take enough vitamin C to just experience the slightest bowel-loosening effect—without experiencing diarrhea.

Simple to say, but in practice, it’s not so simple. If you are taking powdered vitamin C and dissolving it in water, measuring out teaspoons (4 grams) and 1/4 teaspoons (1 gram) can be tedious. So is weighing it. And if you are taking one-gram capsules or tablets, that’s the minimum increment.

Very tedious, but maybe infinitely better than trying to find a physician who will do IVs? Especially after you know you are coming down with symptoms and time is of the essence. Most physicians offering IV therapies were required to close down their offices in 2020 due to local Covid policies. When I say most, I almost mean all. I do not know of a single exception, although I’d like to hear that there was one.

Here’s my favorite protocol: Get a plain, ordinary kitchen timer that measures minutes. Digital or mechanical, it doesn’t matter. At the earliest signs of an infection, set it for 60 minutes, take a capsule of vitamin C, and start the timer. When the timer goes off, assess your gut, take another capsule of vitamin C and re-start the timer at a shorter time if stool is not loosening. Continue in this manner as the infection progresses.

Each time you take a capsule, consider whether you want to increase your dose (if no stool loosening effect) or lower it (oops!). If you want to increase your dose, decrease the time on the timer. By 5 minutes? By 10 minutes? Use your best judgement. Or flip a coin.

It’s a self-correcting process.

If you want to lower your dose, increase the time by five, ten or twenty minutes.

So all you have to do is (1) keep the timer with you, and (2) judge your bowel tolerance (stool softness).

When sleeping, resume the protocol when you wake up. If you wake up in the middle of the night and find that the timer has gone off, take a dose and re-start it. If you wake up with the timer still going, wait.

The typical viral infection increases in severity over time, until the fever breaks, and then trends downward as you are recovering. The same thing happens with vitamin C bowel tolerance; your bowel-tolerance dose will go up and up while you are fighting the infection, and then it will reverse when you have won the battle. So the pivot point is when the bowel-tolerance dose stops going up and starts going down. You stop setting the timer for shorter and shorter periods and start making them longer and longer.

If the time gets too short for your convenience, double the dose and double the time. Instead of taking one capsule every 17 minutes, take two every 34 minutes. If you are taking teaspoons instead of capsules, it’s the same protocol, double the teaspoons and double the time. If you want to increase the time by only 50%, increase your teaspoons by 50% (three instead of two). Just maintain the dose-to-time ratio when changing dose.
This protocol works regardless of the form of vitamin C, the amount of vitamin C or whether it is ascorbic acid or buffered vitamin C. However, bowel tolerance is also affected by the acid-loading effects from ascorbic acid and the mineral-loading effects from the sodium, potassium, magnesium or calcium in your “buffered” C supplement. I do not know any simple way to guide you in this respect. Electrolyte balance can be widely variable between different people. So it can take a bit of experimentation to figure out if buffering is needed, and how much is needed. But I can say with high certainty that high magnesium intake has its own bowel-loosening effect, and high calcium loading can have a significant constipating (anti-diarrhea) effect.

If you did not buy buffered vitamin C, you can make it at home. Adding baking soda (NOT baking powder!) to ascorbic acid converts it to sodium ascorbate in proportion to the amount of baking soda added. You will see fizzing as the acid and bicarbonate react. This takes only a minute. If you add milk of magnesia to ascorbic acid, some of the ascorbic acid will convert to magnesium ascorbate. But this may take many minutes. Instead of fizzing, you will see a change in opaqueness. Milk of magnesia is opaque white and magnesium ascorbate is translucent to transparent.

(+) insert vitamin C pharmacokinetics here)154

**Paying Attention to Your Taste Buds (or not!)**

Copper-deficient cows grazing in field of grass will figure out which of the many salt licks on the field has copper in it. We are trained differently. But one way we can “return to Mother Nature” is to re-engage our attention to our sense of taste. We all have some innate intuitive abilities that we can cultivate if we choose to accept the challenge. This is one way that you can judge your acid and alkaline loading from taking high-dose vitamin C. Ascorbic acid (the cheapest form of vitamin C) is my favorite form when taking medium oral doses. This may be because I have a slight alkaline stress at the blood level that is partly mitigated by taking acidic vitamin C. But when taking high oral doses, I have to partially buffer my vitamin C, which I do with sodium, magnesium and/or sometimes calcium. I let my taste preference/aversion guide me in making these decisions.

When I take pure ascorbic acid dissolved in a glass of water, I notice my reaction to the tartness. When I start having a negative reaction to the tartness, I add (1) baking soda, or milk of magnesia, and/or (2) blend my ascorbic acid with some sodium ascorbate, calcium ascorbate or magnesium ascorbate. The first approach is an acid-base reaction that can take seconds (the fizzing of baking soda) or many minutes (the clarification of milk of magnesia). The second approach, blending different vitamin C powders in a glass jar, may take ten minutes, including shaking the jar to mix the powders evenly, and labeling the jar to remind you what’s in it.

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154 J Lykkesfeldt. On the effect of vitamin C intake on human health: How to (mis)interpret the clinical evidence. *Redox Biol* 34: online pre-release. 23 May 2020. Abstract: “For decades, the potential beneficial effect of vitamin C on human health—beyond that of preventing scurvy—has been subject of much controversy. Hundreds of articles have appeared either in support of increased vitamin C intake through diet or supplements or rejecting the hypothesis that increased intake of vitamin C or supplementation may influence morbidity and mortality. The chemistry and pharmacology of vitamin C is complex and has unfortunately rarely been taken into account when designing clinical studies testing its effect on human health. However, ignoring its chemical lability, dose-dependent absorption and elimination kinetics, distribution via active transport, or complex dose-concentration-response relationships inevitably leads to poor study designs, inadequate inclusion and exclusion criteria and misinterpretation of results. The present review outlines the differences in vitamin C pharmacokinetics compared to normal low molecular weight drugs, focusses on potential pitfalls in study design and data interpretation, and re-examines major clinical studies of vitamin C in light of these.”
However, I have a word of caution that the taste buds might not be tuned to optimization of pH. During most severe viral infections, appetite is suppressed. This might be one reason to encourage sick people not to eat so they can get into ketosis and autophagy. Many vitamin-C clinicians have noted that their patients taking the most acidic ascorbic acid (pure ascorbic acid capsules) do better clinically than those taking buffered vitamin C. There are books and articles that blame this effect on mineral ascorbates having half the number of reducing equivalents as pure ascorbic acid, which is not true. Nevertheless, enough highly qualified observers have noted this buffering effect to warrant that there is some wisdom to it even if the accepted explanation is nonsensical.

Let me offer three hypotheses. Acidic ascorbic acid is more stable against oxidation, which is why it has a longer shelf life, and maybe why it might get less oxidized in the gut before it is absorbed. There may also be a general clinical benefit regarding bacterial and viral infection to gently acidify the body, as per Revici’s model of pro-viral and anti-viral terrain (see Appendix A, starting on page 153). I can also offer a third theory that the blood-pH difference between the deep tissues and the lungs which “pump” oxygen from the lungs into the deep tissues and carbon dioxide from the deep tissues to the lungs might be benefitted by a mild acidification influence.

In searching for IV ascorbate pH data, I found one report from the Riordan Clinic in which the pH of IV “sodium ascorbate solution” from Merit Pharmaceuticals (Los Angeles, California) was pH 5.5-7.0. This is significantly more acidic than both arterial and venous blood). McGuff Pharmaceuticals (Santa Ana, California) lists pH 5.5 to 6.6 for their Ascor-brand IV-C solution. Again, this is quite a bit more acidic than blood.

Given this evidence, it seems wise to make your vitamin C as acidic as you can tolerate at all oral doses. Keep in mind that the bowel-tolerance symptoms are also independently affected by the calcium-magnesium ratio. So if your bowel-tolerance dose goes down when you add more magnesium, consider adding more calcium ascorbate instead.

**Pros and Cons of Teaming Up**

Managing your bowel tolerance and taking vitamin C on a changing basis can become difficult when people are running high fevers or experience coagulopathy (thick-blood syndrome, see Nattokinase, page 114). High fevers induce several enzymes that interfere with mental competence. Aromatase is induced to convert pro-energy steroids (progesterone and testosterone) to estrogens (estrone and estradiol). These can cause or aggravate “senior moments” or “brain fog” events. This is all part of a natural survival mechanisms to spool-up your DNA and decrease protein synthesis to minimum levels, which defends your body from the oxidation caused by the immune system when fighting the infection. But even though it may be natural and a good idea strategically, it might get in the way of your supplement or medication schedule.

On way to manage this is to arrange a Covid-19 or flu partnership, where two or more people agree to assist each other in anticipation of the infection. The agreement is that each person agrees to be “on point” for the other, and to switch roles once the first is over the hump and the other gets infected. The mutual care involves many things that might be forgotten, regarding supplements, hydration, sleep, tracking body temperature, taking medications, cooking broths, showering, keeping logs, and paying attention to symptom development over time.

Indoleamine dioxygenase (IDO) is another enzyme that goes into high gear during a fever. IDO’s job is to lower the tryptophan level in the blood stream (and tissues) by half. This is a good thing because some bacterial infections thrive when tryptophan is high. But it’s a bad thing with viral infections because
(1) viruses do not depend on tryptophan, (2) because tryptophan is used to make the neurotransmitter serotonin, and (3) serotonin is used to make melatonin, the primary antioxidant of mitochondria.

High IDO can lower serotonin and increase dopamine-related behaviors. Normally, serotonin balances dopamine, but lowering serotonin can cause dopamine dominance. Dopamine can aggravate obsessive ideation, increase compulsive behaviors, and make people irritable, which can distract people from their strategic plans, or drive caregivers to distraction. In other words, dopamine can interfere with emotional equanimity. And maybe worse, it can interfere in the quality and depth of sleep, which undermines motor control and sound judgment on a level equal to alcohol (being drunk). This has been studied extensively by the California DMV regarding driving skills, which might be comparable to viral-coping skills.

**Rebound Scurvy**

Do not stop vitamin C abruptly. Always taper off.

I’ll give you two reasons.

There is a phenomenon called rebound scurvy that I have experienced personally, which may not actually exist as postulated in the medical literature. But it may cause adverse effects by a brief, temporary drop in vitamin C levels to insufficiency. This can have extreme consequences if the infection process is not fully ended.

The mainstream medical hypothesis is that high-dose C causes increased metabolism of vitamin C, which seems a bit fanciful. What is known is that oxidized vitamin C (dehydroascorbate) spontaneously hydrolyzes into diketogulonic acid, which is further metabolized into threose and oxalic acid. The first step in this process is not an enzymatic reaction, but rather a thermal reaction with water (hydrolysis). So it cannot be “induced” in the scientific meaning of the word. Or the medical meaning.

Nevertheless, other patients and some clinicians have spoken of it as a real phenomenon that occurs when high-dose vitamin C is suddenly stopped. I have thought of it as an “over-spending” problem, caused by a normally scarce and fully constrained resource that is artificially made abundant, and which goes back to scarcity too quickly, like what happens when people win the lottery, overspend the windfall of money, and end up in deep debt. It is probably more likely that there are critical delays in one or more human compensations to not having enough vitamin C on a regular basis, which may relax when vitamin C is plentiful and which need time to re-regulate when the abundance of vitamin C ceases.

In any case, sudden cessation of vitamin C intake may be risky.

**A case for consideration**

A possibly parallel occurrence in the use of intravenous (IV) vitamin C for treating covid-19 infection has been noted by the Covid-19 Critical Care Group in the USA (see page 93). In this one case, IV-C was stopped “prematurely,” which resulted in a rapid rebound of inflammation (see graph, next page).

This may or may not have been a rebound-scurvy event, but it was potentially dangerous to this patient, whose inflammation was tracked by ongoing C-reactive protein testing.

In previous patients, IV vitamin C was discontinued after CRP dropped without an adverse consequence. But in this case, stopping the vitamin C infusions resulted in a rapid rebound of CRP to even higher levels, indicating that inflammation had returned to pre-treatment levels and the Covid-19 infection was not fully controlled.
Regardless of the specific mechanism that may or may not have been involved, this is a dramatic lesson that vitamin C is powerfully anti-inflammatory and that it should not be discontinued until recovery is not only well underway but actually finished. It’s not worth the dollar or three that might be saved by ramping down the vitamin C intake faster than is indicated by, for example, bowel tolerance.

**The Klenner Protocols and Pre-Vaccine Polio Treatment**

The clinical use of vitamin C was pioneered back in the 1940s by Dr. Frederick Klenner, who used both oral and intravenous vitamin C for treating acute viral infections and fevers. Klenner used multiple and frequent oral doses of vitamin C (1-2 grams every 2-4 hours) and sometimes larger doses intravenously for more severe viral infections. Dr. Klenner’s results were not trivial; in the North Carolina Polio epidemic of 1948, 60 of 60 cases resolved with vitamin C without any neurological impairments, the vast majority in only 3-5 days.

But because the polio vaccine was developed during that same time period, Klenner’s work was ignored.

In another study, oral doses of only 500 mg were able to prevent paralysis in a placebo-controlled study of 70 children with poliovirus infection.

Klenner’s unprecedented clinical experience in resolving serious, debilitating and deadly conditions has been ignored by public-health officials for 70 years. Alternative and complimentary-oriented physicians who have adopted Klenner’s methods affirm that they work exactly as Klenner reported.

**Intravenous Vitamin C**

The high concept with vitamin C is that it is the human “weak link” in our response to the severe oxidative stress of acute infections and traumas. And because we cannot make it, it has to be obtained in the diet, by supplementation, or by intravenous infusion.

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155 Fred R. Klenner M.D., *Southern Medicine and Surgery*. 209: 1949. This paper also records efficacy in a host of other lipid-enveloped viral diseases including influenza, herpes zoster (shingles), herpes simplex, chickenpox, and six of six cases of encephalitis (two from viral pneumonia, one from chicken pox, one from mumps, one from measles and one from a combination of measles and mumps).

156 Abram Hoffer writes: “In the early 1950s, Dr. Fredrick Klenner began his work with megadoses of vitamin C. He used doses up to 100 grams per day orally or intravenously. In clinical reports he recorded the excellent response he saw when it was given in large doses. For example, polio patients given vitamin C suffered no residual defects from their polio. A controlled study in England on 70 children, half given vitamin C and half given placebo, confirmed that none of the ascorbate-treated cases developed any paralysis while up to 20 percent of the untreated group did. This study was not published because the Salk Vaccine had just been developed and no one was interested in vitamins. Dr. Klenner’s work was ignored.”
The degree of our need for vitamin C is what prioritizes these three dosage routes. When we are hale and hearty, our bodies do a good job of compensating for low vitamin C levels. In the intervening millions of years since we lost the ability to make vitamin C, we have increased our levels of uric acid, which acts as an antioxidant. And we recycle our vitamin C efficiently with glutathione and NADPH. This makes vitamin C something that can be obtained from diet, with careful food choices.\(^{157}\)

When we get sick, our bodies have a harder time. More vitamin C gets oxidized, and our bodies increase the recycling of vitamin C by the fever mechanism. We have also altered our glucose transporters on our white blood cells to absorb oxidized vitamin C (dehydroascorbic acid), to scavenge it for recycling by the white blood cells, which have the highest vitamin-C-recycling capabilities of all our cells. But the stress on vitamin C is still strong enough that supplementing vitamin C decreases the severity of our illness and shortens the duration of our illness.

When we get very sick, these compensation mechanisms fail. Vitamin C levels can no longer be maintained at the minimal levels needed to guide the immune system, maintain collagen quality, synthesize hormones and protect our healthy cells from the “killing” free radicals from the immune system, and the oxidative stress of atmospheric oxygen in our lungs.

At this point, vitamin C supplementation becomes a life-and-death issue. If vitamin C is allowed to crash to acute scurvy levels, we can experience a variety of life-threatening complications. The cytokine storm is but one.

Other mammals respond to illness by doubling their synthesis of vitamin C. But because we start with only 1% of the vitamin C that other mammals make, we have to increase our vitamin C supplementation by a factor of 200 to catch up to their levels. Since we are more efficient with our use of vitamin C, maybe that supplementation might be only 20x. But if the illness is especially severe, maybe our best chances are doses of 2000x. But either way, 20x, 200x and 2000x are big numbers, the latter two of which could not possibly be provided through diet.

With 200x, we can easily do it with oral vitamin C. But 2000x is 120 grams of vitamin C, which is likely far beyond the intestinal limit for vitamin C absorption. So the back-up plan for the most severe infections is intravenous vitamin C administration.

**Intravenous Vitamin C in New Zealand: The Allan Smith Family**

In New Zealand, a family member of a man stricken with swine flu knew about vitamin C treatment for viral diseases. Their father, Alan Smith, an Auckland farmer and rancher, had become infected with H1N1 influenza during a fishing trip and declined in health so rapidly that he had to be put on full life support in an induced coma. Family members asked that vitamin C be tried. The hospital refused. He stayed in a coma on full life support for months. Finally, the hospital wanted to “pull the plug” on his ECMO life support machine. The family objected because vitamin C had not been tried. After several back and forths, the hospital reluctantly agreed to begin high-dose IV vitamin C on a Tuesday evening with the understanding that they would pull the plug on the coming Friday.

\(^{157}\) With exposure to air pollution, industrial toxins, amalgam dental fillings, processed foods, medications, vegetables grown with modern agricultural practices (trace-mineral deficient), moldy-corn-fed beef, warehoused chickens, farmed fish and super-gluten wheat, the level of inflammation and oxidative stress that we have to deal with is far beyond “natural” conditions. It is arguable whether diet can still be considered sufficient.
On Wednesday, Alan Smith’s white-out lungs started clearing. On Thursday, a chest x-ray showed air in his formerly fluid-filled lungs. The hospital decided not to pull the plug. On day four, his tube was pulled. And on day seven, he was taken off life support to start breathing on his own.

The hospital administration’s reaction? Bring in an outside consultant to order the vitamin C stopped. Watch the 60 Minutes story\textsuperscript{158} on this case if you do not believe me.

Allan Smith’s rapid improvement stopped in its tracks. The family noticed immediately and started asking embarrassing questions. The hospital refused, again, to give vitamin C. The consultant verbally abused Allan’s wife and one of his sons was “escorted” out of a meeting by hospital security personnel. In the ensuing days, the hospital reluctantly agreed to put vitamin C back into his IV feed.

What they actually did was to give Allan only one gram of vitamin C twice a day instead of 50 grams twice a day. Fortunately, the family found out about this. They transferred Allan to another hospital closer to home, where again, vitamin C was refused. They hired an administrative-law lawyer, who notified the hospital that they were violating official citizen “guaranteed” health-care rights. By this time, Allan Smith was awake and cognizant of his situation. The family “smuggled in” vitamin C in a new, liposomal form, and their father recovered sufficiently to leave the hospital, preventing further medical interference in their father’s recovery.

This became a huge media sensation in New Zealand and generated so much political pressure that a law was passed to make vitamin C an accepted treatment that medical personnel cannot deny.

As of 2016, Allan was still alive and had resumed ranching. The 60 Minutes follow-up story shows him flying a plane over his ranch.

See Appendix C for more details.

As of 2017, vitamin C is not even being considered as a “possible” treatment for ebola by Western public health officials and the WHO bureaucracy. The WHO has a “list” of a dozen potential treatments in development, none of which is yet known to be effective. These are, in fair analogy, desperate “Hail Mary” forward passes in a political football game. And on the sidelines, sitting on the bench, is a proven treatment involving vitamin C, which cannot be considered for political reasons.

Neither can selenium, as a cheap, readily deployable preventive.\textsuperscript{159}

Do you want such people in control your public-health policy?

How about controlling your personal treatment choices?

\textsuperscript{158} You can find the 60-Minutes story on YouTube (http://www.youtube.com/watch?v=VrhkoFcOMII). And here’s an even better link to a 90 minute presentation in New Zealand in which Allan Smith tells his story and Dr. Thomas Levy speaks about the factual evidence for vitamin C therapy for acute viral diseases (http://www.youtube.com/watch?v=z1kD3BoIxnE). However, the best single information resource is a DVD movie / documentary about the story (Living Proof, 2-DVD set) that includes the 60-minutes story and the supporting information that 60 Minutes did not mention, including extensive commentary by Levy, who is not only a world-class medical expert on the subject, but a lawyer as well. Find it at http://www.medfoxpub.com for $7 plus shipping ($6 in the USA).

\textsuperscript{159} Selenite has successfully reversed the epidemic of Keshan’s disease, a virally associated cardiomyopathy endemic to a particularly selenium-depleted region of China. There is some speculation that a virus might be involved in the development of Keshan’s disease.


**Intravenous Vitamin C and Dr. Ryan Padgett**

Dr. Padgett was a first-line physician treating early cases of Covid-19. As a fit, former football player, he was not prepared for how hard he was hit. Evergreen Health hospital treated him with hydroxychloroquine, but he continued to worsen. His oxygen levels were so low that his heart was struggling, his kidneys were failing and he might have brain damage. So he was transferred to Swedish Health Services in Seattle, who put him on an ECMO machine, total life support, like Allan Smith’s case immediately above.

Indicators of inflammation were “astonishingly high,” suggesting that he was suffering from a cytokine storm. He was then given tocilizumab, a promising new anti-cytokine drug, and high-dose vitamin C after seeing reports that it might be beneficial. Within a week, he was showing signs of improvement. His inflammation decreased and his lungs started delivering oxygen above and beyond what the ECMO machine was providing. A week later, he was removed from life support, four days later his tube was removed, his sedation stopped, and he started to wake up.

There is no data available yet on the details of the vitamin C protocol used in his case. Some of the news stories about his case failed to mention the vitamin C part of his treatment.

He is still working to recover both physically and mentally. He is worried now about whether he will regain full cognitive function, noting moments of memory and attention problems. Still, he said, things have improved each day.

**The Covid-19 Critical Care Working Group Protocol**

In 2020, in the USA, an alliance of medical doctors began to use a vitamin C protocol in the treatment of Covid-19 patients. This protocol was developed by Chief of Critical-Care Medicine, Paul E. Marik, for treating sepsis patients. In mid-April, one doctor reported that he had yet to lose a single patient from the coronavirus. Marik reported losing only two patients, both of whom were over 80 years old and suffering from multiple co-morbidity conditions which were considered terminal.

Dr. Marik’s experience with sepsis patients was similar to the group’s with Covid-19. In an interview, he states that he went from losing 1-2 patients a week to losing none in a year after the sepsis vitamin-C protocol was introduced.

The protocol:

1. Intravenous methylprednisolone (a corticosteroid to suppress inflammation).
2. Intravenous vitamin C, three grams four times a day, 7 days or until discharged.
3. Full-dose heparin, low molecular weight (anti-coagulant blood thinner, preventing clots).
4. Optional thiamine, zinc and vitamin D.

**The Shanghai Protocol**

IV vitamin C was also used for Covid-19 infections in China. ¹⁶⁰  

*For treatment of light and ordinary patients* (by translation), “Supportive treatment needs to be strengthened to ensure adequate calories; pay attention to water and electrolyte balance to maintain internal stability; closely monitor patient vital signs and finger oxygen saturation. Give effective oxygen therapy

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measures in time. In principle, antibacterial drugs and glucocorticoids are not used. It is necessary to closely observe the changes in the patient’s condition. If there is significant progress in the condition and there is a risk of turning to severe, it is recommended to take comprehensive measures to prevent the disease from progressing to severe. You can use low-dose short-term glucocorticoids as appropriate (see the application section of glucocorticoids for specific programs). It is recommended to use heparin anticoagulation and high-dose vitamin C treatment. Low molecular weight heparin 1 to 2 sticks / day continues until the patient’s D-dimer level returns to normal. Once the fibrinogen degradation product (FDP) ≥10 μg / mL and / or D-dimer ≥5 μg / mL, normal heparin is used for anticoagulation. Vitamin C is 50 to 100 mg / kg per day, intravenously, and the continuous use time aims to significantly improve the oxygenation index. If the lung lesion progresses, it is recommended to use a large dose of broad-spectrum protease inhibitor ulinastatin 600 to 1 million units / day, until the lung imaging examination is improved. Once a ‘cytokine storm’ occurs, it is recommended to use intermittent short veno-venous hemofiltration (ISVVH)."

Intravenous vitamin C: 50-100 mg/kg = 2.5–10 grams per day

**For prevention and control of cytokine storms** (also translated into English), “It is recommended to use high-dose vitamin C and unfractionated heparin for anticoagulation. High-dose vitamin C is intravenously injected from 100 to 200 mg / kg per day. The continuous use time aims at significantly improving the oxygenation index. Dose broad-spectrum protease inhibitor ulinastatin, given 1.6 million units, once every 8 h, under mechanical ventilation, when the oxygenation index> 300 mmHg can be reduced to 1 million units / d. Anticoagulation can be taken. Treatment protects endothelial cells and reduces cytokine release. When FDP ≥10 μg / mL and / or D-dimer ≥5 μg / mL, heparin (3-15 IU / kg per hour) is used for anticoagulation. The first use of heparin. The patient’s coagulation function and platelets must be rechecked after 4 h. ISVVH is used, 6 to 10 h per day.

Intravenous vitamin C: 100-200 mg/kg = 5-20 grams for 50-100 kg person

According to Dr. Enquian Mao, Chief of Emergency Care at the Shanghai Public Health Center, they successfully treated 50 cases “moderate to severe” Covid-19 patients where all improved and none died.161 Dr. Mao also reported that there were no side effects from the IV vitamin C treatment.

Another, more novel way of introducing vitamin C into Covid-19 patients was suggested ten years ago: inhalational therapy.162 Given the easy availability of nebulizers (for the lungs) and misters (for the sinuses), this seems like a potentially fruitful concept.

An update on vitamin C use relating to the Shanghai protocol was recently published.163 It included an anecdote of a Chinese family situation where the elder female contracted Covid-19 and her five family caregivers took vitamin C (3-10 grams per day, divided dose) during their caregiving, with no personal protective equipment. She had a number of pre-existing conditions and ended up in the Wuhan intensive care department on a mechanical respirator. The ICU team agreed to the family’s request to provide

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161 [https://maapgh.com/blog/2020/03/vitamin-c-vs-the-coronavirus/](https://maapgh.com/blog/2020/03/vitamin-c-vs-the-coronavirus/)


intravenous vitamin C in addition to the rest of her care. Beating the odds, she recovered. What might be especially interesting is that none of her caregivers came down with Covid-19 disease.

This may mean that medium oral doses of vitamin C serve very well for prophylaxis.

The authors go on to report other anecdotes from the field. From a Wuhan hospital medical director:

“On the afternoon of 20 February 2020, another 4 patients with severe new coronaviral pneumonia recovered from the C10 West Ward on Tongi Hospital. In the past, 8 patients have been discharged from hospital ... high-dose vitamin C achieved good results in clinical applications ... high-dose vitamin C can not only improve antiviral levels, but more importantly, can prevent and treat acute lung injury (ALI) and acute respiratory distress (ARDS).” [translated]

Hyoungjoo Shin, a south Korean physician treating Covid cases reported:

“At my hospital in Daegu, South Korea, all inpatients and all staff members have been using vitamin C orally --- Some people this week had a mild fever, headaches and soughs, and those who had symptoms got 30,000 mg [30 grams] intravenous vitamin C. Some people got better after about 2 days, and most had symptoms go away after one injection.”

A higher IV dose of 24 grams per day (12 grams twice daily) was recently tested in Covid-19 patients in three Chinese hospitals on a double-blind basis. These were the sickest-of-the sick, in intensive-care units.

They only got vitamin C for a week. Yet their mortality at 28 days was cut in half.

**Intravenous Vitamin C in Pets**

IV vitamin C is not just for humans because we do not make it. Other animals may not make enough to handle severe kinds of infections. There is little doubt that high-dose, intravenous vitamin C therapy works in all viral infections, so why not our pets?

Belfield and Stone\textsuperscript{164} wrote in 1975 regarding veterinary treatment of viral infections in animals, “The intravenous use of ascorbate is especially valuable in the therapy of the viral diseases as it appears to be an effective, non-specific, non-toxic viricidal agent. We have not seen any viral disease that did not respond to this treatment. Successful therapy appears to depend on using it in sufficiently large doses.”

I have had reports from several veterinarians that they have used IV vitamin C to successfully treat both canine and feline distemper with high efficacy.

Confirming Expectations

There is a widespread belief that vitamin C has not been properly studied on a double-blind, placebo-controlled basis. This is true, but self-serving because that lack of such study is politically based. Smaller studies that do involve controls or blinding are ignored, even though they undermine the ideology used to justify the political decisions not to study vitamin C.

Here are a couple of examples from recent times: (1) the use of vitamin C in college students to decrease symptoms by 85%,\(^{165}\) and (2) the use of vitamin C to lower the incidence of colds and flus in Korean military recruits by 20%.\(^ {166}\)

For extended content about vitamin C, see Appendix C.

Glucose-6-Phosphate-Dehydrogenase Deficiency

Impaired G6PD enzyme function is fairly common in people with African and Mediterranean ancestors. This is believed to be because of the dangers of malaria, to which low G6PD may offer some protection.

It is widely believed that intravenous vitamin C is contraindicated in people with G6PD deficiency. This belief is so entrenched that almost every IV-vitamin-C protocol specifically excludes anybody with this genetic condition. However, this belief may be dysfunctional.

The G6PD deficiency means that one of the primary paths for production of reducing equivalents (NADPH) is weaker than usual, which compromises glutathione recycling in anaerobic cells (red blood cells). As a result, red blood cells become more susceptible to oxidative stress, which can manifest as methemoglobinemia (the failure of red blood cells to carry oxygen because the iron in heme gets oxidized from +2 to +3) and hemolysis (the rupture of oxidatively damaged RBC membranes).

Vitamin C, oral and IV, carries reducing equivalents that have the potential to remedy this condition. And vitamin C can potentiate Fenton reactions that increase oxidative stress.

Which one is more relevant clinically?

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165 H C Gorton and K Jarvis. The effectiveness of vitamin C in preventing and relieving the symptoms of virus-induced respiratory infections. *J Manipulative Physiol Ther* 22(8): 530-33, Oct 1999. This was an unblinded study of a control group of “463 students ranging in age from 18 to 32 years” during the 1990 class and a test group of “252 students ranging in age from 18 to 30 years” from the 1991 class. “Those in the control population reporting symptoms were treated with pain relievers and decongestants, whereas those in the test population reporting symptoms were treated with hourly doses of 1000 mg of Vitamin C for the first 6 hours and then 3 times daily thereafter. Those not reporting symptoms in the test group were also administered 1000-mg doses 3 times daily.” There was an 85% reduction in the cold and flu symptoms in the test group.

166 T K Kim, H R Lim and J S Byun. Vitamin C supplementation reduced the odds of developing a common cold in Republic of Korea Army recruits: randomised controlled trial. *BMJ Mil Health* 5 March 2020. doi: 10.1136/bmjilitary-20190001384. “This was a randomised, placebo-controlled, and double-blind trial of soldiers who enlisted in the Korea Army Training Centre for 30 days from 12 February to 13 March 2018. The study participants were divided into groups (vitamin C vs placebo). The military medical records were searched to determine whether the participants had a common cold.” And the results: “695 received vitamin C (6000 mg/day, vitamin C group), while 749 participants received placebo (0 mg/day, placebo group). The vitamin C group had a 0.80-fold lower risk of getting a common cold than did the placebo group. Subgroup analyses showed that this effect was stronger among subjects in camp A, among never smokers and among those in physical rank 3.”
It turns out that IV vitamin C is reported to ameliorate both methemoglobinemia and hemolysis in patients with glucose-6-phosphate deficiency. The IV doses of vitamin C reported were in the 1-10 grams four times per day (4-40 grams total per day). These are in the low to medium dose ranges for IV-C.

**Glutathione**

Vitamin C and glutathione work in synergy as pools of reducing power in support the redox-buffering and antioxidant-defense systems. Vitamin C plays a larger role in the extracellular compartment and glutathione plays the dominant role within our cells. There has been a many decades long medical history behind using glutathione in viral infections, but it is not well studied outside of a specialty clinical environment.

The theory has been advanced that endogenous glutathione deficiency is the cause of serious reactions and death from Covid-19 infection. However, I have serious doubts about such judgments. It is the oxidative stress of the immune response that challenges glutathione intracellularly and ascorbate extracellularly. I do not know how researchers separate glutathione from ascorbate and come to conclusions about glutathione in the absence of ascorbate data. Even though cellular responses are intimately connected to extracellular phenomena, (1) the oxidative stress from immune activation in generated in the extracellular compartment, (2) vitamin C is the primary extracellular reducing agent, and (3) the oxidative stress that overwhelms immune cell-signaling mechanisms and triggers cytokine storms (and death) takes place extracellularly. So it seems much more likely to me that extracellular ascorbate would be the cause of intracellular glutathione stress. Both glutathione and ascorbate can be comparably recycled, but only vitamin C cannot be synthesized on demand. And of the two, glutathione can only be supplemented safely in low doses. For all these reasons, I have severe doubts that glutathione supplementation would be the treatment of choice. Adjunctive therapy to ascorbate repletion? Yes, indeed. But primary therapy is ascorbate.

One case report mentions a specific benefit for glutathione in labored breathing in two cases of Covid-19 infection (one diagnosis by antibody testing and the other by radiology exam). Both of these patients were considered high risk because of pre-existing conditions (Lyme disease and tick-born co-infections). Respiratory distress improved within one hour of treatment with 2 grams of glutathione. This relief attenuated over time (hours) but was restored each time glutathione was re-administered.

**Cysteine**

Glutathione is not particularly efficient when given orally. This is because glutathione is a tri-peptide composed of three amino acids connected together, and the stomach is a digestive organ designed to break down peptides and proteins. So some of the glutathione is broken down before it can be absorbed.

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Some quackbusters have made the false claim that all glutathione is broken down and that none is absorbed. This is not true. They have also made the false claim that oral enzymes are worthless because they are also broken down before absorption.\textsuperscript{169}

The amount of glutathione (or enzymes) that is broken down depends on many variables involved in stomach conditions: pH, enzyme concentrations, the presence of food, dilution effects from water or beverages, and the formulation of the supplement. When glutathione is fully digested, it is broken down into glycine, cysteine and glutamate, the three amino acids from which it is synthesized.

It is generally much more efficient to orally supplement the precursors of glutathione than glutathione itself. Since most food proteins are high in glutamate (20-35\% of amino acids) and glycine is high in collagen proteins associated with animal products, cysteine is the amino acid that is most commonly the rate-limiting amino acid in making glutathione.

\textbf{NAC}

N-acetyl-L-cysteine (NAC) is a derivative of L-cysteine\textsuperscript{170} with better absorption and transport efficiencies. NAC raises glutathione more efficiently than cysteine does.

Both NAC and cysteine contain the sulfhydryl (thiol) group that is the active site for the antioxidant and reducing activity of glutathione. So both act as glutathione mimics, performing the same basic redox functions even before they get converted into glutathione. This makes them especially rapid in inducing a clinical response.

Both cysteine and NAC have been used successfully in treating formaldehyde and acetaldehyde exposure and in preventing severe alcohol-related hangovers.

\textsuperscript{169} Beware of authorities who make pronouncements strictly from theoretical arguments. They are sometimes right, but wrong often enough to make listening to their advice mostly a waste of time.

\textsuperscript{170} L-Cysteine is the natural form of cysteine. The L merely distinguishes L-cysteine from D-cysteine. Since D-cysteine is not available as a supplement and not found in natural, unprocessed foods to any significant degree, the L is usually omitted from writing and discussions. Regarding pronunciation, cysteine is almost universally mispronounced. The correct pronunciation is sis-teh-een, and sis-teen is the correct pronunciation for cystine (cys-S-S-cys, oxidized cysteine). Rarely, in technical discussions, you might hear the “eh” syllable in cysteine to distinguish reduced from oxidized. But in 99\% of common usage, sis-teen refers to cysteine and “oxidized sis-teen” refers to cystine.
Basal Metabolic Rate

Cultivate a strong metabolism. That’s what I wrote to encompass one of the most important aspects of anti-viral fitness. It is especially relevant today because it has become medical dogma not to treat hypometabolic “pre-existing conditions” without some evidence of abnormal thyroid hormone blood-test results.

As a result, as much as 40% of our population is walking around in a viral-susceptible state believing that they are “fine.” This not just a tiny minority of people with a condition that is difficult to see. It is a blatantly obvious condition that is hard to miss and so common that it alters the statistical basis for its own diagnosis via blood-test normative ranges.

Low basal metabolic rate is one more important “pre-existing condition” that is ignored by media, the WHO, the NIH and the CDC. Check yourself out.

Hypothyroid Symptoms

Here’s a list of symptoms that you can self-check.

Cold hands and feet. This also includes low core-body temperature, difficulty in warming up after getting chilled, and regularly wearing more clothing than others around you. It also tends to include a marked aversion to cold weather and to approaching storm fronts.\(^\text{171}\)

Slow pulse rate. This is slow pulse rate not caused by exercise and aerobic fitness. Sometimes doctors will consider this a good sign rather than a pathology because they presume that it is a sign of fitness. They do not think to even ask a question to qualify the possible athletic contribution.

A tendency towards constipation. This also includes other GI-related symptoms like slow digestion, a long bowel-transit time, acid-reflux symptoms, and intestinal gas.

Fatigue. This category includes lack of strength, poor stamina, and lethargy.

Depression. Mild to moderate depression, loss of drive, apathy, sleeping difficulties, memory problems.

Neuropathies. Tingling or burning sensations. Numbness.

Edema. Water retention in the feet and ankles, facial puffiness (eyelids and bags under the eyes).

Other. Weight gain, coarse skin, dry skin, and hair loss.

Basal metabolic rate lowers as we age, at roughly 1% for every three years of life. This is one reason that the elderly are more at risk from viral infections. But it’s not just aging. Anesthesia lowers metabolic rate, and when the anesthetic wears off, metabolic rate does not always return to its pre-surgery baseline. This is called metabolic entrainment, and it can also happen with highly stressful events in our lives, like having infectious mononucleosis as a child, losing a spouse, or being ill for a long time. Metabolic rate drops when we have inflammation, through cytokine-driven conversion of testosterone and progesterone to

\(^{171}\) Storm fronts (bad-weather systems) are almost always associated with low atmospheric pressure, where oxygen partial pressure drops, further impairing an already-low metabolic rate. The same thing happens when people who normally live at sea level fly in airplanes, which are only pressurized to 7-8,000 feet while they fly at 30-35,000 feet. This is like somebody living in San Francisco traveling to Lake Tahoe to ski, only the altitude transition takes place in 30 minutes in an airplane instead of many hours in a car. Metabolic rate depends on the partial pressure of oxygen. When air pressure goes down, oxygen goes down and metabolism goes down.
estradiol and estrone, and through interference in the neuroendocrine mechanisms regulating thyroid and adrenal hormones. Testosterone and progesterone promote basal metabolic rate, while estrogens suppress it. Exercise and physical activities promote higher metabolic rate, and sedentary lifestyles can inhibit it. However, apparent fitness is no guarantee of a healthy metabolism. Toxicities of almost all kinds can lower metabolic rate, from heavy metals like mercury, lead, thallium, bismuth, uranium, arsenic and cadmium, to mycotoxins and endotoxins from fungi and bacteria. Dental fillings (mercury amalgams) and fluoride treatments can lower metabolic rate. Even eating cruciferous vegetables (broccoli, Brussels sprouts, bok choy, cabbage, kale, cauliflower, etc.) can be a significant adverse influence. The order of adverse influence is raw over undercooked, and undercooked over fully cooked.

The metabolism issues is not a simple, one-dimensional problem. It also overlaps with many other “pre-existing conditions” considered separately.

**Hypothyroidism versus Hypometabolism**

Prior to the 1950s, hypothyroidism was defined by the above symptoms, and thyroid hormone prescribed to ameliorate those symptoms. It worked fine. But all that changed when synthetic thyroid hormone entered the market and thyroid testing became mandatory by administrative-law edict. The definition of hypothyroidism shifted from the lack of effect of thyroid hormones to lack of thyroid hormones themselves. So, overnight, without any scientific, medical or regulatory assessment of the clinical validity of that change, the diagnosis of hypothyroidism shifted from a thyroid hormone problem to a thyroid gland problem. To make this change even more ethically unsound, the thyroid standards for hormone levels were set statistically instead of clinically. The statisticians set the high and low to be equal in numbers of patients, when in reality, hypothyroid patients outnumbered hyperthyroid patients by at least ten to one, and likely a hundred to one. Maybe this might be considered good science, but it was definitely bad medicine.

So the symptom list above was thrown out and replaced with arbitrary thyroid hormone blood tests that were not correlated to clinical findings.

Two trends resulted.

One trend was the doctors who knew this was unethical and continued to practice medicine the old way started to get disciplined by medical boards and lose their medical licenses. Many hundreds of doctors ended up losing their licenses. Some got the message and acceded to this medical faux pas.

The other trend, which still affects us today, is that more and more people with hypothyroidism and hypometabolism were not diagnosed properly and were told, “Your thyroid gland is fine. Just learn to live with it.”

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172 Estrogens have a metabolic-lowering effect in heart, lung, kidney, liver and brain tissues. But for white blood cells, metabolism is inversely wired to estrogens. While non-immune cells “hibernate” during episodes of inflammation, immune cells go into metabolic hyperdrive. They become specialists in absorbing oxidized vitamin C through glucose receptors and recycling it to reduced ascorbate for use by the white blood cell in generating reactive oxygen species for fighting microorganism infections.

173 For example, over-training can seriously undermine aerobic fitness. When Frank Shallenberger first began testing “aerobic threshold” (the point at which the body switches from aerobic metabolism to less efficient anaerobic metabolism), he expected to see a linear relationship between training and threshold. That is not what he found. He found examples of aerobically fit couch potatoes, and examples of aerobically challenged over-trained athletes (he, himself, was one). When he backed off his training, he started winning senior mountain-biking competitions. This is but another way in which appearances can be deceiving.
And some physicians adding, “I’d be happy to refer you to a psychiatrist.”

As bad as this was, it got worse.

It became the standard of care to ignore actual thyroid-hormone levels and instead rely upon testing of thyroid stimulating hormone (TSH), the hypothalamic negative feedback loop that has even less to do with thyroid hormone health and wellbeing than T3 and T4. More about that later.

So all these hypothyroid people, roughly 40% of the US and UK populations, “are allowed to walk the streets freely, as though there was nothing wrong with them.” And the coronavirus is picking them off one by one, with the medical system saying that those deaths do not involve a pre-existing condition.

In a recent interview with the Silicon Valley Health Institute (July 23rd), UK functional medical expert Antony Haynes pointed out that this problem disproportionately affects older women. His estimate was that 40% of women in their 40s, 50% of women in their 50s and 60% of women in their 60s were suffering from “subclinical hypothyroidism” (hypometabolism).

This is medical malfeasance on an institutional level.

If a bit of this is too technical, let me convert it to a more familiar analogy. Let’s consider hearing impairment as analogous to hypometabolism. The hard-of-hearing pathology is the inability to hear sound at normal volume, which we are going to consider analogous to hypothyroidism, where the tissues cannot “hear” thyroid hormone at its normal levels. In other words, it is the thyroid receptors on the cells, nuclei and mitochondria which respond to thyroid hormones to cause the effects of thyroid hormones. In this scenario, you are at your family’s Thanksgiving gathering sitting at the dining table with your grandfather and ask, “Gramps, will you pass the salt?” If he’s hard of hearing, he smiles and nods without passing the salt. You now know that he has heard you say something without understand what you actually said. So you raise your voice a notch, and he then passes you the salt. The second time, he both heard you and understood you. That’s the proper testing method to assess his hearing. But in the 1950s, this became illegal. To continue the analogy, grandfather’s hearing can no longer be directly tested. Only your voice will be measured. And because there is nothing wrong with your voice, the medical conclusion is that there is nothing wrong with Gramp’s hearing.

It’s so stupid, it’s staggering. But because the idiocy is promoted by middle-aged males in white lab coats with stethoscopes draped around their necks, everybody buys it, and generations of people live with chronic hypometabolism without realizing it.

And now some of us are dying from it.

It does not seem so bad at first because hypometabolism is associated with slow-to-develop degenerative diseases and mild-but-cumulative risk factors that are not blatantly visible until ten, twenty to forty years later. That delay between cause and effect makes it non-obvious. So we get a slow upward trend of hypometabolic diseases: autoimmune diseases, cardiovascular diseases, autism, Alzheimer’s disease, chronic fatigue syndrome, POTS, allergies, multiple chemical sensitivity syndrome, and more. And to add insult to stupidity, many of these diseases of hypometabolism are vigorously denied to be “real diseases” by the medical authorities for 1-3 decades before being grudgingly accepted, but only as idiopathological diseases (diseases without an acknowledged cause). But, in reality, the causes are not idiopathic. They are actually known.
It’s a beautiful system from the perspective of a business model. Investors love it. Ever increasing economic returns from ever-expanding disease management, with spin-doctor deniability.\textsuperscript{174} One could not imagine a better business opportunity. But from the perspective of human suffering, it’s a travesty of unprecedented proportions. And it took the unprecedented coronavirus pandemic to make it real enough for everybody to notice.

That is assuming that people will notice, and not blame it on the novelty of the SARS-CoV-2 virus.

**Sinus Self-Defense**

With respiratory viruses, the tissue of first contact is the sinuses. As it turns out, cold extremities is not the only consequence of a below-normal body temperature. Sinus temperature is also affected, to the tune of about 2°C (3.5°F).\textsuperscript{175} Sinus-based evaporation is a cooling mechanisms for many mammals, which is why your pet dog pants when it gets hot. Panting cools the sinuses by evaporation of moisture, so the body can prevent overheating.

Other research is underway trying to connect tissue temperature, surface-tissue hydration and mucous hydration and fluidity with the immune response to viral loading.

\textsuperscript{(+)} update on iodine-vapor sniffing and H\textsubscript{2}O\textsubscript{2} nebulization.

\textsuperscript{(+)} not only antiviral, but antigen presenting.

**Thyroid Self Defense**

First, understand that thyroid hormone testing is deeply flawed. There is nothing that you could possibly do to change it, so compensate for it. Revisit your thyroid hormone tests and re-interpret the “normal” range to something more realistic. For TSH, it means that the highest “normal” numbers are not normal at all. For T\textsubscript{3} and T\textsubscript{4} test results, it means that the lowest numbers are not normal. So re-calculate them.

The easiest scheme for me is to divide the normal range into four equal pieces\textsuperscript{176} (“quartiles”) and consider the bottom quartile to be pathological, the second to be “low”, the third to be “medium” and the fourth to be “high.” This basically “subtracts” at least some of the portion of the population with hypothyroid symptoms that are included as normal when they should be excluded. If you think in percentages better than quartiles, convert your test result to a percentage of the normal range where zero percent of the bottom and 100% is the top of the range. The closer you are to zero, the more you should conduct follow-up assessments.

\textsuperscript{174} Pun intended.

\textsuperscript{175} There have been two studies. This is the confirmatory study. P Assanasen, F M Baroody, Edward Naureckas and R M Naclerio. Warming of feet elevates nasal mucosal surface temperature and reduces the early response to nasal challenge with allergen. \textit{J Allergy Clin Immunol} 104: 285-93, 1999. “Conclusion: Our data show that warming of feet decreases the early response to nasal challenge with antigen. This inhibitory effect is probably related to the increase in the nasal mucosal temperature.”

\textsuperscript{176} Here are step-by-step instruction for the T3 and T4 ranges for linguistic-oriented learners: (1) Add the top-of-the-range number to the bottom-of-the-range number and divide by two. That’s your average or middle of the range. If you then add the bottom number to the middle number and divide by two, you have found the top of the first quartile and the bottom of the second quartile. Then add the middle number to the top number and divide by two. You now have the third and fourth quartiles. So if you are in the bottom quartile, you should not consider yourself to be “fine” without further investigation and follow-up testing. If you are in the second quartile, your hormone levels are “low normal.” If you are in the third quartile, your hormone levels are medium normal. And if you are in the fourth quartile, your hormone levels are high normal. This slight shift can truly change your perspective.
For TSH readings, anything above 2 may be problematic and above 3 considered suspect. Most doctors consider 4 to be within the normal range and 5 to be within the normal range for the elderly.

The second thyroid self-defense strategy to consider is to ask for further testing. Many physicians consider the TSH test to be definitive. That’s all they routinely order. In my opinion, the TSH test is the thyroid test most likely to be misleading. It has the worst correlation with basal metabolic rate, whereas the T3-to-rT3 ratio has the best correlation. These last two tests are the least likely to be ordered.

So if you only have a TSH test, ask for a free T4 test. If you have that, ask for a free T3 test. If you have that, ask for thyroid autoantibody tests or a reverse T3 (rT3) test.

If you have almost all the symptoms of hypothyroidism, and several in severe form, consider the third thyroid self-defense strategy: a thyroid trial. There are three ways to do this, (1) with the full support and participation of your physician, (2) with just “medical supervision” from your physician, and (3) without the cooperation of your physician.

If you pose the prospect of a thyroid trial to your physician, citing your symptoms and their degree of adverse effect to your health and wellbeing, you might get cooperation. The physician can prescribe thyroid hormone to you and make notes in your medical record that he/she recommended against it and counseled you not to do it. But since you stated that you were going to do it anyway, he/she considered it ethically compelling to provide supervision to minimize “the harm that you are risking.” And by prescribing the thyroid hormone, they have one more control of your trial.

In my opinion, the above is the ethical way to practice medicine in this situation of institutional medical malfunction. However, there are two things that need further consideration. One is possible discipline of your doctor by your State Medical Board. The board might not agree that your threat of conducting a thyroid trial on your own is genuine, or that there is no reason to doubt thyroid hormone test results are definitive. This fear on the part of your physician is not imaginary. Medical Boards are administrative-law institutions that can decide your doctor’s fate regardless of facts and testimony. In fact, they do not have to consider any facts or listen to any testimony if they do not want to. They can disqualify any witness regardless of credentials, and can qualify any witness regardless of any lack of credentials. Since they are “defending the faith” from the faithless, your doctors is at genuine risk of losing their livelihood without due process of law (Constitutional protections do not apply to administrative proceedings).

The second thing to consider is automatic insurance coverage of the prescribed thyroid hormone under MediCare or private insurance, where there is a risk that the doctor might be charged with insurance fraud. This, also, is not imaginary. The fines for Medicare fraud are $5000 per day per instance. Ouch. And the charge of insurance fraud does not need any evidence to make; the doctor is automatically guilty until they prove themselves innocent. And the court does not have to listen to any evidence that your doctor or you might want to present. Double ouch.

In that situation, the compromise position is medical supervision without a prescription. This is like strategy three below, but with your physician looking over your shoulder the entire time. This is the way to go if you can arrange it. In this situation, you obtain medical thyroid hormone from Europe, Asia or Africa from the same companies and brands prescribed in the USA. Or you buy pork or bovine tissue as dietary

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177 T3 and reverse T3 (rT3) are both made from T4 by de-iodination. There are four iodine atoms on T4 and only three on T3. When T4 is converted to T3, an outer iodine atom is removed. If, instead, an inner iodine atom is removed, reverse T3 is produced. Iodination (making T4 or T3 from thyronine (no iodine atoms)) and de-iodination (removing one iodine from T4) are selenium-dependent processes.
supplements that are not medical grade and do the trial with possibly a bit less dose standardization. In the USA, you can still make use of either of these options under the FDA’s personal-use import policy, which was implemented to assuage the HIV+ underground which was supporting AIDS Buyer’s Clubs.

The last strategy is to buy thyroid hormone on your own and take it on your own recognizance. In doing any of these options, the thyroid trial requires close monitoring of the progress of the trial. The most important factor to monitor is your pulse rate. Remember, low pulse rate is one of the symptoms of hypothyroidism. When you take thyroid hormone, your pulse rate should increase towards normal without rising to hyperthyroid levels. Going from 45 to 55 is good, going from 75 to 115 is bad. So rigorously monitor your pulse rate over the course of the trial. If you are getting medical supervision, they will repeatedly tell you this. And they will measure it every time you go to see them.

I also find it useful to monitor body temperature.

The rest of what you need to measure has to do with your complaints. The whole idea of a trial is to verify if you actually need more thyroid hormone to be healthy and well, so to prove your case, you should measure everything that is not well. Mood, sensitivity to cold, depression, sleeping problems, lack of strength or stamina, lethargy, difficulty in waking up in the morning, waking up in the middle of the night with anxiety, nervousness or edginess, difficulty in falling asleep, or inability to stay asleep, etc. If these symptoms ease while your thyroid hormone is increasing its influence through replacement, you are proving your case. If you collect good, meticulous data and have it placed in your medical record, there should be no question that thyroid hormone should be prescribed and paid for by insurance.

If you have no intention of staying on thyroid hormone for the rest of your life, you need to plan for phasing out the thyroid hormone replacement before the end of the trial (or before you run out of thyroid hormone) to minimize any sudden withdrawal symptoms, which will tend to aggravate all of your pre-existing low-thyroid symptoms.

(+ ) Add thyroid one pager re-done.

**Hypometabolism Self Defense**

Because hypometabolism and hypothyroidism affect so many aspects of health, wellness and aging on a foundational level, many diverse conditions and causes may play a role in how it manifests in a particular person. Here are some aspects that you can consider, both on a lifestyle basis and a medical basis:

**Age.** We lose about 1% of our metabolic rate for every year that we age.

**Aerobic fitness.** Exercise programs reverse many aspects of aging, including metabolic rate.

**Anesthesia.** Each time you get an anesthetic, you temporarily suppress your metabolism. Local anesthetics have a low systemic effect, and total anesthesia has a global effect. Metabolic entrainment to a lower metabolic rate can happen. Your anesthesiologist may tell you otherwise, but it happens. Maybe one in ten will have difficulty in getting all the way back to their pre-op “normal” temperature.

**Autoimmune diseases.** A common manifestation of hypometabolism affecting young women much more than young men, and older men a bit more than older women.

**Chemical sensitivity syndrome.** Chemical exposures trigger redox stresses that incapacitate.

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178 I remember reading one paper that found that prescription thyroid was less standardized than supplements.
**Chronic fatigue syndrome.** Energy downturn is constant, inflammation is chronic.

**Chronic inflammation.** Causes sequestration of copper, zinc and iron. Causes decreased tryptophan conversion to serotonin by IDO induction. Causes estrogen dominance by aromatase induction. Dysregulates thyroid and immune function. Suppresses normal healing.

**Circadian disruption.** Jet lag and artificial jet-lag syndromes. I’m listing this as separate from sleeping problems.

**Depression.** A common consequence of low basal metabolic rate. The human nervous system uses 20% of the body’s total energy production, despite being only 3% of total body mass. Energy downturns the most energetic tissues disproportionately. Brain, kidney, heart, liver, muscles. Depression might undermine metabolic rate and immune competence in addition to the hypometabolic effect.

**Dysbiosis.** Imbalance in the microbial ecology in the intestine.


**Endotoxins.** Bacterial toxins that can cause inflammation. See also gut dysbiosis.

**Estradiol and Estrone dominance** (E₂ and E₁, respectively). These can be constitutional in some women, the result of infection and inflammation in everybody, and a common cause of grumpy-old-men syndrome. Estrogen dominance is also a primary risk factor for autoimmune diseases.

**Fluoride toxicity.** Dental procedures are one way this can happen. Oral hygiene products are another.

**Heavy metal toxicity.** Lead, mercury, thallium, bismuth, cadmium, tin, antimony, uranium, thorium. These all have an adverse effect on sub-cellular functions, which includes mitochondrial function and DNA transcription. The heavier the metal, the deeper it resides in body compartments.

**Infection.** Downregulation of the immune system, its signaling pathways and antigen presentation can allow chronic, low-level infections to persist.

**Inflammation.** Inflammation causes induction of cytokines that suppress metabolic rate in most cells but stimulate metabolic rate in immune cells. Some of this is mediated by the conversion of progesterone to androstenedione to estrone and the conversion of testosterone to estradiol.

**Insulin resistance.** Before you get diabetes, you get insulin resistance. Insulin is necessary for efficient metabolism of sugar in tissues that are insulin responsive. Most body tissues are insulin dependent. Blood sugars in the high 80s to low 100s are one sign, but an insulin measurement is the silver standard and an insulin glucose tolerance test is the gold standard.

**Leptin resistance.** Related to insulin resistance but modifying immune function through cytokine effects as well. Serious obesity risk.

**Lung conditions.** Hypometabolism has a disproportionate effect on lung health. Because lung tissues are involved in gas exchange, they are exposed to the highest levels of atmospheric oxygen. The gas-exchange process also imposes a pH difference between the lung tissues (most alkaline) and the deep tissues (most acidic). Hypometabolism is an alkalinizing stressor. This can impose synergistic stresses to the lungs that interfere with immune function, mitochondrial function, inflammation, fibrosis, potassium utilization and hydration.

**Mitochondrial senescence.** Oxidative stresses cause mitochondrial DNA damage which impairs mitochondria that are not replaced by regular autophagy episodes.
**Mycotoxin exposure.** Mold toxins. As it turns out, some people are quite sensitive to mycotoxins and others may not be.

**Nutritional deficiencies.** Vitamins, electrolytes, trace minerals, amino acids, antioxidants. Most common: vitamin D3, K2, C, magnesium, then zinc, selenium, iodine, vitamin A.

**Pessimism and hopelessness.** A sour view of life and feelings of helplessness are neuroendocrine regulators affecting metabolic rate.

**Phytotoxins.** Eating cruciferous veggies, especially uncooked (broccoli, Brussels sprouts, cabbage, cauliflower, kale, arugula, etc.).

**Sedentary lifestyle.** Aerobic fitness from exercise is a natural up-regulator of metabolic rate. Strength-training exercise builds muscle mass, which is pro-metabolic.

**Sleeping problems.** This is one of the earliest manifestations of developing disease that people can easily notice. The relationship of sleeping problems to underlying disease pathology is virtually ignored by modern medical care. One well documented mechanism is inflammation-induced decrease in serotonin influence by indoleamine dioxygenase (IDO). This also produces dopamine dominance, which amplifies fear, anxiety, drive, competitiveness, obsessive ideation and compulsive behaviors like shopping, gambling, sex and thrill-seeking behaviors.

**Sunlight.** Sunlight promotes metabolic rate with UV-induced vitamin D production and near-infrared light stimulation of mitochondrial electron transport efficiency. Sun avoidance is a common cultural and lifestyle practice.

These conditions can all be considered “pre-existing conditions” affecting viral morbidity and mortality.

**Vitamin B₁₂**

Although Emanuel Revici (see Appendix A) characterized the metabolic character of vitamin B₁₂ as catabolic-aerobic-acidifying more than a half century ago, its use as an antiviral agent has been underappreciated. I reported on this in the BHT book, where Dr. Ward Dean found that his patients with recurrent herpes outbreaks who responded poorly to BHT (butylated hydroxytoluene), responded quite well to B₁₂ administration. As it turns out, B₁₂ is often insufficient or outright deficient in the elderly due to an age-associated trend in loss of intrinsic factor, which facilitates B₁₂ absorption dramatically.

Given the risk factors of age in Covid outcomes, it is reasonable to anticipate that B₁₂ supplementation will be found to improve clinical outcomes. An early finding gives credence to that possibility.⁴⁹ They report that the combination of B₁₂ with magnesium and vitamin D “was associated with a significant reduction in proportion of [older] patients with clinical deterioration requiring oxygen support and/or intensive-care support.”
Pre-Existing Health Conditions

It is quite clear that the death rate for influenza and coronavirus infections is higher in the elderly and in those people with pre-existing conditions.\(^{179}\) Some of those pre-existing conditions may be blatant, like aging, diabetes, heart disease, kidney disease and autoimmunity. But some are not so obvious. And others are deeply intertwined.

Think of pre-existing conditions under the analogy of an iceberg. There is what you see when traveling on the ocean’s surface, and then there is what is below the water line that you cannot see. The coronavirus is merely exposing some of those underlying risk factors that have well-known longer-term impacts on morbidity and mortality and making them short-term risks.

The good news is that all of these pre-existing conditions are treatable.

The production of the active form of vitamin D (1,25-dihydroxyvitamin drops 50% due to age-related decrease in kidney function.\(^ {180}\) Many of the “diseases of aging” are also risk factors. Most of these are treatable by diet, lifestyle changes, supplements, hormone replacement, and other medical treatments.

The bad news is that pre-existing conditions are on the increase. Just as autism, Down’s syndrome, learning disabilities and falling general intelligence are steadily increasing over time in the USA and western world.

The reason that summertime mortality is the clearest indicator of this trend is that wintertime mortality spikes and troughs with the severity of the viral strains for that particular winter season. In the summer, the viral contribution to mortality drops to its annual minimum. So the summertime data is minimally confused by viral-assisted deaths.

This same pattern is seen with mortality from some kinds of cancer. Regarding cancer diagnoses, there is no blatant seasonal bias. Cancer is diagnosed pretty much evenly throughout the year.\(^ {181}\) But cancer mortality is quite seasonal, peaking in the deep winter months (February, plus or minus). The

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181 A Ho, A Gabriel, A Bhatnagar et al. Seasonality pattern of breast, colorectal and prostate cancer is dependent on latitude. *Med Sci Monit* 20: 818-24, 2014. This study analyzed data from multiple US states at varying latitudes looking at the summer-winter differences. Winter was defined as December, January and February, and summer was defined as June, July and August (actually reported as June and July only, but August was not included in the fall season). They write, “Female
correlation with vitamin D is seen in the dependence of the summer-winter risk differential on latitude. The further north you go, the greater the difference between summer risk and winter risk (see graph at right).

**Being Male**

Men have a more serious reaction to coronavirus than women. This has been argued to be related to the higher levels of ACE2 (angiotensin converting enzyme-2) receptors in males. More recently, von Willebrand factor has been advanced as a Covid risk factor, which also is higher in males and blacks (see Being Black, immediately following).

A collaboration of researchers from the USA, Spain and Brazil are suggesting that androgen sensitivity is a direct risk factor for Covid-19 disease severity.\(^{182}\)

**Being Black**

Having more melanin in your skin is an obvious influence on vitamin D-related risks (see the vitamin D chapter, and historical slavery discussion on page 175). Many diseases disproportionately affecting US citizens of recent African ancestry (see page 175) have been tied to vitamin D insufficiencies to such an extent and for so long that I wonder why this aspect of public health has not been publicized by either US federal public-health agencies or by leaders and spokespersons within black advocacy groups. These racially associated health risks include (1) infectious diseases,\(^{183}\) (2) metabolic syndrome,\(^{184}\)

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breast cancer patients exhibited improved survival when diagnosed in the summer as compared to the winter at all latitudes.”

In the more northern states, prostate and colorectal cancers showed the same therapeutic benefit of starting treatment in the summer season. The linear regression analysis of the summer-winter difference in hazard ratio found a positive correlation with latitude.

\(^{182}\) C G Wambier, G Goren, S Sergio Vaño-Galván et al. Androgen sensitivity gateway to Covid-19 disease severity. Drug Development Research 15 May 2020. doi: 10.1002/ddr.21688. Abstract: “In this communication, we present arguments for androgen sensitivity as a likely determinant of COVID-19 disease severity. The androgen sensitivity model explains why males are more likely to develop severe symptoms while children are ostensibly resistant to infection. Further, the model explains the difference in COVID-19 mortality rates among different ethnicities. Androgen sensitivity is determined by genetic variants of the androgen receptor. The androgen receptor regulates transcription of the transmembrane protease, serine 2 (TMPRSS2), which is required for SARS-CoV-2 infectivity. TMPRSS2 primes the Spike protein of the virus, which has two consequences: diminishing viral recognition by neutralizing antibodies and activating SARS-CoV-2 for virus-cell fusion. Genetic variants that have been associated with androgenetic alopecia, prostate cancer, benign prostatic hyperplasia and polycystic ovary syndrome could be associated with host susceptibility. In addition to theoretical epidemiological and molecular mechanisms, there are reports of high rates of androgenetic alopecia of from hospitalized COVID-19 patients due to severe symptoms. Androgen sensitivity is a likely determinant of COVID-19 disease severity. We believe that the evidence presented in this communication warrants the initiation of trials using anti-androgen agents.”

\(^{183}\) J H Richardus and A E Kunst. Black-White differences in infectious disease mortality in the United States. Am J Public Health 91(8), 1251. August 2001. “Infectious disease mortality among Blacks was higher than among Whites, with a relative risk of 1.53 after adjustment for age and sex and 1.34 after further adjustment for income and education. Death from infectious diseases contributed to 9.3% of the difference in all-cause mortality.”

\(^{184}\) K C Maki, V L Fulgoni 3rd, D R Keast, T M Rains, K M Park and M R Rubin. Vitamin D intake and status are associated with lower prevalence of metabolic syndrome in U.S. adults: National Health and Nutrition Examination Surveys 2003-2006. Metab Syndr Relat Disord. 10(5): 363-72, Oct 2012. “Those in the highest quartile of serum 25(OH)D had 60% lower odds for metabolic syndrome as compared to those in the lowest quartile.” “Compared with the lowest vitamin D intake quartile (excluding supplements), those in the highest intake quartile had 28% lower odds for metabolic syndrome.” “We conclude that higher 25(OH)D, and, to a lesser degree, greater dietary vitamin D intake, are associated with reduced prevalence of metabolic syndrome.”
diabetes, (4) hypertension, (5) stroke, (6) cancer survival and (7) total mortality.\textsuperscript{185} In addition to the vitamin D issue, von Willebrand factor is higher in males and Blacks. This may be the particular risk factor unique to Covid-19 that drives microclotting and tissue fibrosis (see Coagulopathy and Clotting from Coronavirus on page 123).\textsuperscript{215}

**Having Diabetes (and pre-diabetes)**

Diabetes is a well-acknowledged risk factor for Covid-19 risks. Lesser known are the diabetes risk factors insulin resistance, pre-diabetes and metabolic syndrome (aka syndrome X). All of these conditions involve a central dysregulation is insulin function and blood-sugar control that impairs cellular energy production, antioxidant defenses and redox-buffering capacity.

**Blood Sugar, Insulin Resistance, Metabolic Syndrome, Diabetes.**

Blood-sugar abnormalities are strongly correlated with adverse Covid-19 outcomes. Diabetes is cited in the mainstream media as a risk factor, but the pre-diabetes and insulin-resistance conditions that lead to diabetes are rarely mentioned. But all these conditions fundamentally impair energy production pathways that are necessary for mounting a competent immune defense against viral and bacterial infection.

Insulin resistance is the condition resulting from over-consumption of carbohydrate-rich foods, like cereals, breads, pasta, grains and sugar. Despite all the evidence that regular consumption of such foods cause all of the modern “diseases of civilization,” the official US Food Pyramid has carbs as the foundation of a healthy diet. This is not only wrong; it is diametrically opposed to the bulk of the scientific and medical findings regarding healthy diets. And it’s now clear that the “expert committee” for setting the US governmental official dietary guidelines for the 2020-2025 period knows this. As part of their earlier deliberations in 2015, they “officially” reviewed the evidence for low-carb diets, but excluded their review from their published findings.\textsuperscript{186} It is now five years later and they have yet to alter historical policy, despite the preponderance of the evidence.

**Obesity**

Likewise, obesity is widely recognized as a Covid-19 risk factor.\textsuperscript{187} However, what has not been teased out is the relative contribution of obesity itself (a high body fat percentage) and the associated insulin


\textsuperscript{186} Nina Teicholz. A low-carb strategy for fighting the Pandemic’s toll. *The Wall Street Journal* 1 June 2020. “Federal dietary guidelines don’t reflect the evidence that eating fewer carbohydrates can reduce obesity, diabetes and heart disease.” An excellent, must-read article. “Those with metabolic conditions are more likely to suffer worsened Covid-19 outcomes”

\textsuperscript{187} Q Cai, F Chen, T Wang et al. Obesity and Covid-19 severity in a designated hospital in Shenzen, China. *Diabetes Care.* May 2020. https://care.diabetesjournals.org/content/early/2020/05/12/dc20-0576. In this hospital, 393 consecutive patients were classified by their body-mass index:

<table>
<thead>
<tr>
<th>BMI</th>
<th>underweight</th>
<th>normal</th>
<th>overweight</th>
<th>obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>non-severe cases:</td>
<td>4% of patients</td>
<td>53.1% of patients</td>
<td>32% of patients</td>
<td>10.7% of patients</td>
</tr>
<tr>
<td>severe cases:</td>
<td>16 of 16 patients</td>
<td>164 of 203 patients</td>
<td>87 of 123 patients</td>
<td>25 of 41 patients</td>
</tr>
<tr>
<td>deaths:</td>
<td>zero of 16 patients</td>
<td>39 of 203 patients</td>
<td>36 of 123 patients</td>
<td>16 of 41 patients</td>
</tr>
</tbody>
</table>

1 of 203 patients | 1 of 123 patients | 1 of 41 patients |
resistance (discussed immediately above). As it turns out, much of the risks attributed to obesity are actually associated with insulin resistance and metabolic syndrome, not obesity itself. And this tends to get lost in the statistical analysis when obesity is measured but insulin resistance is not.

Roughly 75 percent of people with obesity have insulin resistance and roughly 25% of non-obese people have insulin resistance. Both groups are at similar risk, but because the 75% are “diluted” by only 25% (obese individuals without insulin resistance) and the 25% are diluted by the 75% (non-obese “normals” without insulin resistance), the conclusions of many public health analysis is that obesity is the risk and non-obesity is without risk. This is false. Insulin resistance in involved in both groups, just to different degrees.

When award-winning comedian Sofie Hagen called for the UK’s Cancer Research to cease their ad campaign against obesity causing cancer because it was fat-shaming oriented, I was one vocal critic agreeing with her position.188

**Insulin Resistance**

Insulin resistance is the underlying condition that preceded diabetes, an acknowledged risk factor for adverse influenza and Covid-19 outcomes. In its early stages, insulin resistance is almost symptomless. Most people are totally unaware that they are becoming insulin resistant until their blood sugar nears 99 mg/dl or crosses 100 mg/dl and their doctors finally says something.

Insulin resistance is often considered a normal aspect of aging. And it is intrinsic to pregnancies. However, dietary abuses popular in modern society aggravate insulin resistance and increase the risk of gestational diabetes.189

However, under the hood, glucose-energy pathways are being starved. Insulin is the hormone that tells most of the cells of the body to make more glucose transporters. These glucose transporters are stored inside of cells and travel to the cell membrane only when the insulin signal activates their movement. So as insulin loses its effect during the development of insulin resistance, less and less transport of glucose into cells takes place. And more and more glucose accumulates in the blood.

The burning of glucose for energy supports the redox-buffering system. Each glucose burned generates NADH “reducing equivalents” that get transferred into NADPH, glutathione and vitamin C, or into ATP. So glucose-burning efficiency is a foundational standard for antioxidant defense and immune modulation. Any degree of insulin resistance is a risk factor for poorer Covid-19 outcomes.

It is important to mention the other half of the energy machine; fat-derived energy. This is an antidote to insulin resistance when it is operating properly.

Fat mobilization takes place with an entirely different process that has trivial or no dependence on insulin, so whatever energy shortfall may be present because of insulin resistance in a carb-burning state shifts to

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188 https://www.telegraph.co.uk/news/2018/03/01/award-winning-comedian-accuses-cancer-research-fat-shaming-launching/
Click on “Comments” to read the discussion.

189 During pregnancy, the placenta induces insulin resistance in women. This is adaptive. The increased glucose in the mother’s bloodstream is absorbed by the placenta and fed to the developing fetus, which requires the glucose for energy because protein and fat is being directed into growth and formation of cell membranes. In women with pre-existing insulin resistance, this “additional” insulin resistance can cross the line into diabetes. This is common enough that it has its own diagnostic name: gestational diabetes. This extra glucose energy may be why babies of older, more insulin-resistant first-time mothers tend to be of higher birth weight and head size.
whatever limitations that may be present in the fat-burning process. For normal healthy humans, who are exposed to short-term carb-restriction and periodic fasting on a regular basis, fat-burning is rarely limited to any significant extent. However, in humans who consistently overeat carbs, fat-burning mechanisms may not have been exercised in years or even decades. Such individuals may have great difficulty in invoking fat-burning metabolism, both beta-oxidation (body-wide cellular fat-burning) and ketosis (liver-centered wholesale fat burning). During this time of difficulty, energy may become even more compromised. Their switch from burning glucose to burning fat will be less than graceful.

Feed a cold and starve a fever is an adage that has been around for centuries. This was recently tested in mice under controlled experiments that verified that is was the glucose content of feeding that was dangerous during bacterial infections.\textsuperscript{190} However, a protective effect was observed for viral infections. This definitely needs to be confirmed for humans, and for Covid-19 in particular where cytokine storm is a common clinical manifestation.

Think of the energy systems as a teeter-totter, either tipped to carb-burning or tipped to fat burning. Each suppresses the other. So when you are burning carbs for energy, your body shuts down your fat-burning systems, opens up fat-synthesis from glucose, and activates fat storage. And when you are burning fat, your carb-burning energy pathways are shut down and carb-synthesis pathways (gluconeogenesis) are activated.

The first state is why overeating carbs is the fastest way to obesity. The extra energy from carb overconsumption gets diverted into fat synthesis, which then gets stored.

The second state is why extremes of ketosis may not be sustainable. The body uses glucose in some ways that are very slow to adapt and fat-derived glucose synthesis from glycerol (from triglyceride) is quite limited. When there is a glucose shortfall, the body uses (catabolizes) protein-derived amino acids for meeting the shortfall. This means a loss of lean body mass (body proteins and enzymes).

This is why many practitioners recommend starting with milder states of ketosis and working up only to moderate states of ketosis. You’d do the same thing with physical exercise.

Some public-health officials and bureaucrats disrecommend ketosis outright for these reasons, and other medical practitioners because keto-acidosis was a cause of death for diabetics in the bad ole days.\textsuperscript{191} Such

\textsuperscript{190} A Wang, S C Huen, H H Luan \textit{et al.} Opposing effects of fasting metabolism of tissue tolerance in bacterial and viral inflammation. \textit{Cell} 166(6): 1512-25, 08 Sept 2016. The authors concluded that the protective effects of beta-oxidation and ketosis on bacterial infection (listerosis) were mediated by improved antioxidant defense, not changes in pathogen levels. But for influenza, no such effect was observed. I find it interesting to speculate that the difference is in how differing infections trigger the oxidative response of immune defense, and influenza and Covid-19 might be dramatically different. As evidence of this, the risk of death by cytokine storm does appear to be atypical for lipid-enveloped viruses.

\textsuperscript{191} T B VanItalie and T H Nufert. Ketosis: metabolism's ugly duckling. \textit{Natur Rev} 61(10): 327-41, Oct 2003. Abstract: Ketones were first discovered in the urine of diabetic patients in the mid-19th century; for almost 50 years thereafter, they were thought to be abnormal and undesirable by-products of incomplete fat oxidation. In the early 20th century, however, they were recognized as normal circulating metabolites produced by liver and readily utilized by extrahepatic tissues. In the 1920s, a drastic ‘hyperketogenic’ diet was found remarkably effective for treatment of drug-resistant epilepsy in children. In 1967, circulating ketones were discovered to replace glucose as the brain's major fuel during the marked hyperketonemia of prolonged fasting. Until then, the adult human brain was thought to be entirely dependent upon glucose. During the 1990s, diet-induced hyperketonemia was found therapeutically effective for treatment of several rare genetic disorders involving impaired neuronal utilization of glucose or its metabolic products. Finally, growing evidence suggests that mitochondrial dysfunction and reduced bioenergetic efficiency occur in brains of patients with Parkinson's disease (PD) and Alzheimer's disease (AD). Because ketones are efficiently used by mitochondria for ATP generation and may also help protect vulnerable
prejudices persist to this day. But ketosis was a regular state of health in our ancestors of only 20, 200 and 2000 generations ago. It is a form of metabolic exercise as essential to our wellbeing as musculoskeletal exercise.

There are also ketosis detractors because of a negative personal experience. If you have been there and done that, please consider trying to biohack the bottleneck in your ketosis metabolism. There are a dozen nutrients that play a role in the turning on and sustained function of ketosis, and there are top-down regulators of ketosis like insulin and leptin that also regulate ketosis via mTOR. Please read on about leptin and mTOR for more information.

**Leptin Resistance**

Leptin is a hormone that helps regulate body weight and body fat storage. It acts as a cell-signaling factor, which qualifies it as a cytokine. It also mediates IL-6 expression (another cytokine, especially associated with cytokine storms).

Leptin is produced by fat cells to signal an adequacy of fat storage. High leptin levels promote leptin resistance, which sabotages the leptin signal and directs the body to increase fat storage.

Disturbances of leptin underlie most obesity.

High leptin, leptin resistance and visceral (abdominal) fat are associated with most of the pre-existing conditions known to be associated with increased morbidity and mortality from Covid-19. In addition to obesity, this includes diabetes, hypertension, and old age.

There are, as of today, no drugs for treating leptin resistance. So few doctors know much about it. However, dietary restriction of carbohydrate and to a lesser extent protein can restore leptin sensitivity.

Leptin levels are one of the input signals to mTOR.

Rather than strain my limited understanding of the massive complexities of leptin (and mTOR), let me suggest links to better expertise available via YouTube (see Ron Rosedale, for example). Dr. Rosedale openly predicts that most people who die from Covid-19 will be found to have elevated leptin.

One of the reasons that leptin levels are not considered clinically is the widespread belief that leptin is primarily regulated by the amount of fat in the body. This leads to the conclusion that control of leptin is a long-term phenomenon that requires significant weight loss (lowering of body fat). However, it is the case that leptin spikes, like insulin spikes only slower, in response to high carbohydrate meal. This spiking of leptin is largely independent of how much stored fat is present. Thus, the leptin level is a result of stored

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192 Masahiro Morita. mTOR coordinated protein synthesis, mitochondrial activity and proliferation. *Cell Cycle* 14(4): 473-80. 15 Feb 2015. Abstract: Protein synthesis is one of the most energy consuming processes in the cell. The mammalian/mechanistic target of rapamycin (mTOR) is a serine/threonine kinase that integrates a multitude of extracellular signals and intracellular cues to drive growth and proliferation. mTOR activity is altered in numerous pathological conditions, including metabolic syndrome and cancer. In addition to its well-established role in regulating mRNA translation, emerging studies indicate that mTOR modulates mitochondrial functions. In mammals, mTOR coordinates energy consumption by the mRNA translation machinery and mitochondrial energy production by stimulating synthesis of nucleus-encoded mitochondria-related proteins including TFAM, mitochondrial ribosomal proteins and components of complexes I and V. In this review, we highlight findings that link mTOR, mRNA translation and mitochondrial functions.
fat and dietary composition, the latter of which can significantly drop leptin levels in less than 24 hours.\textsuperscript{193} Dr. Rosedale also speculates that the two-to-three-fold spikes in leptin play a greater role in leptin resistance than fat-derived leptin.\textsuperscript{194}

Although both insulin resistance and leptin resistance are often both associated with obesity, leptin resistance is the greater influence on development of obesity and gut permeability.\textsuperscript{195} In this study of young adults, insulin resistance was not associated with obesity but was associated with gut permeability. The authors suggest that gut permeability may be an independent risk factor for the development of insulin resistance.

**mTOR**

mTOR (the molecular target of rapamycin, or mammalian target of rapamycin) is a protein kinase which coordinates and moderates the many signals of the body regarding energy consumption and production which drive growth and cell proliferation. The ability of mTOR to promote cell proliferation is the basis for the use of rapamycin to treat cancers.

mTOR suppresses autophagy and sabotages the innate cellular-immunity response. It encourages cell proliferation and mitochondrial proliferation.


\textsuperscript{194} https://www.youtube.com/watch?v=dFSjTIyG9ww Episode 67 of *The Fat Emperor Podcast*, with Ivor Cummins.

\textsuperscript{195} L Mkumbuzi, M M Mfengu, E A Godwill and C Sewani-Rusike. Insulin resistance is associated with gut permeability without the direct influence of obesity in young adults. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy* 13: 2997-3008, August 2020. **Objective:** Recent findings have associated insulin resistance and obesity with increased gut permeability. However, it still remains unclear whether obesity may be the underlining factor for the association between gut permeability and insulin resistance. This study investigated the relationship between gut permeability, measures of obesity, and markers of insulin resistance in young adults. **Materials and Methods:** A cross-sectional quantitative study which enrolled 151 young South African adults was conducted. Anthropometric measurements were performed to assess obesity. Adiponectin, leptin, and zonulin, a marker for gut permeability, were assayed. Insulin and fasting glucose were assayed and used to determine insulin resistance (HOMA-IR), insulin sensitivity (%S) and beta cell function (%B). **Results:** Decreased adiponectin and increased leptin were associated (p<0.05) with obesity. HOMA-IR inversely correlated (p<0.05) with adiponectin but positively with leptin to adiponectin (Lept/ADP) ratio (p<0.05) in females. Markers of insulin resistance were not associated (p>0.05) with obesity. Overweight/obese (O/O) females had a significantly (p<0.01) higher zonulin concentration than lean females. Zonulin positively associated (p<0.05) with body mass index and visceral fat, as well as with HOMA-IR and insulin concentration. Lept/ADP ratio, an inflammatory marker, was associated with risk of insulin resistance. Increased insulin, a maker for insulin resistance, was associated with risk of gut permeability. **Conclusion:** Insulin resistance was associated with gut permeability without a direct influence by obesity in young adults. The lack of relationship between obesity and insulin resistance was possibly mediated by the contribution of obesity to gut permeability. This finding suggests that gut permeability may be a potential independent risk factor for the development of insulin resistance in healthy obese young adults.”
Other Therapeutics of Specific or Potential Interest

This section is for discussing the variety of other therapeutics that might be of relevance to viral infections in general, and Covid-19 infections in particular.

Nattokinase and Lumbrokinase

Nattokinase and lumbrokinase are enzymes that specialize in digesting fibrin peptides and proteins. Those fibrin peptides and proteins determine the coagulation and clotting properties of blood. Coagulation is a pre-clotting phase where soluble fibrin proteins are assembled into complexes that ultimately clot. And clotting is the end-stage where clotting closes wounds that are big enough to bleed.

Nattokinase is extracted from the fermented Japanese food natto. Lumbrokinase is extracted from a species of earthworm. Both of these are available over-the-counter in the US and some other jurisdictions.

Because these enzymes counteract the coagulation and pre-coagulation phases of the clotting process, they may be able to substitute for heparin, which is considered the treatment of choice in hospitals for coagulopathy and clotting pathologies.

(+) review literature, add mechanisms and cite references

In a study of hypertensive patients, nattokinase (with vitamin K₂ removed) was found to reduce systolic and diastolic blood pressure in males more than females, and lower von Willebrand factor in females but not males.¹⁹⁶ Natto food contains both vitamin K₂ and nattokinase.

Thiamine, Riboflavin, NADH, Lipoic Acid and Mitochondrial Support

The Marik protocols for treating sepsis (and Covid-19 infections) include thiamine. Thiamine (vitamin B₁) is involved in both potassium utilization and mitochondrial production of reducing equivalents (NADH) via the Krebs cycle. But thiamine is not the only mitochondrial nutrient that can, when depleted, slow down mitochondrial energy-production pathways. The dehydrogenase enzymes of the Krebs cycle need lipoic acid, riboflavin (B₂) and NAD⁺ (fully coenzymated vitamin B₃) in addition to thiamine (B₁) to function at full flux. Full flux is necessary to optimize redox-buffering capacity.

During a fever, mitochondria operate best in a fasting mode where fat is burned for energy. This may be the derivation of the adage, “Feed a cold, starve a fever,” which has come under attack by mainstream medical sources in recent decades.¹⁹⁷ But the adage advice is good if (1) you are mitochondrially “fit” for

¹⁹⁶ G S Jensen, M Lenninger, M P Ero and K F Benson. Consumption of nattokinase is associated with reduced blood pressure and von Willebrand factor, a cardiovascular risk marker: Results from a randomized, double-blind, placebo-controlled, multicenter North American clinical trial. Integr Blood Press Control 9: 95-104, 13 October 2016. “In the subpopulation with low plasma renin activity levels at baseline (<0.29 ng/mL/h), an increase was seen for 66% of the people after 8-week consumption of nattokinase (P<0.1), in contrast to only 8% in the placebo group.”

¹⁹⁷ The evidence for and against this advice has yet to discriminate between ketosis-enables patients and ketosis-disabled patients. The entire benefit of “starve a fever” is in inducing fat-burning metabolism. In people who routinely overeat carbohydrate foods in accordance with the official USA food-pyramid recommendations, ketosis has been suppressed so efficiently that induction of ketosis is significantly delayed, with temporary adverse effects on the immune system. This does not happen in people who are metabolically fit and capable of inducing beta-oxidation in hours and ketosis in a day. So this advice is compromised by the pre-existing condition of insulin resistance and lack of metabolic fitness in carb-overfed populations.
burning fat, or (2) you supplement mitochondrial nutrients in high doses to “jump start” fat mobilization and sluggish mitochondria.

One of the key players in inducing mitochondrial fitness is carnitine (and acetyl-L-carnitine). The human body is capable of making carnitine, which is one reason why it is medically under-appreciated. But carnitine needs to be “in place” before the switchover from carb-burning to fat-burning can take place, and if that is to be “as soon as possible” because of influenza, Covid-19 or ebolavirus infection, you do not want to wait around while your body figures out that it needs to make more carnitine. So if you are not “fat-burning fit,” supplementing carnitine can greatly shorten the time it takes for mitochondria to burn fat.

Another way to mitigate a carnitine insufficiency is to consume fat rich in medium-chain fatty acids. Medium-chain and short-chain fatty acids do not need carnitine to be burned. So they are immediately available to generate NADH and ATP, even in people with pre-existing conditions who are insulin resistant, diabetic and carb-inhibited. The best natural fat for this is coconut oil, which is 60% medium-chain fatty acids. Palm oil is second choice at about 50% MCT, with milk fat (butter and cream) trailing the pack at 5%. All mammal milk contains roughly 5% MCT fat to facilitate newborn survival.198

Temperate vegetable oils have no MCT content. These include corn, soybean, canola, cottonseed, olive, sesame, safflower, sunflower and sesame-seed oils.

As it turns out, the feed-a-cold-starve-a-fever advice does not apply to fat. It applies to carbohydrate and protein, both of which have the ability to suppress fat-burning systems when in excess.

There is one study of nutritional supplementation that occurred by accident when insufficient multivitamins were delivered to a group of five emergency treatment units in Liberia and Sierra Leone which were treating Ebola viral infections with a common treatment protocol. This resulted in only a subset of patients taking the multivitamin.199 This was not planned, but it served to create two populations with enough similarity for an analysis.200

Of the 261 patients who received early multivitamin treatment, 46% survived. Of the 163 patients who did not get the multivitamin supplements, 36% survived.

They then matched patients based on pre-existing conditions, sex, age, etc. During their report they write: (1) the mortality among matched patients differed significantly with supplementation, 53.5% for those receiving multivitamins, 66.2% for those not receiving multivitamins, (2) the mortality did not differ between those getting vitamin A plus multivitamin and those just getting vitamin A, and (3) mortality did differ between matched patients receiving C plus multivitamin versus vitamin C without multivitamin.

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198 If you feel worse taking MCT-rich oils over time, you may have a medium-chain beta-oxidase enzyme inefficiency caused by a genetic polymorphism. In this kind of situation, poor beta-oxidation causes increased omega-oxidation, which can be seen on a urine organic acids test as elevated suberic and adipic acids.

199 There were actually two multivitamin formulas being used, Medipharm and CSPC. Both contained vitamins A and D, and B1, B2 and B3, although the amounts varied. One contained a tiny amount of vitamin C. The quantities reported: (Medipharm / CSPC / US RDA). Vitamin A (2500 IU / 800 IU / 2310-3000 IU), Vitamin D3 (300 IU / 200 IU / 600 IU), vitamin B1 (1 mg / 0.5 mg / 2 mg), vitamin B2 (0.5 mg / 0.5 mg / 1.1-1.3 mg), vitamin B3 (7.5 mg / 7.5 mg / 14-16 mg), vitamin C (15 mg / none / 65 mg), no vitamin B6, B12, folic acid, etc.

I find it hard to judge this finding because of the methodological difficulties in matching patients, the low dose of nutrients that are present in a multivitamin supplement, regional differences that may hide hidden variables, and the vague language used in reporting what was done and the therapeutic context of the results. The authors write, “In addition to the multivitamins, the IMC guidelines also recommended Vitamin A (1000 retinol units on Day 1 and 2) and Vitamin C (500 mg three times daily) supplementation, though actual care varied based on supply availability.”

Melatonin
Melatonin is well known as a dietary supplement that promotes sleep and can help with symptoms of jet lag. But what is not so well known is melatonin’s role as the primary antioxidant in mitochondria.201 (+) add content re Doris Loh202 re hemochromatosis, RBC lysis, hypercoagulation and thrombosis. (+) cyclophilin A (+) insert more references for development here.
The “antioxidant cascade” of melatonin.203 Tan.204 Melatonin directly scavenges hydrogen peroxide.205 Melatonin 2015 review at meta level.206 “These features make melatonin a potent endogenously-occurring antioxidant that protects organisms from catastrophic oxidative stress.”

Quercetin and other bioflavonoids
(+ ) insert content, antioxidant, classes, flavones, isoflavones, anthocyanins, etc.

Potential Involvement of Neurological Covid-19
It is well known that lipid-enveloped viruses have the potential to infect the brain. Central nervous system infection by viruses is known as viral encephalitis, and this has already been observed in Covid-19 cases at autopsy and with viral analysis of cerebrospinal fluid from spinal taps.

There are multiple published papers making claims

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206 Dun-Xian Tan, Lucien C Manchester, Eduardo Esteban-Zubero, Zhou Zhou and Russel J Reiter. Melatonin as a potent and inducible Endogenous antioxidant: Synthesis and metabolism. Molecules 20(10): 18886-906, October 2015. “In addition to its enzymatic degradation, melatonin is metabolized via pseudoenzymatic and free radical interactive processes. The metabolic products of these processes overlap and it is often difficult to determine which process is dominant. However, under oxidative stress, the free radical interactive pathway may be featured over the others.”
There are questions about how to diagnose this. One sign that has been cited is fluid on the brain. Yet that happens from a lowering of basal metabolic rate that has no necessary or essential dependence on viral infection of the brain. There are also many reports of sudden and delayed psychiatric symptoms, like schizophrenia and psychosos episodes associated with Covid-19. But again, the assumption that these conditions are necessarily caused by CNS viral infection is unwarranted. and psychosis. stion is, however, open as to the signs , and viruses are one way that inflammation can be induced

**Vaccines, Vaccination and Vaccine Interference**

There is lots of talk about the rush to develop a vaccine for Covid-19. Huge profits are at stake for anybody who succeeds in getting a vaccine to market.\(^\text{207}\)

The general problem with coronavirus vaccines during epidemics is that the timeframes are disparate. It takes years to develop and test a vaccine, and a typical coronavirus outbreak lasts for months. You can see this time incompatibility in the way influenza vaccines are developed; some experts guess what the next flu outbreak will be and arranges for a vaccine to be made a year in advance, and sometimes they guess right. Odds of that are roughly 50:50, but this is still considered a successful vaccination policy.

Coronaviruses and influenza viruses are both lipid enveloped. This means that they both are made with a fatty coating that hides most of the viral components from immune surveillance. Such lipid-enveloped viruses are the toughest candidates by far for vaccine development. And those few high-efficacy vaccines that we do have are based on “live,” attenuated (weakened) viruses, which do not induce the same degree of immune memory as the unattenuated (full blown) viruses.

The problems with creating a vaccine for SARS-CoV-2 and Covid-19 are immense. Similar attempts to create a vaccine for the SARS epidemic of 2002-3 were not only unsuccessful, they were counterproductive. The vaccines potentiated the susceptibility of those vaccinated rather than attenuated them. The effort was discontinued because of this. Now we are seeing a new phenomenon called vaccine interference, in which vaccines affect each other and other viral diseases they were not intended to affect.

Vaccine interference can be both positive and negative. In other words, a vaccination can increase or decrease your immune system’s reaction to a future infectious event. There are two reports of flu vaccinations that may have increased mortality from Covid-19, and one report where BCG vaccination may have decreased mortality from Covid-19.\(^\text{208}\)

What does this mean, practically?

If vaccine interference is made part of the safety testing process, it’ll be 2-3 years instead of 1-2 years until we have a covid-19 vaccine that has passed safety testing.

See “The SARS-CoV-2 Neo-Vaccine Boomerang” on page 203 for further information on potential mRNA vaccine problems.

\(^{207}\) Even if the vaccine causes serious adverse events, the profit will be high for the duration of its commercial use. There are laws that exempt vaccine manufacturers from all liability for vaccine “damages.” Because of this liability exemption, there is no disincentive for producing an unsafe vaccine. The only disincentive for the company is in public relations, to which a “we did our best” and “we had good intentions” statements are effective responses. The political disincentive for the US government is also of minor affect given their extreme commitment to a pro-vaccine policy, even when outcomes are tragic.

The Rush to Vaccinate

There are massive political and economic reasons why Covid-19 vaccines will be immediately deployed. One is the massive success of the Covid-19-fear campaign. This fear is now pervasive in the world and especially the United States, where doctors and hospitals have been paid to make misleading and false reports of Covid-19 fatalities. As a consequence, people are lining up to get vaccinated with the “latest and greatest” Covid vaccine to be placed in the pending-approval queue.

The bad news is that some people will likely be seriously damaged by this rush to vaccinate.

The good news is that we will get vaccine test results faster than normally possible. The deployment of Covid vaccines in the middle of the 2020-2021 flu season will give us efficacy data for preventing or lessening Covid-19 severity, and possible interference data on how flu and Covid-19 vaccines interact with each other and the viral infections they are intended or not intended to remedy.

The Rush to Drug Approval

Covid-19 fear is putting pressure on politicians and regulators alike to approve treatments specific for Covid-19. Their agenda is to “approve something” to ease the political pressure. The public is not just interested in the promised vaccines, they want to get Covid-19 drugs for treatment, too.

The rush has negative consequences. Not paying enough attention to risks is one. The FDA’s recent rapid approval of remdesivir for emergency use may be such an example. A recent paper announced that it has a powerful, irreversible inhibiting effect on an important detoxification enzyme that may be counterproductive for treating Covid-19.\(^{209}\) This effect was unknown at the time of approval and this contraindication was not known by clinicians using remdesivir with other pharmaceuticals.

A more basic risk is rushing approval of a drug that does not work as well as the pharmaceutical company reports. This is a pervasive problem because there are no regulations requiring negative studies to be reported to the FDA. The month before the above research was reported, the Guideline Development Group of the WHO downgraded its recommendation for remdesivir to a “conditional recommendation against” its use, citing several studies showing no benefit to Covid-19 survival, need for respiratory support or to hospitalization stay. The one study run by the US NIH that showed clinical efficacy is not contradicted by multiple studies.

One of the other problems with early approval is the influence of politics and egos on studies that are equally rushed. The WHO’s Solidarity trial was one of these, finding a lack of support for not just remdesivir, but also hydroxychloroquine, which is actually effective against Covid-19 when used properly. The WHO study just used it improperly, coming to the wrong conclusion. None of these kinds of details are ever considered in media stories or in professional attitudes based on such media reporting. They are the perspective sacrificed by the rush to approval.

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\(^{209}\) Yue Shen, William Eades and Bingfang Yan. Remdesivir potently inhibits carboxylesterase-2 through covalent modifications: signifying strong drug-drug interactions. *Fundamental and Clinical Pharmacology* Short Communication, 23 December 2020. doi: 10.1111/fcp.12643. Abstract: “Remdesivir was recently approved to treat COVID-19. While this antiviral agent delivers clinical benefits, several safety concerns in many cases have been raised. This study reports that remdesivir at nanomolar concentrations inhibits carboxylesterase-2 (CES2) through covalent modifications. CES2 is a major drug-metabolizing enzyme. The combination of high potency with irreversible inhibition concludes that cautions must be exercised when remdesivir is used along with drugs hydrolyzed by CES2.”
Pharmaceuticals

Descriptions of non-natural pharmaceuticals can be found in Appendix D (starting on page 194).

Stem Cells

The first blinded study of the therapeutic effects of stem cells on Covid-19 patients has been published.

The findings are statistically significant for mortality (84% decrease), survival (doubled) and speed of recovery (more than doubled). See the illustration at right.

The authors write, “The results of this trial indicate that UC-MSC infusions in COVID-19 with ARDS [acute respiratory distress syndrome] are safe. Moreover, UC-MSC [umbilical cord mesenchymal stem cells] treatment was associated with a significant reduction in SAEs [severe adverse events], mortality, and time to recovery, compared with controls.” Among adverse events, the more severe, the greater reduction; mild adverse events did not differ.

The lowering of inflammatory cytokines by stem-cell treatment was also statistically significant.

Nebulization

(+) Lung delivery system for water-solubles and liquids.

(+) Colloidal silver for opportunistic bacterial and fungal infections in late-stage lung disease.

(+) Administration of Na-C, micellized A, methylene blue, etc.

Dairy Products

(+) Stop eating dairy products. Keep your sinuses and lungs unencumbered.

Alkalinizing foods can aggravate lung and skin diseases.

Dairy can worsen lung and sinus infections via thickening mucous.

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210 G Lanzoni, E Linetsky, D Correra et al. Umbilical cord mesenchymal stem cells for Covid-19 acute respiratory distress syndrome: A double-blind, phase 1/2a, randomized controlled trial. Stem Cell Translational Medicine, 5 January 2021. doi: 10.102/sctm.20-0472. This study was well balanced in most pre-existing conditions. Specific comorbidities (treatment:controls) were: diabetes (5:6), hypertension (7:9), obesity (11:5), cancer (0:1), heart disease (1:3), heparin (12:12), heparin prophylactic only (9:7), therapeutic heparin (3:5), remdesivir (9:7), convalescent plasma (3:4), corticosteroids (10:9), tocilizumab (1:4), hydroxychloroquine (1:2), and alteplase (0:2).
Viral Quirks

Some of the viruses cause unique pathologies. Here are ones that I know about.

Potassium Depletion from Coronavirus

Doctors at Wenzhou Hospital in mainland China\textsuperscript{211} have identified a novel potassium-depletion effect in a majority of those hospitalized with coronavirus. In China, they measure plasma potassium, which is slightly different procedure that the serum-potassium testing done in the USA. Plasma readings from unclotted blood (mmol/L) are about 0.5 lower than serum readings from clotted blood (mEq/L).

The “normal range” for potassium is 3.5 to 5.5 mEq/L, and most apparently “healthy” adults test slightly low, between 4.1 and 4.4 mEq/l, because hypometabolism is fairly common and it is considered “normal” by most physicians—and the statisticians who set the normative ranges. In the elderly, the normative range is sometimes considered to be 3.5 to 5.0 mEq/L for this same reason; metabolism declines with age.

But in the Chinese hospital-patient population in Wenzhou, 22\% of coronavirus patients were considered “severe hypokalemic” with below-normal plasma potassium (<3.0 mmol/L) and another 40\% were considered “hypokalemic” with low-normal potassium readings (below 3.5 mmol/L).

That’s six out of ten patients with observable depletion of potassium. Their hypothesis is that “SARS-CoV-2 binds angiotensin-I-converting enzyme 2 (ACE2) of the renin-angiotensin system (RAS) and causes prevalent hypokalemia.” The effect was confirmed by measuring urine loss of potassium, which was found, and which correlated with the degree of potassium depletion. Those patients with “underlying disease” were more likely to have below-normal and low-normal potassium results. And those patients with the most severe potassium depletion were statistically more likely to have a higher body temperature (0.4°C, 0.7°F).

“Patients responded well to K supplementation when they were inclined to recovery.” This suggests that potassium loss is only a minor player in the overall coronavirus pathology. Nevertheless, this finding does suggest that monitoring of urine potassium will tell when the renin-angiotensin system regains its functionality.

This potassium-depletion effect in Covid-19 patients has now been supported by a preprint from Italian researchers.\textsuperscript{212}

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\textsuperscript{211} D Chen, X Li, Q Song, et al. Hypokalemia and Clinical Implications in Patients with Coronavirus Disease 2019 (COVID-19). medRxiv preprint. “Gastrointestinal symptoms were not associated with hypokalemia among 108 hypokalemia patients (P>0.05). Body temperature, CK, CK-MB, LDH, and CRP were significantly associated with the severity of hypokalemia (P<0.01). 93\% of severe and critically ill patients had hypokalemia which was most common among elevated CK, CK-MB, LDH, and CRP. Urine K+ loss was the primary cause of hypokalemia. severe hypokalemia patients was given 3 g/day, adding up to an average of 34 (SD=4) g potassium during hospital stay. The exciting finding was that patients responded well to K+ supplements when they were inclined to recovery. CONCLUSIONS: Hypokalemia is prevailing in patients with COVID-19. The correction of hypokalemia is challenging because of continuous renal K+ loss resulting from the degradation of ACE2. The end of urine K+ loss indicates a good prognosis and may be a reliable, in-time, and sensitive biomarker directly reflecting the end of adverse effect on RAS system.” Hypokalemia was also associated with C-reactive protein (CRP) as well.

A century ago, Dr. Emanuel Revici (see Appendix A) connected potassium to metabolic rate. He described the association as “potassium utilization,” which may be a different phenomenon than what is seen in Covid-19 infections. We cannot tell from the clinical data of the two studies because potassium utilization is determined by the potassium difference between the cells and the serum, whereas potassium depletion is determined by only urine and serum test data. Without knowing the potassium status of the cells, we cannot distinguish low potassium from potassium overutilization.

But I believe there is reason to connect the two. First, metabolic rate is strongly changed during viral infections, especially when inflammation and fever are present. Second, low metabolic rate is directly associated with viral susceptibility. Third, the pre-existing conditions that are directly associated with hypometabolism are all associated with adverse Corona-19 outcomes. Fourth, there is a negative correlation between potassium utilization and water utilization. So when people increase their metabolic rate by switching from glucose-derived energy to fat-derived energy, cells absorb water and excrete potassium, and the tissues dump water with the excess potassium.

Many people have a direct experience of this shift in water use when starting weight-loss program that restricts carbohydrate or calories. The initial downturn in energy from diet is offset after 2-4 days by the induction of beta-oxidation and ketosis, which are fat-burning pathways. Once fat is being burned, metabolic rate goes up, which results in cells absorbing a small proportion of the tissue water burden (edema), the rest of which is dumped into the blood and excreted in urine. What people notice is that pounds (half-kilos) of water are dumped. So the weight-loss dynamic is biphasic, many pounds of water are lost in days followed by only ounces of fat per week or month.

This rapid water loss is an almost universal experience of dieting and fasting.

There is also another connection that may tie potassium to metabolism and coronavirus: thiamine. Thiamine is not only part of the successful Marik protocol for treatment of sepsis and Covid-19 infection, it is an essential vitamin needed for mitochondrial energy production. The three dehydrogenase complexes of the Krebs cycle require thiamine, lipoic acid, NAD+ and riboflavin as cofactors.

Why thiamine, and not riboflavin, niacin, lipoate, etc. would be singled out? I do not know. The only hypothesis I can suggest is fairly complicated: that mercury may be involved. There are published reports of morbid and fatal thiamine deficiencies in gold miners in French Guiana. Although these miners use liquid mercury to extract gold from the local rock, the connection between acute thiamine deficiency and mercury exposure was not considered. Yet mercury is known to inactivate thiamine by oxidative coupling to form thiochrome, which is a fluorescent quantitative indicator for detecting low-level mercury levels.

There is also a recent hypothesis that “oxidatively emergent” mercury toxicity may underlie chronic obstructive pulmonary disease (COPD) based on a couple of spectacular clinical responses to emeramide therapy (see page 149). Emeramide is an irreversible mercury chelating agent, which was not expected to have any clinical use in treating COPD. But its striking efficacy suggested an unknown connection to mercury. Emeramid scientist Boyd Haley hypothesized that local oxidative stress in the lungs could release mercury by oxidizing glutathione, which binds mercury when glutathione is reduced but not when it is oxidized.213

213 The specific pathology would be mercury displacing iron in the lung tissue’s mitochondrial complexes responsible for generating ATP energy. There are three iron-sulfur complexes that serve as electron hosting sites in Complex I, Complex III and Complex IV which are linear, planar and cubic in structure, with one, two and four iron atoms, respectively. The iron-
Indeed, the lung pathology associated with Covid-19 is conspicuously unlike any standard pneumonia presentation and ventilation has an 80-90% mortality rate. So might this lung pathology be primarily oxidative rather than typically infectious?

Fibrosis is one clue to support that. Oxidative stress that would compromise the glutathione redox couple (the ratio of reduced to oxidized glutathione) inside the cell where mitochondria live would also compromise the vitamin C redox couple outside of the cell. In the extracellular space (matrix), vitamin C is necessary to form cross-links between adjacent collagen fibers to fully mature freshly deposited collagen.

As collagen ages, matrix metalloproteinases (MMPs) digest “expired” collagen, the first step in replacing old collagen with new collagen. The activation of MMPs is an inflammatory influence. This has little systemic effect when the MMPs do their job and matrix stability and function is restored. But in the presence of a vitamin C insufficiency, the MMPs fail to restore healthy collagen and inflammation becomes sustained. MMPs remain in a collagen-digesting state and collagen turnover becomes perpetual instead of episodic. This promotes larger-scale inflammation and fibrosis. Fibrin protein is the Band-Aid for bad collagen. Fibrin provides structural stability when collagen cannot do the job.

The solutions to this multi-faceted failure is to restore redox-buffering capacity. By shutting down oxidative stress, the glutathione becomes reduced again and capable of binding loose mercury within cells. And vitamin C becomes reduced to properly participate in collagen maturation in the extracellular matrix.

Restoration of redox buffering can happen on either side of the cell membrane. Raising cellular energy-production capacity means more recycling of oxidized glutathione to reduced glutathione by NADH and NADPH. Flooding the body with vitamin C restores matrix redox buffering, which couples to improved ascorbate-mediated glutathione recycling inside the cell. Vitamin C and glutathione are coupled together, help one and you help the other.

So what we might have here is an intrinsic connection between metabolic rate and (1) potassium utilization, (2) potassium loss, (3) mercury containment by glutathione, and (4) collagen maturation by ascorbate.

Will anybody bother to investigate this?

**Arginine Growth Dependence of Herpesvirus**

The herpesvirus family of viruses appear to be more active in an arginine-rich, lysine-poor environment. Lysine supplement have been used for half a century to reduce the frequency and severity of herpes flare-ups.

Arginine and ornithine participate in the urea cycle. Ornithine picks up urea to become arginine, and then arginine is hydrolyzed to release the urea. So supplemented arginine raises ornithine levels, and supplemented ornithine raises arginine levels. Because of this, ornithine must be considered equally contraindicated as arginine for control of a herpes outbreak.

Nuts and seeds are naturally high in arginine. Popular and scientific sources for specific amounts of arginine in different kinds of seeds and nuts are not consistent, but as a general rule, 1-4 grams of arginine per cup or 100 grams quantity of nuts is a general rule of thumb, with almonds, pine nuts, walnuts, pumpkin seeds and peanuts being towards the high end.

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sulfur bond is relatively strong when sulfur is reduced, but the mercury-sulfur bond is stronger. This would theoretically allow oxidatively-released mercury to replace iron and sabotage electron transport in mitochondria.
Coagulopathy and Clotting from Coronavirus

It is now becoming clear that Covid-19 infections are associated with a broad combination of coagulopathy and clotting pathologies. Tiny blood clots are found in lung tissue and elsewhere. There are blood-oxygenation deficits. There is widespread fibrosis, not just of the lungs. And some young people without known pre-existing disease conditions are dying of strokes.

(+ ) add further clinical findings relating to viscosity and clotting.214

It’s not that this does not happen with other viruses. Many of the “switches” for coagulopathy, like inflammation, estrogen and cytokines, are present with most viral infections. But the degree of the coagulation effect seems markedly more serious with Covid-19 infections.

In hospitals, heparin is used to reverse coagulopathies and lower clotting risks. At home, nattokinase and lumbrokinase (see page 114) are potential substitutions for heparin, which is not well absorbed through the GI tract. On rare occasions, heparin has been used clinically via sublingual administration, but the taste is sufficiently unpleasant that compliance is problematic. Nattokinase and lumbrokinase are enzymes that come in a capsule, making dosing simple. These specific enzymes break specific peptide sequences that are found on fibrin peptides and proteins.

The clotting cascade has pre-coagulation, coagulation and clotting phases. In the transition from pre-clotting to coagulation, the assembly of fibrin subunits into complexes causes viscosity changes in blood. Thick blood does not flow gracefully, and thereby fails to perfuse tissues efficiently. This results in diminished oxygen delivery from the lungs to the deep tissues, and decreased transport of deep-tissue CO2 to the lungs and deep-tissue waste products to the liver and kidneys. The enzymes in nattokinase and lumbrokinase supplements inhibit these early, pre-clotting changes, keeping the blood thin and flowing.

Von Willebrand Factor

A recent hypothesis has been offered that von Willebrand factor is a possible cause or marker of the vascular, coagulation and clotting pathologies of Covid-19.215

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215 A Yu Aksenova. Von Willebrand factor and endothelial damage: A possible association with Covid-19. Ecological Genetics 18(2): 5-8, 2020. “Here I discuss a possible association of COVID-19 complications with von Willebrand factor (VWF) level and endothelial damage. VWF is an important prognostic marker of endothelial dysfunction and its level fluctuates depending on age. VWF level is also variable depending on sex and race. Importantly, chloroquine, a drug that showed potential efficacy for Covid-19 treatment, can influence VWF secretion and consequently its level and activity. I propose that VWF level and activity might be predictors of the Covid-19 morbidity and mortality; moreover the VWF might be involved in the pathogenesis of the disease. I suggest that a comprehensive study of VWF level in SARS-CoV-2 positive groups of people with mild and severe course of the disease should be undertaken.”

“VWF is an essential factor of the blood coagulation system which is synthesized and secreted by the endothelial cells. VWF multimers secretion from intracellular organelles known as Weibel-Palade bodies is required for platelet adhesion to the damaged vessel walls. Importantly, VWF level in plasma is an indicator of endothelial activation and damage. VWF is also a marker of pulmonary endothelial injury and some studies suggest that level of VWF can be linked to ARDS and Acute Lung Injury (ALI). It should be noted that autophagy plays an essential role in VWF secretion. Moreover, chloroquine the drug
Bradykinin Storm

(+ The most recent reported quirk for SARS-CoV-2 is a possible bradykinin storm.

Tissue plasminogen activator (t-PA) is released by the epithelium (cells that line blood vessels) to activate plasminogen by converting it to plasmin. t-PA is a serine protease (protein-breaking enzyme) that detaches a piece of the plasminogen protein to make it active against clots. Plasmin is a major clot-inhibiting and clot-digesting protein of the blood that is released after a clot has successfully formed after a clot has done its job and is no longer needed.

Interference in the t-PA system is hypothesized to play a role in cardiovascular disease. The idea is that decreased t-PA capacity decreases the influence of plasmin on the surface of a forming clot and the clot becomes larger than needed. It’s like a teeter-totter with clot-forming tendencies on one side and plasmin on the other and the tipping point is when the clot has stopped the bleeding and the plasmin stops further clotting.

One of the factors that may play an important role in sabotaging the plasmin system is antibodies to t-PA. Bradykinin triggers t-PA release.

t-PA release is lower in obese people at 12.5, 25 and 50 ng/100 ml tissue /minute

(+ Update this section.

Selenium depletion from hemorrhagic viruses and HIV

The hemorrhagic viruses (ebola, Marburg virus, etc.) have a selenium-sequestration mechanism in their RNA genome. There are UGA codon repeats that specify selenocysteine incorporation into viral proteins, which reportedly depletes infected individuals of selenium reserves. During this process, hemorrhagic symptoms tend to be minimal, until selenium bottoms out and the supply of vitamin C abruptly crashes from lack of selenium-mediated recycling. At that point, hemorrhagic symptoms manifest in striking rapidity.

Although these viruses are described by the word hemorrhage, which means bleeding, the condition is not limited to bleeding. Fluids of all kinds are leaking from containment, not just blood. The true underlying pathology is loss of tissue integrity, the biochemical feature of which is deterioration of the collagen infrastructure of the extracellular matrix (the structure or scaffolding in the space between cells). The extracellular matrix is occupied by, and structurally stabilized by, an interconnected network of collagen protein fibers, which are maintained by a large family of extracellular MMP enzymes (matrix
metalloproteinases) that also serve cell-signaling functions involved in coordinating tissue-level homeostasis.\textsuperscript{216}

There are multiple human diseases and conditions that are characterized by pathologies of the extracellular matrix. On the common side is ease of bruising, which represents a general loss of matrix toughness. So many people have this condition that it is virtually ignored. More serious is cardiovascular disease, especially when it has progressed from high MMP activity (an early, inflammatory stage) to plaque formation (an advanced, immune-active stage). Next on the severity list is classic scurvy, the disease caused by a chronic lack of sufficient vitamin C in the diet, and in the extracellular matrix where things are falling apart. Teeth are loosening, gums swell and bleed, bruising happens spontaneously, subcutaneous bleeding occurs, wounds heal exceedingly slowly with extensive scarring, and swelling and pain in joints. Lastly, we have hemorrhagic fevers, which can be considered sudden-onset scurvy, where all of the above pathologies are occurring but are not visible because people die so fast, or recover too quickly.

**HIV**

The HIV virus is also associated with selenium losses, although they are small and insidious. It takes years for the selenium deficiency to build up to the point of compromising redox potential, sabotaging cell-signaling pathways and facilitating widespread opportunistic infections.

Peter Duesberg is one of the many honorable scientists who have had their characters assassinated by US public health officials with the assistance of US media. He had the scientific integrity to point out the conspicuous fact that the HIV-AIDS association utterly fails to meet Koch’s postulates, by which a causal inference can be made between the association of an infectious agent with a disease. There are four conditions that must be met:

1. The infectious agent had to be present in every case of the disease. This was falsified.
2. The infectious agent had to be isolatable from the infected host, and cultured. This was also falsified.
3. The disease is reproduced when the cultured infectious agent is given to a new host. Also falsified.
4. The infectious agent must be again recovered from the new host.

Koch’s postulates are not scientific requirements, but rather an ad hoc standard to minimize error on the part of scientists, doctors and public health officials. Sometimes one or more of the postulates is impossible to undertake. For examples: (a) Sometimes the infectious agent cannot be cultured. Only about 1\% of infectious bacteria can be cultured by today’s hospital laboratories. But they can be positively

\textsuperscript{216} D Rodriguez, C J Morrison and C M Overall. Matrix metalloproteinases: What do they not do? New substrates and biological roles identified by murine models and proteomics. *Biochimica et Biophysica Acta (BBA) - Molecular Cell Research* 1803(1): 39-54, Jan 2010. A Review article with more questions than answers. (1) “The biological roles of the matrix metalloproteinases (MMPs) have been traditionally associated with the degradation and turnover of most of the components of the extracellular matrix (ECM). This functional misconception has been used for years to explain the involvement of the MMP family in developmental processes, cell homeostasis and disease...” (2) “MMPs regulate cell behavior through finely tuned and tightly controlled proteolytic processing of a large variety of signaling molecules that can also have beneficial effects in disease resolution.” However, the almost complete lack of understanding of the subtleties of these interactions give us no practical means of pharmacological intervention in situations of matrix destabilization or collapse. What we are left with is natural matrix support strategies, like ascorbate, copper, silicon, energy substrates, and reducing agents. Scurvy is the classic example of matrix destabilization and hemorrhagic fevers are the current example of matrix collapse, with coronavirus as a special case. High-dose intravenous ascorbate is clinically effective for all three.
identified by their RNA and DNA sequences. And (b) sometimes, there is no animal model for the re-infection step, and the disease cannot be ethically given to a human being.

HIV fails at least three of the four postulates. There are people with AIDS who do not test positive for the HIV virus. At least a hundred cases, so far. That falsifies postulate 1, 2 and 4. There are a huge number of people who are known to be infected with HIV but are showing no signs of AIDS after extended time periods. This falsifies postulate 3.

Public health officials explain away the AIDS-without-HIV cases by attributing it to an arbitrarily small percentage of total cases. But the fact is that those cases prove that HIV is not the only way to develop AIDS. Public health officials also explain away the huge numbers of no-signs-of-AIDS cases by an arbitrarily long disease-development timeframe. This may seem reasonable until one considers that timeframe looks likely to be longer than the human lifespan.

So why do we believe so strongly that HIV causes AIDS when the scientific case for that causal relationship is do dismally inadequate?217

Public health officials say so.

Other hypotheses have been advanced,218 yet none have been taken seriously after the public-health commitment to the HIV-causes-AIDS hypothesis was publicly endorsed in 1984.

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217 E Papadopoulos-Elepulos, V F Turner, J Papadimitriou, et al. A critique of the Montagnier evidence for the HIV/AIDS hypothesis. Med Hypotheses 63(4): 597-601, 2004. “By 1984 HIV was almost universally accepted to be the cause of AIDS. However, 20 years later, HIV cannot account for the phenomena for which the retroviral hypothesis was proposed, namely, Kaposi's sarcoma, decrease in T4 lymphocytes and thus the opportunistic infections in AIDS patients which were assumed to be the direct results of this decrease. Agents other than HIV to which patients belonging to the AIDS risk groups are exposed cause decrease in T4 cells. Neither have the main predictions of the HIV hypothesis been fulfilled. HIV seropositivity in the developed countries still remains restricted to the original high risk groups, no HIV vaccine exists, and no successful animal model has been developed. In this communication, we critically analyse the evidence which in 1983 was claimed to prove the existence of HIV.”

218 L Broxmeyer and Alan Cantwell. AIDS: “it’s the bacteria, stupid!” Medical Hypotheses 71(5): 741-8, November 2008. From the abstract: “Acid-fast tuberculous mycobacterial infections are common in AIDS and are regarded as secondary ‘opportunistic infections.’ According to the National Institute of Allergy and Infectious Diseases, TB is the major attributable cause of death in AIDS patients. Could such bacteria play a primary or causative role in AIDS? Certainly. In screening tests for HIV, there is frequent, up to 70%, cross-reactivity, between the gag and pol proteins of HIV and patients with mycobacterial infections such as tuberculosis.” Continuing: “Since 1982 Cantwell et al found acid-fast bacteria closely related to tuberculosis (TB) and atypical tuberculosis in AIDS tissue. On the other hand, molecular biologist and virologist Duesberg, who originally defined retroviral ultrastructure, has made it clear that HIV is not the cause of AIDS and that the so-called AIDS retrovirus has never been isolated in its pure state. Dr. Etienne de Harven, first to examine retroviruses under the electron, agrees. In 1993 HIV co-discoverer Luc Montagnier reported on cell-wall-deficient (CWD) bacteria which he called ‘mycoplasma’ in AIDS tissue. He suspected these as a necessary ‘co-factor’ for AIDS. Remarkably, Montagnier remained silent on Cantwell’s reports of acid-fast bacteria which could simulate ‘mycoplasma’ in AIDS tissue. Mattman makes clear that the differentiation between mycoplasma and CWD bacteria is difficult at best and cites Pachas’s 1985 study wherein one mycoplasma was actually mistaken for a CWD form of a bacterium closely related to the mycobacteria. It is important to realize that the statement ‘HIV is the sole cause of AIDS’ is just a hypothesis.”
Cytokine Storm

Cytokines are inflammatory mediators which guide the inflammatory response to infection and injury. Cytokine storms are the result of the cytokine system going off the rails in responding to the infection or oxidative stress. Although most physicians may not know this, but antioxidants, and specifically reducing agents, are a remedy for this condition. This includes vitamin C, glutathione, cysteine, N-acetylcysteine and lipoic acid.

The role of nuclear factor kappa-B in cytokine storms may be foundational. NFkB is required for several genes expressing inflammatory factors, many of which are seen in acute respiratory distress syndrome (ARDS) and other conditions involving inflammation. Antioxidant therapies are reported to mediate NFkB signaling and decrease NFkB itself. The list of antioxidants includes glutathione, N-acetyl cysteine (a glutathione precursor and mimic) and alpha lipoic acid. Despite finding deficiencies of glutathione in alveolar fluid from patients with ARDS and sepsis (out of control microbial infections), this has yet to be investigated by public-health agencies.

(+ redox cross-references
(+ C cross-references
(+ selenium cross-references
(+ refer to Appendix C.

Redox Therapies

(+) Vitamin C, see pages 78, 90, 175.
(+) provide list and cross-references.
(+) cross-reference methylene blue??
(+ + Antioxidants stabilize gut microbiome and butyrate production by anaerobes exposed to oxygen. This indicates that oral and IV vitamin C can stabilize anaerobes against oxidative stress and keep the colonocytes fed with microbial butyrate.

219 E R Pacht, A P Timeraqn, M G Lykens and J Merola. Deficiency of alveolar fluid glutathione in patients with sepsis and the Adult Respiratory Distress syndrome. Chest 100(5): 1397-1403, Nov 1991. Abstract: The adult respiratory distress syndrome (ARDS) is a devastating clinical illness characterized by refractory hypoxemia and high-permeability pulmonary edema. Reactive oxygen species such as hydrogen peroxide and hypochlorous acid may play a key role in the pathogenesis of the acute lung injury. Glutathione (GSH) is a tripeptide that is able to react with and effectively neutralize oxidants such as hydrogen peroxide and hypochlorous acid. The present study found that the alveolar epithelial lining fluid of patients with ARDS was deficient in total GSH compared to normal subjects (21.7μmol ± 7.8 μmol vs 91.8μmo1 ± 14.5μmo1; p = 0.002). In addition, if GSH was measured in unconcentrated bronchoalveolar lavage (BAL) fluid and indexed to total BAL protein, there was also a deficiency in patients with ARDS compared to normal subjects (0.004 ± 0.003 nmol of CSH per microgram of total protein vs 0.026 ± 0.005 nmol of GSH per microgram of total protein; p=0.002). Since patients with ARDS are subjected to an increased burden of oxidants in the alveolar fluid, principally released by recruited neutrophils, this deficiency of CSH may predispose these patients to enhanced lung cell injury."

220 M Millio, N Armstrong, S Khelaifia, E Guihot, M Richez, J_C laiger, G Dubourg, E Chabriere and D Raoult. The antioxidants glutathione, ascorbic acid and uric acid maintain butyrate production by human gut Clostridia in the presence of oxygen in vitro. Scientific Reports 10(7705): 2020. Abstract: Uncontrolled oxidative stress, reported in Salmonella and HIV infections, colorectal cancer or severe acute malnutrition, has been associated with anaerobic gut microbiome alteration, impaired butyrate production, mucosal immunity dysregulation and disruption of host-bacterial mutualism. However, the role
Hot Spots

In the past, it has been a standard practice for public health officials to direct their resources towards studying the basis for foci of infection, which have in Covid-speak become “hot spots.” There are areas of the world where the death rate for Covid-19 is much higher than immediately adjacent areas where infection rates appear to be similar. I find it odd that this is no longer a priority with Covid-19.

What kinds of explanations are being offered in the vacuum of official silence?

Northern Italy

One hypothesis for the conspicuously higher death rate in northern Italy is the vaccination for influenza in the prior year. This particular region of Italy had a unique influenza vaccine distributed that was made from canine kidneys instead of the normal porcine kidneys.\(^\text{221}\) As a result of this difference, there was canine coronavirus contamination in that vaccine. The hypothesis is that immunization to those coronaviruses caused the increased risk to Covid-19 less than a year later. This hypothesis is consistent with previous coronavirus vaccines prepared for SARS and MERS, many of which increased risks from re-infection from the same and different coronaviruses.

Another hypothesis is that high air pollution was the influencing factor.\(^\text{222}\)

Recently, a second air-pollution hypothesis was offered that related increased mortality to a synergistic effect between air pollution and obesity.\(^\text{223}\)

Of major antioxidant molecules in the human body, such as glutathione, ascorbic acid and uric acid, has been neglected in this context. Here, we performed an in vitro metabolomics study of the 3 most odorous anaerobic microbes isolated from the human gut in our laboratory (Clostridium sporogenes, Clostridium subterminale and Romboutsia lituseburensis) when grown in anaerobiosis or in aerobiosis with these 3 antioxidant molecules via gas and liquid chromatography-mass spectrometry (GC/MS and LC/MS). There was no growth or volatile organic compound production in aerobic cultures without the 3 antioxidant molecules. In anaerobiosis, the major metabolic products of the bacteria were thiols, alcohols and short-chain fatty acid esters. The production of alkanes, cycloheptatriene and, paradoxically, increased butyrate production, was observed in the cultures grown in aerobicism with the 3 antioxidant molecules. The qualitative shift suggests specific molecular mechanisms that remain to be elucidated. The increased production of butyrate, but also isobutyrate and isovalerate in vitro suggests that these 3 antioxidant molecules contributed to the maintenance and active resilience of host-bacterial mutualism against mucosal oxygen and uncontrolled oxidative stress in vivo.

\(^{221}\) (+) insert link to SVHI talk.

\(^{222}\) E Conticini, B Frediani and D Caro. Can atmospheric pollution be considered a co-factor in extremely high levels of SARS-CoV-2 lethality in Northern Italy? Environmental Pollution 261: 114465, June 2020.

\(^{223}\) C Lubrano, R Risi, D Masi et al. Is obesity the missing link between Covid-19 severity and air pollution? Environmental Pollution 266: 115327, July 2020. DOI: 10.1016/j.envpol.2020.115327 From the abstract, “we hypothesized that obesity may be one of the links between Covid-19 severity and high level of air pollution. First, obesity is a predisposing factor for SARS-CoV-2 infection and worse Covid-19 outcomes, and unequivocal evidence demonstrated that fat mass excess is independently associated with several pulmonary diseases and lung inflammation. Moreover, it has been shown that obesity may intensify the detrimental effects of air pollution on the lungs, and this is not surprising if we consider that these conditions share an excessive activation of the immune system and a lung inflammatory infiltrate. Finally, fat mass excess has also been speculated to be itself a consequence of air pollutants exposure, which has been proved to induce metabolic disruption and weight gain in murine models. In conclusion, although many variables must be taken into account in the analysis of the pandemic, our observations suggest that obesity may act as effect modifier of smog-induced lung-injury, and the concomitant presence of these two factors could better explain the higher virulence, faster spread and greater mortality of SARS-CoV-2 in Northern Italy compared to the rest of the country.”
Wuhan

The air pollution hypothesis was one of the first hypotheses offered for Wuhan’s severe mortality risks. Even though China does not allow ground-level data to be published, visitors to Wuhan experienced the severe pollution directly and have spoken about it publicly.

A recent study\(^\text{224}\) used satellite data of atmospheric observations to draw a correlation between air pollution and Covid morbidity and mortality. Although aspects of that study were uncontrolled and atmospheric sampling was intermittent and averaged, they found a strong relationship. The strongest relationship was with northern Italy, but correlations were also found for China and the USA (New York, in particular).

Cold spots

Just as hot spots have made the news and shaped public-health policy, cold spots have been conspicuously absent and are more of an embarrassment to public-health officials. It seems quite odd to me that those few researchers attempting to shed light on what the differences are generally not funded by public-health agencies, despite this being their official mandate.

While their motives for not doing their jobs may be open to all kinds of speculation, I prefer to merely point out that those people in charge of your public-health wellbeing are simply not doing their jobs.

Japan

Despite no lockdown, Japan has one of the lowest death rates for Covid-19 among the Western countries. The Japanese people have adopted mask-wearing behaviors as part of their cultural deference towards politeness and public courtesy. Although this is often cited as being evidence of the efficacy of wearing masks, the correlation appears to be superficial rather than causal. Something deeper is going on.

The differences between Japan and other Western countries are numerous regarding diet. Two aspects relating to the specific content of this book are (1) consumption of high amounts of seafood, and (2) consumption of high amounts of fermented natto (a rich source of nattokinase and vitamin K\(_2\)). These are just as appropriate for investigation as mask-wearing to explain Japan’s conspicuously low mortality rate regarding Covid-19 in the face of an official no-lockdown policy.

A fascinating study funded by the donation of testing kits was conducted in a Japanese company during the Japanese “second wave,” from May 26\(^{\text{th}}\) to August 25\(^{\text{th}}\).\(^\text{225}\) Two things were fascinating to me. One was the fact that they found SARS-CoV-2 antibody tests turning positive in a completely asymptomatic population. The other was that they used two different testing systems side-by-side. It is a source of personal incredulity that public-health agencies have not endorsed nor undertaken such scientific

\(^{224}\) Riccardo Pansini and Davide Fornacca. Initial evidence of higher morbidity and mortality due to SARS-CoV-2 in regions with lower air quality. MedRxiv (preprint). doi: https://doi.org/10.1101/2020.04.04.20053595. From the abstract, we investigated “the geographical expansion of the infection and correlate it with the annual indexes of air quality observed from the Sentinel-5 satellite orbiting around China, Italy and the U.S.A. Controlling for population size, we find more viral infections in those areas afflicted by carbon monoxide (CO) and nitrogen dioxide (NO\(_2\)). Higher mortality was also correlated with poor air quality, namely with high PM2.5, CO and NO\(_2\) values. In Italy, the correspondence between poor air quality and SARS-CoV-2 appearance and induced mortality was the starkest. Similar to smoking, people living in polluted areas are more vulnerable to SARS-CoV-2 infections and induced mortality.”

comparison of different tests, which is necessary research for determining the validity of data that becomes the basis of public policy. It is intriguing that a few unsupported individuals have engaged in collaboration to remedy this abrogation of official responsibility.

A collaboration between two Japanese medical researchers working in the field of regenerative medicine and two US researchers out of Boston (one with the Pulmonary Medicine division at Boston Children’s Hospital and the other with the General Medicine and Primary-Care division at Beth Israel Deaconess Medical School.
Wisdom in Moderation versus Acute Treatment

I firmly believe there is wisdom in moderation. Generally. There are philosophical aspects of this view, and practical ones. For example, I often give the advice, “When in doubt, trust Mother Nature.” This position is one reason why the paleolithic and ancestral diet concepts have so much appeal—and utility. There is a very slow adaptation of each species to its environment. If we deviate from this environment, for example, with processed foods, fluorescent lighting, staying up late at night, driving cars fueled with leaded gasoline, paying taxes, watching the news on television, we might suffer, and possibly suffer greatly. And likewise, if we return to some aspect of our native environment, we might decrease our suffering.

In modern times, this has proven quite useful. The return to natural foods, for example, is making people healthier in so many ways. It’s a sad thing that mainstream medical advice is no longer in alignment with this foundation for health. Most doctors promote the official US food pyramid despite its seriously flawed science and the plethora of contradicting evidence that supports its perpetuation of the diseases that are plaguing Americans and ever-rising as this dietary advice is increasingly adopted.

But there is a hidden side to “natural.” Mother Nature never optimizes anything but survival to reproduce. In other words, Mother Nature takes into account the cost of each resource when investing in benefits. The cost-benefit ratio of any given investment can change. For example, at some distant point in human evolution, we lost the ability to make vitamin C. Presumably, this happened at a time when vitamin C was plentiful in our diet. But that is no longer the case. A million years ago, a hundred thousand years ago, ten-thousand years ago, and a thousand years ago, obtaining an extra gram of vitamin C was costly. One had to climb a tree, forage in the next valley, import tropical fruit, or who knows what to find that extra vitamin C. Mostly, we simply did without.

I think the wisdom of Mother Nature is merely foundational. It certainly fails to consider the value of us as individuals in relation to each other. We have attachments to each other that might vaguely affect reproduction, survival of the next generation, and the stability of family (and tribe), but these attachments are supremely valuable to us as individuals. We care about each other more than we care about our species. We care more about the wellbeing and survival of our family and friends than we care about total strangers.226

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226 This is why socialism, communism and welfare states tend to fail, but why families and charities tend to work. Even though most people think that the ethical principles of all of them are equivalent, they are far from the same. With families and charities, the donor voluntarily donates with the knowledge of who is receiving the donated aid. With charities, we may not know the final recipient of our donations, but we do know the pastor, priest, mullah, rabbi, director, CEO, friend or neighbor who is in charge of determining the “need” of the unknown recipient. So it’s still personal. But when we are dealing with socialism, communism or a welfare state, the giving is no longer personal—or voluntary. It’s anonymous. And because of this difference, it is ethically analogous to theft rather than charity. If it is perceived to be “unfair” by citizen standards, especially taxpayers, it evokes resentment rather than satisfaction. It is too easy to question the deservedness of the anonymous recipient of tax-based support. People know from story-telling and direct experience that bureaucrats cannot judge fairness if their life depended on it, nor do they have the discretionary authority to make their giving value dependent. So the citizens AND charities suffer from the resort to taxation. Unresolved resentment breaks down social cohesiveness. There is nobody accountable for misjudgments of need. The absence of negative feedback institutionalizes mistakes. Things fall apart and nothing gets fixed until a catastrophe occurs, and even then it is only the appearance of things getting fixed. The FDA shuts down successful Covid-19 practices because the clinical results belie the government propaganda. The cost of vitamin C infusions is increased by 20-fold for political reasons. There is no trustworthiness in any official institution.
Regarding Covid-19 infection, we can see that the risks are not primarily relative to reproduction. So the advice of Mother Nature may be less than helpful in the immediacy of the infectious crisis. Regarding the elderly, it’s OK with Mother Nature if they pass away.

Those of us who are younger and have one or more of the “diseases of civilization” are also not Mother nature’s concern. We have successfully adapted to our environment in the past, and it’s not her problem if we left the Garden of Eden, to use a metaphor, or choose to ignore our genetic and epigenetic heritage.

To the extent that we accept the social strictures of the disease management industry, that’s on us, not Mother Nature. Our issue is seeing the good from the bad. We are trained by societal pressures to accept bad advice from middle-aged men dressed in white lab coats with stethoscope plumage. We are trained by ostracism penalty to follow a particular religion, to listen to social gossip, to accept bullying and racism, to buy the latest and greatest new toys, and to vote for the people who promise us what we want to hear.

Social conformity may be one of Mother Nature’s best survival adaptations for successful reproduction in humans, but she also designed us to avoid unpleasant people, leave lands that no longer have sufficient food, to seek mates outside of our family circle who manifest the complementary “energy type” of our grandparents. Some of these instincts, behaviors and tendencies are no longer critical to survival. Hunting and gathering food is now a trip to the supermarket. High-energy foods (sugar, fat, organ meats) have gone from rare and fortuitous finding in the wild to everyday choices from a shelf or refrigerator. So adaptive mechanisms to dietary scarcities become addictions when such foods become plentiful. Yet other aspects of our instincts, behaviors and tendencies are still quite serviceable, helping us navigate the modern mess we are embedded in.

Are we willing to engage discipline in exchange for decreasing our pre-existing conditions?

Some, certainly. Others, no.

**Medical Advice versus Medical Supervision**

There is standard advice to seek medical advice for medical conditions. The problem here is that nine out of ten doctors will not know anything about some aspect of what is discussed here, and unlikely to have

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227 See “four-energies” work by Julie Motz. http://www.fourenergies.com/four-personalities.html. The four types are genetically hardwired. “Connectors” pair bond with “Performers,” and Performers with Connectors. Their children are “Comprehenders” and “Perceivers” depending on the sex of their parental types. If the father is a Connector and the mother a Performer, all the children are Perceivers, who will choose a Comprehender spouse. But if the mother is a Connector and the father a Performer, the kids are Comprehenders who will seek a Perceiver mate. This genetic scheme minimizes sexual selection risks between adjacent generations by imposing an every-other-generation pattern. Comprehender-Perceiver pairings result in Performer-Connector nextgen pairings, which complete the cycle with Performer-Connector grandkids. So Performer males find mates that match the types of their paternal grandmother and maternal grandfather, and Performer females find mates that match the type of their paternal grandfather and maternal grandmother. It makes sense to me that this is the simplest two-trait genetic (or epigenetic) system that separates generations to promote optimum reproductive survival. One trait is passed through the male line and involves an energy dynamic Julie calls parasympathetic and sympathetic, which is not synonymous with type-A and -B personalities and may not correlate with actual adrenal-gland activities, but may be likely associated with some kind of autonomic polarity. The other trait is passed through the female line, influencing the “boundary” of consciousness or “sense of self” that separates the inner, private being from the public, visible being, whether it is closely held in deep tissues or loosely held in surface tissues. This personality schema also manifests in physical traits, like posture, walking and dancing “style,” and body proportions. The specific genetics of this pattern remain to be identified. But it is consistent with the survival value of Mother Nature by preventing inbreeding and promoting genetic diversity.
read even a tenth of a percent of the literature cited in these pages. So what is she/he going to tell you? One of my physician co-authors in other books, Ward Dean, M.D., summed it up succinctly,

“Doctors tend to be down on things they are not up on.”

Still, there may be wisdom in running your prospective antiviral-defense program past your physician and medical team. The bad part may be in trusting their advice. As you may or may not know, the medical “standard of care” is defined by what other doctors would do in the same situation. So if most doctors are ignorant about something, the standard of care is bad medicine, and good medicine is a regulatory violation.

Even giving advice is intrinsically part of the standard of care. They cannot get around it, you cannot get around it, so your best defense is simply understanding the rock and the hard place they are trapped between.

This situation applies to the United State and medically associated countries like Great Britain, Canada, New Zealand and Australia, all of which follow the US in policy and practice. If you are in a different country, like China or the Democratic Republic of the Congo, the legal, regulatory and market structures may be entirely different. In the USA, for example, there is an atypical degree of freedom regarding access to dietary supplements that is not tolerated in most other countries with USA-style medical regulations. In some places in the world, you may not be able to openly buy vitamin A or selenium.

Who is the most trustworthy person for your health and welfare? There is only one answer, you! Not your doctor. Your doctors do not share your values. Your doctor has to worry about losing their license. You do not have a license to lose. I do not have a medical license to lose. Your doctor has a reputation to worry about. This reputation comes partly from his or her peers, but it also comes from Yelp reviews, any media reports about them, regulatory actions against them, and lastly the opinions of their patients.

I write the facts as I see them under the protections of free speech.

**Ebola Virus**

The recent emergence of Ebola virus from rural African communities into urban settings, and the spread of cases outside of Africa, has made lipid-enveloped viral disease a more immediate threat in the minds of many.

It is important to understand that the acute hemorrhagic viral disease (as opposed to chronic viral disease) requires two therapeutic strategies, not just one. This book presents at great length the decrease of viral virulence and increase in host viral resistance with a combination of BHT, nutrition and/or metabolic therapies. But for Ebola virus, that’s only half of the potential solution; changing viral virulence takes time, and the therapeutic timeframe can be quite short with hemorrhagic fevers. The acute effects of such viruses on selenium status, blood clotting, vitamin C and collagen infrastructure can kill by fluid loss and internal bleeding in less than a day. If you are lucky, it might take a few days. Therefore, it is critical to combine the antiviral therapies of this book with acute therapies like intravenous vitamin C administration and selenium supplementation, to mitigate the collateral effects of hemorrhagic viruses on tissue and vascular integrity, so that people can live long enough to have the Ebola virus attenuate.

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228 E W Taylor and C S Ramanathan (1995) argue that the DNA of Ebola and other hemorrhagic viruses is known to carry abnormally large numbers of UGA codons, which code for the amino acid selenocysteine. Therefore, the faster the virus replicates, the faster selenium is depleted, and the greater the effect on clotting and bleed out.
According to public-health officials, viruses do not attenuate. They assert that virulence is hard-wired into the genes of viruses (and other infectious organisms), despite clear scientific evidence to the contrary, and compelling epidemiological evidence to the contrary. This faulty view can lead to drastic unintentional consequences if public-health officials make bad triage decisions, mismanage resources based on erroneous “scientific” knowledge, and impose quarantines that deny choice of therapy for treatments that work due to a belief that they do not work.

This is exactly what happened to Allan Smith and his family in New Zealand regarding Allan’s case of swine flu. Swine flu is H1N1 influenza, a lipid-enveloped viral disease that is not a hemorrhagic fever. Nevertheless, in Alan’s case, it progressed to white-out pneumonia (where the lungs are not visible on x-ray images because of being filled with fluid), an induced coma, and a full measure of life-support machinery. The hospital dismissed the family’s request to use high-dose vitamin C therapy, insisting that it did not work, could not work and would not be allowed in the hospital. Because the hospital wanted to “pull the plug” and the family refused to cooperate until vitamin C therapy was administered, the hospital administrators reluctantly agreed to do the high-dose vitamin C.

Overnight, his lungs began to clear. At 24 hours, his lungs were again visible on x-ray. On day 3, the ICU team decided not to pull the plug. On day 4, his tube was removed, on day 7, he was safely taken off life support and began to breathe for the first time in months. On the 60 Minutes New Zealand story about his case, Allan said, “They tell me that I’m not one in a million, I’m one in a billion.” From her diary of the events, his wife Sonia said her husband was the “worst case in New Zealand and Australia” to ever be put on ECMO [life-support] machine.”

Despite his rapid and continuing recovery, the hospital administrators stopped his vitamin C. For three days he got no vitamin C, which is the ideal time for “rebound scurvy” to manifest (see page 89). Allan’s progress slowed precipitously. The family noticed. When they found out what had been done, they raised hell with the management consultant who had been brought in and placed on the ICU team by the hospital administrators to order the change in treatment. With grudging reluctance, the new consultant agreed to resume Allan’s vitamin C, but childishly did so at only one gram. They had to move Allan to another hospital and hire a lawyer, which was less than effective. Finally, when Allan’s induced coma was finally ended, the family had to smuggle in LivOn-brand liposomal vitamin C, 6 grams per day, which was quite effective in restoring Allan’s recovery. It did not take long to get him checked out of the hospital.

The New Zealand doctors were merely following medical policy set in the USA. Please do not dismiss the likelihood that official Ebola-quarantine policy in the USA will be to prohibit vitamin C treatments. I’d count on it.

**Coronavirus, Selenium and Vitamin D**

The December 2019 coronavirus outbreak in China is following the same pattern, in the media and on the streets, as the SARS outbreak of 2003. Words like epidemic and pandemic are being used as I write this note (February). However dire the news reports and speculations are, there is little, if anything, new. History is merely repeating itself.

The SARS virus outbreak was actually caused by a coronavirus. It was named, however, by its symptoms (severe acute respiratory syndrome) instead of by its virus type. This name-change spin has people thinking that this latest outbreak is something brand new, and that a dramatic new level of danger is involved.
I very much doubt that that is the case. I believe that the 2020 coronavirus will attenuate, like 2003 SARS virus did, as it emerges from China to enter a more selenium-replete population. Please see the section on corona and Ebola viruses on page ??? for details of the reasons why. But let me end here with the observation that the SARS outbreak of 2003 started in February and was fully attenuated by July, five months later. This current corona viral outbreak emerged in January, and if history repeats itself, it will attenuate before the coming summer.229

(+) Intrinsic Factor and B₁₂

The B₁₂ viral-susceptibility issue is not just a matter of the availability of dietary B₁₂. In normal, young, healthy people, B₁₂ absorption is facilitated by the secretion of intrinsic factor, a protein which is secreted by the stomach to selectively bind to B₁₂ and facilitate its absorption into the body. The secretion of intrinsic factor seems to be easily impaired by illness, stress and advancing age, so many middle-aged and elderly people do not efficiently absorb B₁₂, even when they take it supplementally. Without any intrinsic factor, only a small percentage of ingested B₁₂ is absorbed.

People who do not secrete intrinsic factor may need ten to a hundred times more dietary B₁₂ than those who do. Alternatively, they may opt to receive regular B₁₂ injections from their physicians.

Before we advance to a major discussion of hypothyroidism, I’d like to cover other risk factors first.

Iron: Subclinical and Clinical Hemochromatosis

Iron is anabolic, particularly in its reduced (ferrous) form. It is therefore a potential aggravating factor for viral disease [Drakesmith and Prentice 2008]. Iron toxicity is also relevant to other conditions [Weinberg 2009], including virus infections of the liver (hepatitis C).

Iron is a known risk factor for many bacterial diseases because of iron’s central role in cellular and subcellular energy processes. This is evidenced by two defining phenomena: 1) the body sequesters (stores) iron during activation of the immune system (a mechanism triggered by cytokines), and 2) iron administration during a bacterial infection can be fatal (it overrides the sequestration mechanism). At the very least, iron administration aggravates some bacterial infections and interferes with treatment. [See Sikorska, et al., 2010 for viral-equivalent effect.]

Iron is equally important to human cellular and subcellular metabolism. Because iron availability is almost always a rate-limiting problem for human growth, iron absorption is an efficient, one-way process. In other words, we do not have a good way to get rid of excess iron—except by bleeding. Menstruating women often have difficulties maintaining sufficient iron levels for good health, but for men, the opposite condition tends to apply—too much iron absorption. I believe that this is a contributing factor to male cardiovascular disease deaths; after 30-40 years of one-way iron absorption, iron starts to “leak” out of overloaded storage systems and causes free radical stress, destruction of vitamin C, breakdown of collagen in the vascular system, decreased immunity, increased vascular infection, plaque formation, and, ultimately, clotting or hemorrhaging. I do not understand why modern Western medical doctors do not assess iron status in all middle-aged males with ferritin, transferrin and TIBC calculations. It’s not expensive. Men should be

229 As I’m writing this, the fourth version of the book in September, the epidemic is slightly attenuating clinically, which is offset in the media by reports of “new cases” going up. This is because testing is no longer being rationed and testing has expanded greatly. Old, asymptomatic cases are now being tested for the first time and being called “new cases” by public-health authorities and the media.
assessed every 5-10 years starting at age 45, and every year if above-range iron levels are found, and every month if they are very high and being actively treated. It’s a cheap test. Why stint?

If your iron is elevated, do what I do: donate blood regularly. If they won’t take your blood because you have hepatitis C or traveled to the wrong part of the world, have your doctor draw the blood and discard it. Or feed it to your plants.

If your iron is over-the-top elevated, get chelation therapy to bring it down quickly. Then start donating blood.

The interaction between iron and vitamin C is problematic, so I suggest that people with very high iron levels consider bringing their iron levels down before administering more than 200-500 mg of vitamin C per day. Also, vitamin C triples iron absorption from vegetable foods. So men and post-menopausal women taking vitamin C might be more susceptible to iron overload.

See also the Haley hypothesis of iron involvement in COPD (page 139).

(+ ) add cd147 disruption and oxidative RBC hemolysis as an acute version of hemochromatosis.

Iron and Copper: the Fenton Chemistry Catalysts

Although iron gets much of the attention due to its central role in cellular and especially subcellular processes, both iron and copper are what I call single-electron redox catalysts. Iron cycles between +3 and +2, which is a difference of only one electron. Copper cycles between +2 and +1, which is also a single electron catalyst.

The reason this is important is because the vast majority of electrons in the human body are paired. Think 99.99%. And think “happily” paired, because physicists and chemists (see Appendix A) have noted that molecules are at their least reactive when their electrons are paired. But stability is not the only necessity for a successful living system. A small number of electrons need to be unpaired for such reactions such as enzyme catalysis, energy generation, and immune defense. However necessary, unpaired electrons (i.e., free radicals) are generally quite reactive, which is a good thing when controlled properly, and a bad thing when they get out of control.

The term “reactive oxygen species” refers to the combination of oxidation with electron-unpaired free radicals. These are generally considered pathological in origin and effect. However, some reactive oxygen species are not only manageably mild, but conditionally essential. The molecules of oxygen gas in the air we breathe are atypically stable with two unpaired electrons. And superoxide, an oxygen molecule with an added electron contributes to roughly 15% of our oxygen metabolism. We call these “negative ions” because they occur in natural air when air and water mix, like when the ocean waves break on the beach, or during rain showers, or at a waterfall, or in the shower at home, or when sprinklers water your lawn, garden or children.

So anytime a purposeful redox chemical reaction has to take place, iron or copper are within nanometers of the site of the reaction to “unpair” electrons to lower the activation energy that is needed to make the

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230 There are multiple choices for iron-selective chelating agents, but no consensus for the best. So some research if you need this option. Another option worth considering for subclinical hemochromatosis is aggressive bloodletting. One cup per week (250 cc) is four times more blood than the Red Cross allows for general donors (one pint every 8 weeks) and twice that allowed when medically prescribed (one pint every four weeks). As total-body iron load decreases, total iron-binding capacity (TIBC) rises. This should rapidly control “leaking” iron and decrease viral susceptibility.
Each iron and copper atom can be recycled to be re-used again and again to facilitate the separation of more electrons for additional reactions.

The problem is not that iron and copper cannot be used safely. There are complicated systems to regulate copper and iron that guide the use of iron and copper for well-regulated biological purposes. The problem is when those systems malfunction. When storage proteins like ferritin and mobilization proteins like transferrin lose control of iron, loose iron atoms catalyze the same kinds of chemical reactions as biologically bound iron, but in an uncontrolled manner.

The end result is the widespread generation of hydroxyl radicals that damage essential molecular systems rather than support them.

The one-electron catalyst is a necessary and essential mechanism of both dual-electron systems and single-electron systems. For dual-electron systems, unpairing electrons allows more reactivity for making more strenuous chemical reactions possible. For single-electron systems, the already unpaired electron can be combined with another unpaired electron to form a pair, which deactivates (dismutates) both free radicals into a less reactive, paired-electron form. This latter function is the defining characteristic of the redox-buffering system, to combine two single-electron glutathione radicals to make oxidized glutathione, which is then reduced by a two-electron redox reaction by NADPH, a carrier of hydride (H⁻, or H-minus).

The workhorses of the redox-buffering system are two-electron donors, the molecular fragment carrying the reducing power within the NADH and NADPH molecules.

In the above diagram of oxidative stress, there are two Fenton chemical reactions taking place, one with vitamin C and molecular oxygen, and the other with vitamin C and hydrogen peroxide. On the left is what takes place in living animals, and on the right (on the tan background) is what takes place in vitro (laboratory cell cultures). The reason these are different is because there is 20 times more oxygen in cell cultures than normal tissue, which makes the Fenton chemistry decidedly non-physiological. This means that cell-culture research can come to conclusions that do not apply to real-life situations.
The perpetual cycling of iron from its oxidized state (+3) to its reduced state (+2) and back again happens when there are oxidizing and reducing molecules in the same environment. Vitamin C is a reducing agent and wants to donate an electron to something, and oxygen is an oxidizing agent that wants to absorb an electron. The iron accommodates both wants. Iron’s $+3 \rightarrow +2$ redox reaction accepts an electron from ascorbate, and iron’s $+2 \rightarrow +3$ redox reaction donates an electron to either oxygen or hydrogen peroxide. The combination of those two reactions creates a cycle. This was named after Henry John Horstman Fenton, who in the late 1800s first developed a powerful oxidizing reagent by adding an iron catalyst to hydrogen peroxide.

Normally, hydrogen peroxide and oxygen would be considered two-electron oxidizing agents. Add two electrons to a molecule of oxygen and it becomes hydrogen peroxide. Add two more and it becomes water. However, in cell-culture experimental work, dramatically higher oxygen levels from air exposure might be reacting with ascorbate in its dianion form. While the pKa (the pH balance point between the two forms) is extremely alkaline, tiny traces of the dianion might react with the dramatically higher oxygen levels to become significant.

The specific role that “loose” iron may play in Covid-19, cytokine storm, coagulation, clotting, fibrosis, and inflammation remains to be fully characterized. But the use of iron chelators during Covid-19 has been suggested as a therapeutic strategy.231

**The Biological Layering of Fenton Reactions**

During the course of evolution, iron and copper have been used to different degrees.232 In the earliest stages of evolution, before plants and multicellular organisms evolved, the atmosphere was reducing (very low in oxygen) and iron and manganese concentrations in the oceans were very high. At that time, copper and zinc concentrations in seawater were extremely low.

At the point where plants evolved photosynthesis and started emitting oxygen gas during daylight, everything began to shift. As oxygen entered the oceans, redox chemistry started to shift on a global level. Not only was oxygen a toxic influence to anaerobic single-cell species of the time, iron and manganese began to oxidize and precipitate to the seafloor. At the same time, copper and zinc salts became soluble and started to rise.

The environmental depletion of iron and manganese as nutrients and the emergence of copper and zinc as toxins (by their antagonism of iron and manganese nutrition) represented a powerful evolutionary force to which life needed to develop defenses. Improved iron and manganese absorption helped, as did detoxification (sequestering) of copper and zinc. As this progressed towards our present circumstance of a highly oxidizing atmosphere and high copper and zinc bioavailability, copper and zinc detoxifying systems evolved from simple sequestration to active enzyme functionality.

This ecological crisis of oxidative stress resulted in (1) the death of untold millions of species of single-cell life and (2) the evolution of aerobic life, mitochondria, multi-celled organisms, fish, amphibians, reptiles, mammals, primates and humans. Yet we carry the evidence of the earlier times in our cells. Mitochondrial superoxide dismutase uses manganese as its coenzyme. Cytoplasmic superoxide dismutase uses copper

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and zinc. And most of the coenzymes in the mitochondrial electron transport chain use iron in the redox-transfer coenzymes. Only the final one uses copper, and it is at the final (most oxidized) step in the transfer of electrons to oxygen.

**Dr. Haley’s Hypothesis for Emeramide and COPD**

Sometimes science advances in leaps. All it takes is a bizarre observation to raise questions about something that we thought we understood, that we previously took for granted. In this case, Boyd Haley was beginning to study the clinical effects of emeramide, a new-concept, mercury-chelating agent that he had been developing for roughly a decade. The new concept was (1) to make the chelator fat soluble, so it would be excreted in feces instead of urine, and (2) that the chelation be strong enough that the gut microbiome could not convert chelated mercury to methyl mercury.

The animal studies had gone well, human studies were underway, and emeramide had successfully treated a test group of Ecuadorian gold miners in a short-term study.\(^\text{233}\) Several clinics in Europe were testing emeramide in carefully selected patients with suspected heavy-metal burdens. One of those clinics tried emeramide in two patients with chronic obstructive pulmonary disease (COPD). There was a rapid and unprecedented recovery.

Definitely unexpected, and maybe to the level of bizarre.

What does mercury toxicity have to do with COPD? Although it is known that smoking tobacco delivers heavy metals to the lungs, and air pollution delivered lead to the lungs from the use of leaded gasoline, the mainstream view has been that this was a minor mechanism that became manageable when people stopped smoking or stopped burning leaded gasoline. Only functional medicine physicians and a few small pharmaceutical companies were developing heavy-metal-oriented therapies, and such treatments were not part of full disclosure to COPD patients in most clinical settings. In other words, heavy metals were just considered one more straw heaped upon the camel’s back.

But the emeramide clinical observation prompted Dr. Haley to reconsider a possible mercury mechanism, and he came up with a very plausible theory: oxidative stress decreasing glutathione’s ability to detoxify mercury. Although the role of glutathione in detoxifying mercury is well established medically and scientifically, most physicians have never considered its clinical implications. It is only the reduced glutathione that acts as an antioxidant and also binds to mercury ions.

In many areas of the world, liquid mercury is used to mine gold. Liquid mercury penetrates into gold ores and dissolves the gold out of the rock. The dissolved gold is then recovered by boiling off the mercury. The problem with this approach is that mercury vapor becomes an occupational hazard for the miners and an environmental hazard for everybody. So these Ecuadorian miners were the ideal group of test subjects for emeramide, which was designed to irreversibly bind mercury and to excrete it through the gall bladder into feces instead of through the kidneys into urine. The intestine and colon are relatively insensitive to mercury, whereas the kidney is exquisitely sensitive to mercury.

Mercury binds strongly with sulfur. The primary mercury ore is mercury sulfide (cinnabar). And the body burden of mercury in healthy people is bound to glutathione. In Alzheimer’s disease, the brain’s burden of mercury shifts from the sulfur atoms of glutathione (SH) to the sulfur atoms of enzymes (also SH). Boyd Haley’s team at the University of Kentucky and their colleagues at the University of Calgary were able to demonstrate this special sensitivity of neurons to subtle shifts in the ratio of mercury to glutathione’s SH groups. They then identified that 100% of the enzymes known to be inhibited in Alzheimer’s disease contained a sulfhydryl (SH) group at or near the site of enzyme activity. And none of the enzymes that were not inhibited in AD had sulfhydryl (SH) groups. So this was the smoking gun for Alzheimer’s pathology, and it was discovered

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The idea is that lung tissue is at special risk of oxidative stress because of its high exposure to atmospheric oxygen. Add in mercury in relatively small amounts and suddenly you have a meta-stable situation where lung status pivots on glutathione’s redox couple (the ratio of reduced to oxidized glutathione). When the redox-buffering capacity is high, mercury remains contained. When redox-buffering falters, mercury leaks from its glutathione containment to poison nearby enzymes. With Alzheimer’s disease, it’s beta-tubulin, creatine kinase and a set of phosphatases and kinases that regulate the brain’s phosphorylation cycle. With COPD, it’s likely to be the sulfur centers in lung mitochondria.

So what emeramide may have done is bind loose mercury and restore lung mitochondrial function. In days, the lungs went from an organ failure state to a functioning state. Given the number of people with COPD, this needs further investigation.

I’d like to extend Haley’s hypothesis one step further and hypothesize that (1) mercury release from glutathione may be the proximal cause of oxidative stress-induced death in Covid-19 patients, or (2) mercury binds to sulfur to displace iron in the mitochondrial electron transport chain, and the resulting Fenton chemistry spikes oxidative stress beyond biologically manageable levels.235

Further Discussion of Mercury and Iron on FACT Forum

The FACT Forum started out as a place where doctors interested in chelation therapies and oxidative medicine could talk to each other and share information. Started by Garry Gordon long before I knew about it, it’s one place where I glean nuggets of information from smarter-than-average doctors.

Asked: “There is a theory that makes sense (to me anyway) that Covid-19 displaces O2 and Fe [iron] from the RBC [red blood cell]. The lung damage seen is not just pneumonia- it’s damage from overwhelming oxidative stress from the high circulating iron. Obviously antioxidants such as vitamin C help. My old ACAM notes are packed away and I’m not sure they cover iron anyway. Anyone know the efficacy of DMSA or EDTA with iron? Any rational for continuous low dose chelation for those who are sick with COVID (eg DMSA 25mg po q2h)? Anyone doing Fe testing on hypoxic patients in their clinics?”

My answer: One of the bizarre observations I have run across is the report of unexpected efficacy from emeramide (aka, OSR, NBMI) in COPD, a chelator of mercury and other sulfur-oriented heavy

by dental scientists, not Alzheimer’s researchers. Maybe that’s why nothing has been put into clinical study for 18 years, and counting. So Alzheimer’s disease is caused by a shift of mercury ions from glutathione (in control) to enzymes (out of control). So why not COPD?

235 I am intrigued by the multiple synergies that this mechanism integrates. (1) The century-and-a-half chronic human exposure to mercury via amalgam dental fillings. (2) Add to that the evolving standard of care in the last century to put amalgam fillings in children and pregnant women. (3) Include the ever-increasing consumption of refined carbohydrate foods (sugar and flour), with increased dental carries, gut dysbiosis and rising insulin resistance, the latter of which contributes to chronic inflammation, cancer, the potentiation of gestational diabetes and bigger-brained babies. (4) Then consider the introduction of strains of genetically hybridized wheat into US agriculture which added gluten indigestibility and more chronic gut inflammation to the mix. (5) The switch from symptomatic treatment to blood-test treatment for hypothyroid symptoms that leaves 40% of the population with inadequate redox-buffering capacity. (6) The popularization of microbes as a cause of disease by industries selling new-and-improved antimicrobials caused widespread sterilization obsession, resulting immune naïveté, an acceleration in the decline in the gut microbiome, and the emergence of widespread childhood diseases like asthma, inner-ear infections, eczema, migraines and allergies. (7) Add to that a public-health policy of adding a new childhood vaccine every other year, none of which have every been safety tested by inert placebo. And now the stage has been set for a virus that kills by oxidative stress to run amok. And the tipping point for this is possibly the high background level of mercury that is found in pretty much everybody after ten generations of mother-to-child accumulation.
metals. Boyd Haley hypothesized a mechanism regarding an interaction between mercury and iron to explain it, and this might relate directly to your question.

The idea is that mercury has the capability of disrupting the iron-sulfur clusters in mitochondria. These sulfur-iron clusters are the sites where the redox reactions of the electron transport chain operate, the interference in which would predict decreased ATP synthesis (loss of aerobic capacity). The specific mechanism is mercury displacement of iron. This happens in the lung because it is the most oxygen-exposed tissue, and it is the most alkaline-stressed tissue (from outgassing of CO₂).

Mercury is a conspicuous sulfur-phile, and if one of the sulfur atoms coordinating the iron-sulfur complex is displaced to bind to mercury, the iron-sulfur complex either loses an iron atom or falls apart completely. I do not think Boyd has an informed opinion about that. Nor do I. But the displaced iron atom is capable of reacting with oxygen and vitamin C to generate hydroxyl radicals via the Fenton reaction. This can run amok when there is depletion of ascorbate, selenium deficiency or loss of glutathione recycling.

All three may be happening with Covid-19 infection, as evidenced by the prevalence of cytokine storms. And if not all three, then two: depletion of ascorbate and loss of glutathione recycling.

I hypothesize that it is the latter which triggers the process. Glutathione has two duties (1) redox stabilization, and (2) mercury detoxification. During Covid-19 or Ebola infection, there is acute oxidative stress that draws down ascorbate into the scurvy range and shifts the glutathione redox couple towards oxidized glutathione. Oxidized glutathione does not bind mercury. So it’s not a stretch to argue that oxidation of glutathione not only interferes with mercury binding, but might also release mercury from the GS-Hg+ and GS-Hg-SG complexes. This is also the suggested mechanisms of onset for Alzheimer’s disease, so why not COPD and Covid-19 respiratory distress?

I think many practitioners are realizing that the respiratory distress seen in Covid-19 infection is not typical. For one observation, the suppression of breathing appears to be central rather than peripheral, suggesting that coronavirus has neurotrophic properties. But even if it does, that does not in any way diminish the possibility that loss of redox defense is causing scurvy of the lung, mercury-mediated iron displacement, Fenton-chemistry and mitochondrial dysfunction.236

And let’s not forget fibrosis of the lung from loss of ascorbate-mediated collagen maturation. Immature collagen is an inducer of matrix metalloproteinases (MMPs), which would add an independent inflammatory mechanism to the multiple existing ones.

If this schema is correct, chelating iron may not be necessary. All that is needed is to restore redox potential through some kind of combination of vitamin C, glutathione, selenium mitochondrial fuel or carb-restricted diet. Since glutathione is both recyclable and synthesizable, and vitamin C is only

236 It might turn out that emeramide’s effect on COPD may be partially mediated by the temporary chelation of iron by emeramide. I do not favor that hypothesis due to the observed orders of magnitude lower binding of first-period transition metals (iron, copper, zinc, manganese, chromium, etc.) to emeramide than third-period transition metals (mercury, thallium, lead, bismuth, etc.). I much prefer Boyd’s hypothesis of released mercury, towards which emeramide has a profound binding efficiency. The emeramide-mercury complex is reported to be so stable that tetrasodium EDTA will not pull the mercury out of it.
recyclable, I suggest that vitamin C is the obvious therapeutic front runner for resetting the redox-defense system in the face of imminent collapse.

It is also active within minutes of starting IV therapy. Oral use takes an hour to manifest.

If anybody wants to discuss testing this hypothesis, please feel free to call or email. —Steve

Type-1 and Type-2 Copper Deficiencies

Copper deficiency\textsuperscript{237} may have pro-viral consequences (Yörük, \textit{et al.}, 2007). It comes in two forms:

**Type-I** is the dietary kind, which can be corrected by eating copper-rich foods like shellfish (especially oysters), nuts (especially sesame seeds, cashews and sunflower seeds), grains (buckwheat and wheat), beans (especially garbanzo and navy) and calf’s liver, or by taking copper supplements.

**Type-II** is not caused by diet, but by chronic inflammation. The activation of the immune system causes excessive copper storage (sequestration) in ceruloplasmin and resulting deficiencies in the body tissues. This cannot be treated by foods and supplements, as the administered copper is filtered out of the blood supply from the stomach and intestine as it passes through the liver before going on to the rest of the body. Getting rid of the inflammation by resolving the underlying cause is the therapeutic strategy to consider.

Although the literature documenting the antiviral effects of copper are not as robust as other agents mentioned here, copper has secondary effects on inflammatory mechanisms that indirectly affect viruses and viral damage to tissues. First, copper is an essential cofactor for superoxide dismutase (SOD-1), which plays a central and critical role in cellular antioxidant defenses. And second, copper is an essential maturation factor for collagen, the primary structural protein for the body. Antioxidant failure and immature collagen independently promote chronic inflammation.

Zinc also has type-I and type-II deficiencies (see page 72).

Hypothyroidism

See also the Basal Metabolic Rate chapter on page 99.

Hypothyroidism (low thyroid hormone levels) and hypometabolism (low effect from thyroid hormone) are a lot more common than most people think. The terms “subclinical hypothyroidism” and “thyroid resistance” are often used to describe hypothyroidism that occurs in people with thyroid hormone levels that fall within the “normal” range. However, this “normal” range is set so broadly that there is good reason to question its validity. First, the range is set statistically, with the lowest 2.5% of readings being defined as hypothyroidism and the top 2.5% being defined as hyperthyroidism. This completely ignores the clinical presentation of symptoms. And second, hyperthyroidism symptoms are comparatively rare, but hypothyroid symptoms are rampant. So the inclusion of so many hypothyroid people in the normal range skews it clinically. Estimates are that 40% of the population exhibits moderate to pronounced hypothyroid symptoms, while at most 1% exhibit hyperthyroid symptoms. People with significant hypothyroid symptoms should not be included in setting the normal range. It’s positively bizarre that such a scientifically non-sensical approach has become the standard of care in the USA.

\textsuperscript{237} Copper deficiency has been described as “copper toxicity” by several clinicians in popular writings. High ceruloplasmin is not necessarily copper toxicity, unless the copper is “leaking” from the ceruloplasmin. In fact, high ceruloplasmin more often means chronic copper sequestration and low copper bioavailability. Please keep this in mind.
To make this situation worse, many doctors do not actually test thyroid hormones (T₃ and T₄), but rather test thyroid stimulating hormone (TSH), which has a dubious connection to actual hormone levels and an even more tenuous connection to thyroid hormone effect on body tissues. Even some alternative/complimentary doctors believe that TSH suppression is a stable marker for the increased effect of thyroid hormone supplementation. It is not. The data suggesting that TSH is a good test is very weak, being measured only in young, healthy males. All of this folly makes thyroid diagnoses one of the most botched clinical assessments in modern medicine.

Even if you are lucky to get T₄ testing, and very lucky to get T₃, and exceedingly lucky to get reverse T₃ (rT₃) tested, thyroid hormone acts at the cellular and subcellular levels (nuclear and mitochondrial). All of these standard blood tests ignore what happens at those “deeper” levels of the body.

I prefer the term “hypometabolism” to describe this problem because it bypasses the issue of how much hormone is or is not being secreted by the thyroid gland and stresses the net effect of thyroid hormone at the cellular and subcellular levels. If the metabolism-enhancing effect of thyroid hormone is deficient, then you have all the symptoms of hypothyroidism, whatever it may be called. And whatever your blood tests are.

The official medical dogma in the US is that “subclinical” hypothyroidism does not exist, yet one in four people in the US experience a broad spectrum of overt hypothyroid symptoms, and one in two experience a few of the symptoms. The most objective symptoms are caloric: a depressed body temperature, a tendency to get chilled easily, a difficulty warming up after getting chilled, and cold hands and feet much of the time. But the subjective symptoms of fatigue, weakness, poor stamina, depression, sleep difficulties and cognitive problems are just as real a manifestation of insufficient cellular energy production as a lack of warmth.

In talking with the many hundreds of people who experience chronic herpes problems, I now suspect that hypothyroidism/hypometabolism symptoms are substantially more common among herpes sufferers than the general population.

The underlying biological dysfunction of hypothyroidism and hypometabolism is a depressed basal metabolic rate. In other words, this means that people with this condition have a less-than-ideal cellular energy production. Basal metabolic rate is regulated by thyroid hormones (most notably T₄ and T₃). So a simple deficiency of either T₄ or T₃, or both, can produce hypometabolic symptoms. Medical tests of these hormones are useful for precisely this reason. However, the normal range is set far too wide and people with low levels are routinely told by their physicians that their thyroid is “normal” and that “nothing is wrong,” despite the fact that their levels are low and the high likelihood that thyroid medication would readily relieve their oppressive symptoms.

If your physician has told you this, look at the test results for yourself. Ask for a photocopy of the thyroid test results, or ask for a copy of all your medical records and keep a copy at home.

Find the T₃ and T₄ readings and see if they are in the middle of the normal range. If they are near the bottom of the range, you might want to reconsider the prospect of thyroid hormone replacement therapy and nutritional supplementation of thyroid-related nutrients.

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238 I’ve produced a one-page summary of hypothyroidism/hypometabolism for the Project Wellbeing web site that not only has a thorough summary of common symptoms, but reviews all of the medical thyroid tests with what they tell you and what they do not tell you. This is available as a free PDF download from http://www.projectwellbeing.com/steve, the same site that makes this book available as a free download.
Although orthodox physicians are trained to look only at thyroid hormones in the bloodstream, this is only a superficial view of the full process by which thyroid hormones regulate basal metabolic rate. There are other steps involved, any one of which can malfunction to produce insufficient metabolic rate.

First, each of the tissues of the body has the ability to convert the low potency thyroid hormone (T₄) into the high potency thyroid hormone (T₃). T₃ is four times more potent than T₄. Since the thyroid hormone excreted by the thyroid gland is 85% T₄ and only 15% is T₃, this tissue-level control of T₄-to-T₃ conversion is potentially a huge determinant of basal metabolic rate. If doctors do not test both T₄ and T₃, they do not have a clue about thyroid hormone conversion.

Second, the enzyme that converts T₄ into T₃ is selenium dependent. So selenium nutriture is involved in more than one way.

Third, T₄ can get converted into reverse-T₃ (rT₃), which has no thyroid hormone activity at all, and which undercuts basal metabolic rate. When doctors do not measure both T₃ and rT₃, they have no idea of the relative contribution of activation and deactivation of thyroid hormone. Not one in a hundred patients presenting with hypothyroid symptoms has their rT₃ measured by mainstream doctors before being told to “just live with it.”

Fourth, there are receptors on the surface of cells that bind to thyroid hormones and transfer it within the cell. These receptors are essential for thyroid to be able to affect metabolic rate. Changes in the function of those receptors can undermine metabolic responsiveness to thyroid hormone. There is no medical test for this. Only a thyroid “hormone challenge” can determine whether thyroid resistance is taking place.

Fifth, there are also thyroid hormone receptors on the surfaces of the nucleus and mitochondria, which are also essential to thyroid’s hormone function. Standard thyroid blood tests done by your doctor ignore these receptors.

Sixth, there are thyroid immune antibodies to consider. These thyroid antibodies can interfere with thyroid activity. US physicians are trained to look only at blood TSH, and sometimes T₄, and rarely T₃ levels in people without severe symptoms. It is no wonder that there are lots of hypometabolic people walking about who believe that their thyroids are “normal.” Their doctors said so.

There is an acknowledged medical condition called generalized resistance to thyroid hormone (GRTH), in which serious hypothyroid symptoms coexist with normal blood thyroid hormone levels. If GRTH is possible, why is it impossible that a subclinical version of this condition might exist? It isn’t.

Many orthodox doctors are trained to ignore blatant hypothyroid symptoms when blood thyroid tests fall into the “normal” range—which is set by statistical criteria, not medical ones. The scientifically valid test for basal metabolism is not blood thyroid levels, but whole-body calorimetry. But whole-body calorimeters are rare, expensive, troublesome to operate (they need constant calibration), and they make patients feel claustrophobic in a coffin-like enclosure. Fortunately, simple body temperature measurements provide a reasonable indication of basal metabolic rate. Thermometers are inexpensive and easy to operate.

The best time to measure body temperature is in the early morning (4-6 AM), just after waking and before getting out of bed. This minimizes the contribution of muscle activity, stress and digestion to body temperature, So basal temperature is more obvious. So take your temperature before stretching, going to the bathroom, or engaging in any other kind of physical activity.

Because body temperature is affected by a variety of things, it is a good idea to take repeated measurements on successive days to determine how much your morning body temperature fluctuates.
Some people advocate the use of axial (armpit) body temperature. This may be a good idea. However, most people do not have major variations between axial and mouth temperatures. The most common exceptions are “mouth breathers,” who breathe through their mouths instead of their noses while they sleep. If you don’t know, why not test both and compare. The cost of a second thermometer is not going to break you.

With the exception of ear thermometers, pretty much any kind of thermometer can be used. Battery-powered electronic thermometers are the fastest, but they may produce varying (unstable) results depending on the electronics package, how long you leave them in, and how fresh the battery is. Some electronic thermometers have accuracy errors of one degree, even though they say that they meet “medical standards” on the label. Mercury thermometers and the new gallium thermometers are quite stable, but you have to shake them down after each use, and it takes many minutes for them to reach their stable readings. Gallium also solidifies at normal room temperatures and has to be warmed up to melt before use. “Fertility” thermometers are often excellent due to their easier-to-read more finely graduated temperature markings. Any thermometer may have calibration errors (i.e., produce readings that are off by a set amount). If you have more than one thermometer, you can play them off against each other for speed of use, temperature agreement, and consistency. Once you have a favorite, stick with it for your daily temperature readings.

Normal body temperature in the early morning is usually a half-degree to almost a full degree lower than the “normal” daytime temperature of 98.6°F (37°C). But if it is significantly more than a degree low, then it may be appropriate to investigate thyroid and metabolic issues, especially in those people who have specific health complaints that may be related to hypometabolism. Read up on the symptoms. See if they describe you.

**Adrenal Hormone Issues**

Hypothyroid symptoms can be caused by adrenal exhaustion, too. Thyroid hormone works intimately with adrenal hormone (cortisol), and a cortisol deficiency produces a nearly identical set of symptoms as hypothyroidism and hypometabolism. Some hormone-replacement specialists will not work on thyroid hormone replacement without simultaneous low-dose adrenal hormone replacement.

Cortisol has a strong circadian (daily) pattern. Levels are very high in the early AM, drop rapidly during the morning hours, and then less rapidly during the afternoon, to bottom out in the evening hours. Therefore, it is necessary to measure cortisol at a particular time to have any hope of assessing its sufficiency, and I strongly recommend that you consider four-times-in-a-day salivary assessment to get the shape of the cortisol curve. I’ve seen too many jet-lag-like disturbed cortisol patterns to trust one-time salivary testing or one-time blood testing.

The opposite side of adrenal exhaustion is elevated cortisol. This is the classic stress maladaptation syndrome associated with the “type-A” personality in a high-stress job, post-traumatic stress disorder (PTSD), or a type-B personality trapped in a really bad situation. Since cortisol is catabolic-aerobic-acidic to an extreme, this can cause down-regulation of other catabolic-aerobic-acidic systems in an attempt to restore homeostasis. The four-time salivary adrenal stress test identifies this problem as well as adrenal exhaustion.

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239 It is a USA prejudice to report temperatures in Fahrenheit. For readers in other countries, you can approximate the Fahrenheit relative descriptions by halving them. When I write that body temperature is half-degree lower at night, that’s roughly a quarter degree Centigrade lower at night.
Estrogen Dominance and Sex-Hormone Replacement Therapies

Estrogen dominance is a risk factor for hypometabolism, autoimmune diseases, cancer and viral diseases. This is one of the reasons why:

1) younger women are at much higher risk for autoimmune diseases than young men,
2) older men develop autoimmune diseases (their testosterone converts to estrogen with aging),
3) in women, herpes flare-ups synchronize with the estrogen-dominant phase of menstruation, and
4) cancer is strongly age related.

Estrogens promote these processes by suppressing protein synthesis and energy metabolism. In other words, estrogens are anabolic/anaerobic/alkaline. There are three primary estrogens, estrone (E1), estradiol (E2) and estriol (E3). Of the three, estriol is the least potent estrogen and is actually protective for autoimmune diseases.

The following Steroid Tree illustration shows how steroids are metabolized. On the right side, the lowest branch is the mineralocorticoids and the branch right above it is the cortisol (corticosteroid) branch.
dominates, with androstenedione, testosterone and DHEA playing minor roles. In both sexes, estrogens are the off-switches.

Because of the on-off antagonism or balance between estrogens and other steroids, estrogen must be measured in its hormonal context. In men with high testosterone, high estrogen is not as big a risk factor as it is in low-testosterone men. Likewise, estrogen dominance in women is defined by the estrogen/progesterone ratio.

Because of the on-off antagonism or balance between estrogens and other steroids, estrogen must be measured in its hormonal context. In men with high testosterone, high estrogen is not as big a risk factor as it is in low-testosterone men. Likewise, estrogen dominance in women is defined by the estrogen/progesterone ratio.

During perimenopause, progesterone levels fall before estrogen levels do. This creates a stronger estrogen dominance than generally exists in post-menopausal women who have low estrogen but even lower progesterone. This estrogen dominance is one reason why menopause is so metabolically stressful. According to changes in melatonin levels, women age twice as fast during the menopausal transition as they do before and after (see blue line in the illustration at right).

The estrogen context is also important because it is modified by inflammation. Inflammation is triggered by infection, allergy and oxidative stresses, which cause immune system cells to send out cell-signaling factors called cytokines, which turn on inflammation and activate the estrogen-forming enzyme. This enzyme, aromatase, converts testosterone to estradiol, androstenedione to estrone and hydroxyandrostenedione to hydroxyestrone. So it is possible that estrogen dominance can be triggered by inflammation and perpetuated by chronic inflammation.

In men, testosterone declines gradually over time while estrogen slowly rises. However, sudden changes in estrogen levels can be caused by infection, allergy or iron toxicity. This is best measured by simultaneous measurement of testosterone and estradiol, where the ratio provides information about the activity of aromatase. Men whose testosterone is low due to inflammation often have severe side effects from testosterone replacement therapy due to the aromatase-induced skyrocketing of estradiol levels. This can be easily detected by measuring estradiol along with testosterone testing. Since this is not standard practice, men need to insist that their estradiol be tested.

**Infection**

It might seem weird to talk about infection and chronic infection in a book about viral disease. After all, isn’t herpes an infectious disease? And isn’t viral disease, in general, infectious? Yes, certainly. However, other infections can co-exist with viral disease and cause metabolic shifts that are favorable to viral
replication. Sometimes, inflammation cannot be turned off merely by shutting down viral replication. If there is another infectious disease present, it may persist long after viral load goes to zero.

**Allergy**

Allergy is an independent inflammatory influence. Allergies can be generally split into two kinds: immediate hypersensitivities and delayed hypersensitivities. The immediate hypersensitivities are the kinds of reactions that you notice: rashes, itching, boils, sores, swelling, redness, coughing, sneezing, watery eyes. These are obvious symptoms, and they tend to occur rapidly after exposure (minutes to hours). They are mediated by IgE antibodies, and dermatologists specialize in such allergies.

The delayed hypersensitivities are a different kettle of fish, involving IgA, IgG and IgM antibodies. Many allergists vociferously denied that they even existed less than 20 years ago. Reactions can be spread out over time, and delayed by up to 6-8 days. These kinds of sensitivities are often referred to as food allergies and they can be related to gut permeability (“leaky gut syndrome”). Wheat, milk, yeast, corn and eggs are probably the most common foods causing delayed hypersensitivities, but this may be because such foods are so common. With leaky-gut syndrome, any food eaten regularly can become an allergic influence.

Both immediate and delayed hypersensitivities trigger immune responses and cytokine activation of aromatase (the estrogen-driving enzyme) and indoleamine dioxygenase (IDO, a tryptophan-destroying enzyme). Aromatase converts energy-enhancing hormones (progesterone, testosterone) into energy-conserving hormones (estradiol and estrone). This exacerbates hypometabolism and increases viral susceptibility. IDO catabolizes (degrades) tryptophan, 5-hydroxytryptophan and a host of other indoleamines that might be found in certain herbs. The most predictable consequence of this is serotonin deficiency, which would otherwise be synthesized from the destroyed tryptophan. This can lead to depression, sleep problems, moodiness, emotional volatility, aggravation of obsessive and compulsive tendencies, and irritability. It can also result in sugar and carbohydrate cravings, impulse-regulation problems, violence-control problems and alcoholism.

**Heavy Metals**

Low metabolism can also be triggered by heavy metals, which sabotage enzyme systems in the body and impair mitochondrial metabolism. I have seen heavy metal toxicity show up in unexpected situations, so some kind of screening is probably the only way to know. In my opinion, a chelation challenge is the best option. With this test, a chelating agent is administered by mouth or by injection, and the heavy metals are measured in a 6-hour, 8-hour or 24-hour urine-collection sample. Without the chelation challenge, the majority of people with heavy metal burdens will show no heavy metal or only traces of heavy metal in their hair or urine tests. In other words, the false-negative risk is high for non-challenge testing.

EDTA is the most common chelating agent, and it works well for most toxic metals/minerals. DMSA or DMPS are generally favored for measuring mercury levels. Nano-colloidal zeolite seems to facilitate the safe transport of mercury to the urine. Regular zeolite does not work in this manner.

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240 Some experts recommend several days to a week of taking the chelation protocol before the “after” test is done. This may do a better job of identifying “deeper” pools of heavy metals than testing the next day.
The Special Case of Mercury

Mercury is a problematic heavy metal to have in your deep tissues. Mercury +2 ions are pro-viral in their metabolic effect. And that effect is manifesting in the deep tissues where viruses operate.

The problem with mercury is also worse because our main mercury-detoxification system is highly dysfunctional. In the distant past, when mercury burden was very low, it might have been sufficient. But in today’s environment, it is overwhelmed and actually increases mercury toxicity to the brain.

Our bodies detoxify mercury by binding it with glutathione. This is amazingly effective because the mercury-sulfur bond is quite strong and therefore stable. But when the liver binds mercury with glutathione, it secretes it through the gall bladder into bile, which has to travel all the way down the GI tract, through the bowel, to be fecally excreted. With very low levels of mercury, this may have worked well in our distant ancestors. But today, during this gut transit, there is high enough mercury to cause toxicity to the bacteria in the colon. Glutathione-bound mercury may be relatively low in toxicity to us, but it is quite toxic to bacteria. For bacteria, methyl mercury is less toxic. So they convert the glutathione mercury into methyl mercury, which is efficiently absorbed back into our bloodstream. Methyl mercury is far more toxic to us than ionic mercury. It has a special affinity for fat and preferentially partitions into the brain, causing more toxicity than was originally present.

There is a potential solution to this: emeramide. Unlike other chelation substances, emeramide is fat soluble and forms the strongest known (and possibly irreversible) complex with mercury. It is believed that this prevents bacterial conversion to methylmercury.

Emeramide is relatively new, only a decade old. So there is a lot that is not yet known about the clinical subtleties of its best use. But it is a new wrinkle in the problem of mercury that holds much promise. Emeramide is not yet approved as a pharmaceutical, but it is in clinical use in specialty medical practices in Europe. It is also in clinical trials in other countries. It is commercially available as a research chemical. Mercury-toxic people are purchasing it on this basis and using it personally. For better access to this group of people, there are Internet groups devoted to emeramide.

See also the Haley hypothesis discussion on page 139. This may be the actual mechanism by which oxidative stress causes death.

Urine pH Biofeedback

Another method that can be used to track metabolic state is sequential urine pH testing. This biofeedback technique involves 1) testing urine pH every time you urinate, 2) plotting the test results on paper, 3) connecting the dots to see the pH-change curve, and 4) finding correlations between urine pH changes and symptoms. If a correlation is found, then the metabolism (and urine pH) can be manipulated to influence symptoms and, hopefully, undermine a disease process (i.e., viral susceptibility).

There are specific pH features that may be closely associated with viral susceptibility. The first one is alkaline dominance. In this situation, the urine pH is either frequently alkaline, most particularly during the

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241 The emeramide-mercury complex is so strong that EDTA cannot dislodge it.
242 Emeramide is also known as NBMI, OSR, BDTH2 and Irminix.
day when it is supposed to be acid, or the pH baseline (the pH average) is significantly higher (more alkaline) than 6.0-6.2 (the approximate average pH in healthy people). These features may be caused by a multiplicity of factors. Some are metabolic, like hypothyroidism or hypometabolism. Some are dietary (like a vegetarian diet, a specific nutrient deficiency, or an unsuitable diet). Some are ecological, like from pesticide poisoning, heavy metal toxicity (dental amalgams), gut dysbiosis or allergies. Whatever the cause, the urine pH is a reflection of a biological dysfunction just as viral susceptibility is.

If you are fortunate to see an alkaline dominant pattern, then you can sequentially investigate what factors do or do not change that dominance. This is biofeedback training, based on urine pH as the biofeedback signal. When you find something that moves your alkaline dominance towards normal, that something is likely to reduce your viral susceptibility in the bargain. It may also reduce other symptoms that you might not think are associated with viral susceptibility, like migraine headaches, asthma, fatigue, depression, sensitivity to cold weather, mental fuzziness, and sleepiness in the mid or late afternoon.

Unfortunately, alkaline dominance may be masked by inflammation. When alkaline dominance reaches a point where it becomes biologically dangerous, the body produces tissue hormones (prostaglandins) which mitigate the alkaline stress. If you remember Revici’s model, alkaline stress is a manifestation of anabolic dominance, and prostaglandins are the body’s catabolic defense mechanism to an anabolic crisis. These prostaglandins (particularly prostaglandin E2) cause the urine to swing strongly acid, which “masks” (hides) the underlying alkaline stress. This is the second pH pattern to look for: constant acidity. If the urine stays acid all the time, especially during the night when it is supposed to swing alkaline, then your alkaline stress has progressed to a state of chronic inflammation. While urine pH is masked by chronic acidity, urine pH testing cannot be used for biofeedback purposes—until the inflammation is resolved (ended).

Therapeutically, chronic inflammation is troublesome to treat. Most orthodox doctors don’t even attempt to treat it. Even doctors familiar with nutritional and ecological medicine have difficulty dealing with chronic inflammation. This is because there are so many potential causes.

Chronic infection can trigger chronic inflammation. Chronic infections may result from parasites, protozoans, viruses, fungi or bacteria. Sometimes such infections are easy to overlook, or take for granted, like toenail fungus.

Chronic allergies can trigger chronic inflammation. These may take the form of classic respiratory allergies (pollen, grasses, animal dander) and skin allergies (rashes from fibers or chemical exposures) that orthodox allergists recognize and treat with corticosteroids (cortisone) and other drugs. Classic allergies are triggered by immunoglobulins of the E series. These are quick. The exposure and reaction are separated by seconds or minutes, and rarely hours.

Alternatively, inflammation may be triggered by hypersensitivity reactions to foods (food allergies), chemicals (chemical sensitivities), and traditional allergens (molds, dust mites, animal dander, etc.). Delayed hypersensitivities are triggered by immunoglobulins of the A, M and G series (IgA, IgM, IgG), which most orthodox allergists ignore. Delayed hypersensitivity reactions are difficult to recognize because some symptoms can follow the exposure by up to 6-8 days!

The food allergy issue can be specific to a narrow group of foods that tend to be frequently consumed. Wheat and yeast allergies are common examples because wheat protein (gluten) and the cell walls of yeast are very difficult to digest. So in a marginal digestive system, undigested wheat proteins or yeast cell-wall polysaccharides may become a trigger for hypersensitivity and chronic inflammation.
In some people, allergies to foods can be extensive, involving dozens to hundreds of foods. Usually, this is a direct result of 1) dysbiosis (a disturbance in the intestinal flora that would otherwise assist human digestion), and/or 2) intestinal hyperpermeability (a breakdown in the integrity of the intestinal lumen, the internal “skin” of the digestive tract). There are now high-tech medical tests for dysbiosis and gut permeability. Although most doctors still deny that intestinal permeability is a disease, it is now becoming easier to find physicians that will test for this syndrome.

The intestinal lumen relies on the amino acid glutamine for much of its energy requirements. Oral glutamine is often prescribed to speed up intestinal healing, along with probiotics (e.g., acidophilus). The bowel lining relies upon butyrate (butyric acid), a short-chain fatty acid released by microbial digestion of fiber in the colon. When fiber is deficient, or when fiber-digesting microbes have been killed off by antibiotic use, colonocytes become hypometabolic and risk of colon cancer increases.\textsuperscript{243}

The third pH pattern to look for is the “jet lag” pattern. In this situation, the normal pattern of acid (catabolic) dominance during the day and alkaline (anabolic) dominance during the night is reversed.

Jet-lag syndromes not only result from high speed east/west long-distance travel, but from dietary, metabolic and neuroendocrine disturbances. Sequential urine pH testing is one way to identify any kind of circadian (daily) metabolic rhythm dyssynchrony (timing disturbance).

\section*{Wearable Devices}

One of the advantages of being a tech geek is seeing technology trends up close and personal. For example, we are now at the end of the computer age and are transitioning into the age of the sensor.

The age of the computer was heralded by the invention of the solid-state transistor, and its incorporation into microprocessors (large-scale integrated circuit chips). This age of miniaturization allowed progressively more and more transistors to be placed on a chip. The computational power of car-sized mainframe computers have transitioned into hand-held computers we now call cell phones. Half of the citizenry of the world hold processing power in the palm of their hands that was un-dreamed of when I was born. The 5 megabyte hard-disk drive of the 1950s was loaded onto an Air Force airplane by a forklift. When I designed my first database, the same sized memory drive was the size of a large paperback book and cost $5000. Now, you cannot even buy a flash drive with that low a memory capacity. Instead of megabytes, its gigabytes and terabytes.

Moore’s Law accurately predicted that chip densities would double every 18 months, and that held for many decades. But it is finally failing. Microprocessor circuitry is now so small that the actual atoms in a conductive or semiconductive trace can be counted by a two-year-old grade-school child.

Miniaturization can go no further. Random compositional defects and quantum effects become fatal flaws. Super-capacitors require insulators with 99.9999% purities, and those 0.0001% impurities cause electron leakage that will drain a supercapacitor in a day. So we are down to doubling capacities in a decade instead of 18 months.

So what we now face is the age of nanotechnology, which can be thought of as the technology of molecular precision. Incredibly cheap sensors are one of the first breakthroughs of this nanotech revolution.

\textsuperscript{243} Colonocytes can be fed by ketone fuels, as an alternative to butyrate. The primary ketone fuel, beta-hydroxybutyrate, has a structure very similar to butyrate. More importantly, butyrate is metabolized into beta-hydroxybutyrate when it feeds the colonocytes. Ketone fuels also feed the rest of the body, even the brain, which raises metabolic rate.
In the consumer market, we now are seeing ever-increasing numbers of wearable devices with inbuilt sensors. These sensors track pulse, blood sugar, heart-rate variability, breathing rate, body temperature, factors relating to sleep quality, and the list goes on and on. The pulse oximeter I just bought to track blood-oxygen saturation stores continuous data for 12 hours and costs $99. I think it’s likely that in a decade, it’ll cost $19.95.

In the very near future (right now?), these sensor devices and wearables will serve as early warning devices for monitoring our real-time health on a continuous 24-7-365 basis. Trial programs are already underway. The fever, low oxygen levels, changes in breathing and coughing will be noticeable before they become full-blown symptoms. More importantly, the next generation of sensors will track things that are directly related to the development of pre-existing conditions that put people at risk of negative Covid outcomes.

I will not matter that the medical profession tolerates blood-sugar pathology if you, personally, do not tolerate it. Hypothyroidism and hypometabolism will be easily identified. Even the distressing events in our lives that sabotage heart-rate variability and lead to autonomic dysregulation can be detected passively.

In the last two decades, wearables have grown from a fringe movement populated by innovators to a niche market that is crossing over into early adopters.

In certain parts of the world, insurance customers get discounts when they agree to monitor certain risks that the insurance actuaries can predict future costs. Manage your risks and you get a discount; be careless and you pay double, triple or quadruple the rates. I predict that this will expand immensely once insurance carriers realize how much more money they can make by sharing some of their increased profit from cooperative clients with their cooperative clients.

With Covid, a good case can be made that early intervention greatly affects outcome risks. This case has been made for many decades with other viral diseases, but it is being drawn into focus because of the perception of dramatically increased risks. I think the case has been made that oxidative stress and redox-buffering are hugely influential in balancing the immune response, providing both increased competence and decreased malfunction at the same time. And cases are being made that early use of oxygen, corticosteroids, hydroxychloroquine/zinc, and vitamins D, A, B₁ and carb restriction diets can significantly improve survival odds.

Wearable devices and at-home testing provides a very real opportunity for the earliest detection.

**Getting from Here to There**

During this period of adjustment, where institutions are hopelessly out of touch with reality and posing genuine dangers to our health, prosperity, freedoms, privacy and mental wellbeing, awareness of off-the-shelf products will provide solutions without the need to affect political process.
Appendix A: The Biophysics of Life

This section starts the virus discussion at its foundation.

All living systems are based on biochemistry. I think it would be hard to find a medical scientist or practicing physician who would disagree with that assertion. Yet the bigger picture also includes physics, where the intrinsic properties of elements and electron orbitals determine how biochemistry actually works on a causal, functional level.

For example, quantum physics is the basis for quantum chemistry, which is a functional basis for photosynthesis in plants, nitrogen fixation in bacteria, and mitochondrial energy production in animals.

If that’s not important enough, consider that physics is also the basis of elemental chemistry, which includes both pH (acidity and alkalinity) and redox potential (oxidation and reduction). These latter two foundational chemical phenomena are treated as trivial by most medical researchers and only rudimentarily assessed by clinicians who are supposed to be monitoring your disease risks and giving you sound health information.

I’m sorry to say, most clinicians are not monitoring your disease risks or giving you sound health information. In fact, it’s almost 180 degrees backwards. Cholesterol causes heart disease? Wrong. Eating complex carbohydrates is good for diabetics? Wrong. Vitamins are only enriching the sewer? Wrong. Vaccinations are completely safe? Wrong. Fasting blood sugars of 99 mg/dL are OK? Not if you catch coronavirus. Earin three square meals day after day is a healthy way to eat? No. Periodic partial or intermittent fasting is needed to “exercise” your energy system to have a good redox potential to fight virulent viral infections.

Misinformation is the rule, not the exception.

Those pre-existing conditions that everybody is talking about to explain coronavirus deaths are just the tip of the iceberg. The obvious ones like age, obesity and diabetes are easy to see and clearly associated with increased mortality. That’s the part of the iceberg above the waterline. But what nobody wants to talk about are the invisible, under-the-waterline pre-existing conditions that are not easy to see, unless you test for them, or get diagnosed by a medical doctor.

On your annual CBC blood test results, blood sugars between 90 and 100 mg/dl are a hidden risk factor, not because the data is not on the page, but because your doctor does not tell you that you have insulin resistance and are pre-pre-diabetic, both of which are risk factors for coronavirus mortality. It’s not part of the official “standard of care” to measure your selenium, zinc, electrolytes, vitamin C, vitamin D, thyroid hormones or metabolic rate. Yet every one of them is a risk factor for coronaviral infection.

Body temperature and pulse rate are measured—once. But they consider a low body temperature to be inconsequential and low pulse rate to be healthy, when both are risk factors for coronavirus morbidity. And the best temperature measurement to take is first thing in the AM when you have not eaten, not exercised.
and are not stressed, all of which are not the case when you are in a doctor’s office getting your pulse and body temperature measured.

The bottom line is that the things that matter for surviving coronavirus with grace are mostly ignored by medical professionals. And those medical professionals who do know about them are treated badly by those that don’t.

So let’s go back to the basics.

Let’s consider the realities of biology, chemistry and physics that, when ignored, lead to such high mortality from virulent viruses. And let’s let our personal self-interest, our concern for our families and friends, and our regard for the welfare of each other to uncover the propaganda that has led to us being compromised instead of prepared.

The coronavirus is merely exposing hidden aspects of disease that are routinely or deliberately ignored.

**The Law of Gravity**

Living systems must comply with all of the “laws” of the universe. I would assume that a basic understanding of those laws is necessary and essential aspect for any scientific understanding of biology. But as it turns out, most of medical practice is not based on pH and redox science. They are treated as self-correcting systems that do not need either measurement or diagnostic consideration, except in rare and atypical conditions. But mild pH and redox pathologies are neither rare nor atypical.

Dr. Emanuel Revici was the first medical scientist that I know who addressed this deficiency. Not only did he study these as basic phenomena, but he built a system of measurement and diagnosis upon it. It is somewhat ironic that such a heresy was not the actual basis for the US government’s persecution of Revici. It was his applying such insights to the treatment of cancer.

Many who have followed the history of cancer treatments know, the US has a firm policy that surgery, radiation and chemotherapy are the only legitimate cancer treatments, despite any and all evidence to the contrary.

Revici made one more unforgiveable mistake, he developed a one-injection treatment for heroin addiction that eliminated all cravings for three days. While clearly a blessing for ameliorating much human suffering, for addicts and their families alike, it ran afoul of the US government’s new “crony” policy of treating heroin addiction with methadone. Millions upon millions of pharmaceutical-industry dollars were on the line.

Talk about unforgiveable!

The government responded with character assassination, regulatory malfeasance and outright fraud. Well-meaning scientists were recruited towards this end, and at the last minute bypassed for the “consensus” conclusion that Revici was a fraud. Sadly, none of those well-meaning scientists who had been manipulated spoke up, or asked that their names be removed from a conclusion where they had seen none of the evidence gathered or presented. It was a “hatchet job,” both political and preconceived. For Revici, himself, a well-meaning scientist and physician, this was devastating. From reading his work, and having talked with him at length, I am convinced that he was the brightest medical mind of the last century. He died in public obscurity in 1997 at the age of 101.

It may seem grandiose to go from such simple basics as pH and redox to such advanced (and speculative) emergent phenomena as nutrition, metabolism, cell signaling, development, immune function, pathology,
epigenetics, neuroendocrine regulation, wellbeing and consciousness. But just as a house’s many functionalities depend on the quality of its foundation, our health, wellbeing and “spirituality” depend on the underlying chemical and biological systems from which they emerge. I have seen too much evidence that abnormalities of pH, redox potential and “metabolic balance” have profound adverse effects on wellbeing to doubt that ignoring it is at our risk.

Just as ignoring history causes history to repeat itself, ignoring the fundamentals of biology, physics and chemistry is a prescription for chronic disease. Science and medicine have a long history of suffering the arrogance of rational-dominant, ego-driven scientists. This has given rise to the popular saying that “science advances one funeral at a time.”

Those that believed that washing one’s hands after touching a cadaver and delivering a baby was folly had to die before Semmelweis’s sterilization hypothesis would be taken seriously. Thousands upon thousands of women and babies had to die horribly painful deaths before the institutional resistance to innovation waned sufficiently for hygiene reforms to be implemented.

And those who suppressed Revici’s science will have to die before his research will be replicated, and expanded in it detail and sophistication. Make no mistake, much will be learned when modern technologies and analytical methods are brought to bear on what Revici was able to measure with tools from a century ago.

What does all this mean to you? It means that there are aspects of health and wellness that you can understand and embrace which are being ignored by the authorities that tell you, with utter assurance and blatant error, what is healthiest to eat, drink, supplement and medicate.

As the Internet has expanded in scope and access, many flawed truths of the establishment have become appreciated by more than a few. Most nutritional sophisticates know that the US government’s “food pyramid” is 99.44% folly and that the pyramid is, in its most basic essence, upside down. The American Diabetes Association (ADA) recommendation of high complex carbohydrate diets for diabetics (and prediabetes) actually worsens their condition. The recommendations of the American Heart Association (AHA) to avoid saturated fat and cholesterol to avoid heart disease is just plain wrong. And this is not to
single out the USA for abuse. But it is a sad observation that the USA leads the world in nutritional, dietary, pharmaceutical and political folly.

Before I go on, let me mention a last example in recent times, the “prudent diet” advocated by Ansel Keys, and ultimately, by the entire US Government, the vast majority of NGOs, and hundreds of large, processed-food companies. His/their thesis was that animal fats were “bad” and that vegetable fats were the remedy for the bad that animal fats caused. This was even supported by hundreds of US researchers, who were paid by tax dollars (and US-vegetable-oil-industry money) to support this idea. Medical schools trained new doctors in this belief. This bias was exhibited in the early, “preliminary” reports from the Framingham studies that the vegetable-oil diet did appear to be lowering cardiovascular mortality risks. But the rose-colored-glasses data did not hold up. There were contrary findings, which were minimized by being rejected by the most prestigious medical journals, like the New England Journal of Medicine and the Journal of the American Medical Association. When the final findings of the Framingham Study concluded that the diet was worthless at best—and likely hazardous at worst—the finding were again rejected by NEJM and JAMA.

In the final analysis of a half-century of data, it turns out that there was no significant cardiovascular benefit from the “prudent” diet. But it was established that the diet did significantly increase cancer risk. This was not noticed (or noted) in earlier analyses because the cancer risk shows up most clearly in older ages. In the very oldest group of participants, cancer was increased by a factor of eight.

Modern folly of unprecedented magnitude.

And you paid for it with your tax dollars.

So my message is that it is time for you to bypass the bureaucracies and NGOs and go straight to the research. In this day of the Internet, this is entirely up to you. Much of what you read here will be something you may never have heard from the US Food and Drug Administration (FDA), the National Institutes of Health (NIH), the Centers for Disease Control (CDC), the American Heart Association (AHA), the American Medical Association (AMA) and the Office of the President of the United States (POTUS). And it is my hope that you see this as a good thing, with an open mind balanced by skepticism, and with a personal investment in protecting you and yours from these viral risks that these bureaucracies have “aided and abetted.”

__It (Alfred A Knopf, 2010), which does an excellent job of reviewing the way carbs contribute to insulin resistance, diabetes and obesity.__

247 To anthropomorphize bureaucracies and institutions can be both misleading and counterproductive. Even though the FDA, CDC and NIH are corrupt and callous institutions, the vast majority of people in the FDA, NIH and CDC are well meaning people who do care. But those employees who do care are embedded in a hierarchical bureaucracy with 5% Machiavellian schemers and 5% yes-men (and women), where those that care are marginalized, frustrated, intimidated and rendered unable to accomplish what they know is right. Therefore, anything I say about the FDA being arbitrary, capricious or evil tends to be rejected by anybody at the FDA or anybody trusting in the FDA because the pejorative attributions do not apply to the personally, as individuals. And during most workdays, FDA employees see the positive intentions of their co-workers; they do not see the evil face to face, even though they may see illogical, arbitrary and capricious decisions being made around them that do not make sense to their sensibilities. What to do about this unfortunate state of affairs? Maybe, let it be? Having personally compared political-oriented action to grass-roots activism over decades, the latter wins hands down. I no longer waste my time on the former. I’m suggesting that you’ll be happier investing in diet, lifestyle, supplements and medications than you will trying to change entrenched institutions through political means. Despite any moral outrage you may experience in hearing and reading about the FDA’s long history of malfeasance, the frustrations of political mass action are just as real outside of the FDA as they are inside the FDA.
**pH**

Acidity and alkalinity is pretty easy for most people to understand. We have every-day experiences of acid and alkali, some of which we experience in childhood when our brains are still plastic (flexible) and we do not need to try to learn. Lemons and vinegar are acidic. Milk of magnesia and baking soda are alkaline. If you put baking soda into lemon juice, it fizzes.

Scientists measure acidity and alkalinity by a pH scale. In water-based systems, the strongest acids approach pH zero and the strongest alkalis (i.e., “bases”) are close to pH 14, the highest number you can get. These are the kinds of pHs that you would see if you bought drain-cleaner products. But the kinds of things that we eat and drink are much, much closer to 7, the halfway point on the scale.

For the vast majority of living systems, the pH stays pretty much in the middle, near seven. Blood pH is near 7.4. Typical urine pH ranges from 5 to 7. Cellular pH is commonly 7.0 to 7.4 but can be slightly below 7 in some tissues. In some cellular organelles with specialized chemical processes, pH can be significantly more acidic (pH 4.5) or alkaline (pH 8).248

The pH scale is like the Richter earthquake scale, each increasing number means a shift of 10-fold in acidity or alkalinity. At pH 7, the acid and alkalinity are balanced, but at pH 6, there is ten time more acid than alkali, and at pH 8 there is ten times more alkalinity than acidity. So at pH 3.5-4.0 (the approximate pH of orange juice), the acidity is more than 1000 times stronger than the alkalinity. And at pH 2-3 (the pH of lemon juice), the acidity is high enough (10,000x) to pucker your lips and distress your tongue.

But the pH of the food or beverage when you eat or drink it is not the issue for your metabolism. Different foods and beverages have an acid or alkaline “ash.” This is the term that the Macrobiotics school uses to describe the ultimate pH effects of eating, digesting, metabolizing and assimilating foods and beverages. It is the pH effect on your biology, not the pH effect on your tongue.

Revici called it a “catabolic” and “anabolic” effect, which was measurable in the acidity and alkalinity produced in body fluids. Even though the roots of Macrobiotics are in traditional Chinese medicine (TCM) and Revici studied these pH phenomena from a Western perspective, their lists of acid-and-alkaline foods are 85%+ overlapping.

As it turns out, the acidic lemon juice is highly alkalinizing. And green leafy vegetables, which do not taste alkaline, are quite alkalinizing—with sea greens (seaweed) being the most alkalinizing of all. On the opposite side, vinegar, with the same acidic taste as lemon juice, is acidifying. And so are polyunsaturated vegetable oils and fish oils, which do not have an acid taste at all.

This means that you have to learn the pH effects of foods to be able to navigate this field. It is not conveniently intuitive.

Table 1 (next page) lists Revici’s chart of foods, nutrients, chemicals and drugs:

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248 Tissue pH’s are substantially disturbed in cancers, being frequently 1-2 pH units off the norms for healthy tissues. It was one of Revici’s insights that cancer came in two forms, one of which had tissue pH higher in the cancer than adjacent non-cancerous tissue, and the other of which had a deviation towards lower pHs.
### Table 1: Metabolic character of foods, nutrients, drugs and chemicals

<table>
<thead>
<tr>
<th>Categories of Revici’s metabolic influences</th>
<th>catabolic-aerobic-acidifying</th>
<th>anabolic-anaerobic-alkalinizing</th>
</tr>
</thead>
<tbody>
<tr>
<td>vitamins</td>
<td></td>
<td></td>
</tr>
<tr>
<td>vitamins A and D</td>
<td>vitamins E and K</td>
<td></td>
</tr>
<tr>
<td>vitamins B₆ and B₁₂</td>
<td>vitamins B₁, B₂, B₃ and B₅</td>
<td>choline, inositol, folic acid</td>
</tr>
<tr>
<td>amino acids</td>
<td></td>
<td></td>
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<tr>
<td>glutamate and aspartate</td>
<td>lysine and ornithine</td>
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</tr>
<tr>
<td>methionine and cysteine</td>
<td>tryptophan and histidine</td>
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</tr>
<tr>
<td>carnitine and acetyl-L-carnitine</td>
<td>arginine</td>
<td></td>
</tr>
<tr>
<td>elements and minerals</td>
<td></td>
<td></td>
</tr>
<tr>
<td>magnesium, calcium, strontium</td>
<td>sodium, potassium and lithium</td>
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</tr>
<tr>
<td>oxygen, ozone, hydrogen peroxide</td>
<td>chloride, bromide, iodide, fluoride</td>
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</tr>
<tr>
<td>superoxide, hyperbaric oxygen</td>
<td>chromium and iron (reduced)</td>
<td></td>
</tr>
<tr>
<td>selenium and sulfur (reduced)</td>
<td>zinc</td>
<td></td>
</tr>
<tr>
<td>manganese, vanadium and copper</td>
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<td>heavy metals</td>
<td>lead and tin</td>
<td>cadmium, thallium and mercury</td>
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<td></td>
<td></td>
<td>arsenic, antimony and bismuth</td>
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<td>lipids</td>
<td>fatty acids</td>
<td>fatty alcohols</td>
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<td></td>
<td>polyunsaturated fatty acids</td>
<td>cholesterol and other sterols</td>
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<td>testosterone and progesterone</td>
<td>estrogen and cortisol</td>
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<td>chemicals</td>
<td>phosphoric acid</td>
<td>glycerol, ethanol, sugars</td>
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<tr>
<td></td>
<td>vinegar</td>
<td>sodium bicarbonate (baking soda)</td>
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<tr>
<td></td>
<td>magnesium thiosulfate</td>
<td>salt (sodium chloride and sea salt)</td>
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<td></td>
<td>BHT</td>
<td>alcohol (distilled spirits)</td>
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<td>drugs</td>
<td>antibiotics</td>
<td>pain killers, aspirin (NSAIDs)</td>
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<td></td>
<td>sulfonamides</td>
<td>narcotics (opiates)</td>
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<tr>
<td></td>
<td>chloroform</td>
<td>benzodiazepines and antidepressants</td>
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<tr>
<td>foods, spices and herbs</td>
<td>meat, nuts and hard cheeses (aged)</td>
<td>dairy (and soft cheeses, kefirs, yogurts)</td>
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<tr>
<td></td>
<td>preserved meats</td>
<td>green leafy veggies</td>
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<td></td>
<td>whole grains and seeds</td>
<td>refined grains, wine and beer</td>
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<td></td>
<td>fried and hard-boiled eggs (hard yolks)</td>
<td>soft-boiled and raw eggs (liquid yolks)</td>
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<tr>
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<td>mayonnaise, butter and oils</td>
<td>soy sauce and salt</td>
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<td>hypericin from St. John’s wort</td>
<td>chocolate, coffee</td>
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<td>ginger and tomatoes</td>
<td>rutin, pollen, alfalfa, kelp, most herbs (and tobacco)</td>
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<tr>
<td></td>
<td>cranberries, cherries, pomegranates</td>
<td>vegetables and fruits (almost all)</td>
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</tbody>
</table>
Before I go any further, I need to clarify that Revici’s turn-of-the-last-century use of the words anabolic and catabolic have different meanings than their present-day scientific or technical definitions. To break this potential misunderstanding, I will refer to them as anabolic-anaerobic-alkalinizing and catabolic-aerobic-acidifying in character, with anabolic and catabolic as shorthand.

Now that some familiar things are categorized, let me go back to the physics and chemistry foundations of biology. There are two things on the previous chart that will illustrate why this phenomenon happens the way it does. First is the classification of functional groups (the “pieces” of molecules), where oxidized functional groups (carboxylic acids, aldehydes and ketones) have a catabolic-aerobic-acidifying character, and reduced (anti-oxidized) functional groups (amines and alcohols) have an anabolic-anaerobic-alkalinizing character. So sugars, which are mostly hydroxy groups, are alkalinizing. And on the other side, vinegar and fatty acids are acidifying because they contain a carboxylic acid group.

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So what happens when you combine them together in the same molecule?

Fats (triglycerides) are close to neutral because the carboxylic acids on the three fatty acids are bonded to glycerol, which has three hydroxy groups. Three on one side and three on the other side. This gives a kind of balance that allows fat to be stored in high concentrations without causing a local metabolic stress.

This same balancing act can be seen in amino acids. Amino groups are anabolic and carboxylic acid groups are catabolic, so amino acids, one of each, are fundamentally balanced. So when biology strings lots of amino acids together to form proteins, peptides and enzymes, they are balanced. But note that some of the amino acids have an extra carboxylic acid group (glutamic acid, aspartic acid) and others have an extra amine group (lysine, ornithine). So these become slanted towards catabolic and anabolic character, because of a two-to-one ratio between the catabolic and anabolic.

Similar kinds of balancing effects may be dietary in nature. For example, the anabolic-anaerobic-alkalinizing effects of a green salad are balanced by oil-and-vinegar salad dressing.

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249 This means that the protein basis of living systems is set in an intrinsically balanced state, with 50% anabolic and 50% catabolic balance of amine and acid functional groups. The oxidized carboxylic acid group of the amino acid is electron deficient and can react with electron-rich amine groups, and the reduced amine group of the amino acid is electron rich and can react with electron-poor carboxylic acid groups. Such a reaction forms a peptide bond, and this is the necessary and essential chemistry of living systems to form peptides, proteins and enzymes.
Next, let’s look at the elements and minerals section.

**Revici’s Periodic Table**

The epicylindrical periodic table (on the previous page) and the flat illustration below highlight the “anabolic” and “catabolic” nature of the elements in blue and red colors. For application to viruses, note the antiviral red colors, and most significantly, the brightest red colors for calcium, oxygen, sulfur and selenium. These four elements were determined by Revici’s testing systems to be the most potent. And each of these keys into natural viral self-defense.

This is why much of this book is devoted to those elements. Oxygen ties to aerobic therapies are mentioned starting on page ??? and the chart on page 158. Sulfur ties to glutathione, cysteine, N-acetylcysteine and thiosulfate (see page 158). Selenium ties to glutathione peroxidase (the primary glutathione recycling enzyme), thioredoxin reductase and viral resistance. And calcium ties to vitamin D3 (see the metabolic Balancing chapter on page ???, and pages ??? and 165) and possibly vitamin K2.

Revici’s periodic table illustrates that there is a physicochemical law of nature behind the anabolic-catabolic continuum.²⁵⁰ Even though Revici only tested 32 of the more-than-a-hundred elements, he was

²⁵⁰ This every-other-column, odd-even pattern reverses at the boundaries marked by deeper electronic orbitals. When going from the p-shell to the d-shell, the odd elements switch from being anabolic-anaerobic-alkalinizing in character to catabolic-aerobic-acidifying. These “switched” elements are the transition metals. And when going from the d-shell into the even-deeper f-shell, they reverse again (the rare-earth metals). I’d appreciate hearing from any chemists or physicists who have any speculations to offer as to why this phenomenon happens this way.
able to see a clear and unmistakable pattern of alternating anabolic-catabolic effects. The alternating columns of blue and red make this quite conspicuous.\(^\text{250}\)

Despite the fact that this elemental pattern underlies biology, it remains unintegrated into mainstream biology and medicine. Considering the widespread roles that these anabolic-catabolic properties play in influencing basic pH effects on a variety of metabolic compartments, this is a serious oversight. Furthermore, Revici’s characterization of pH disturbances in a variety of diseases suggests that these effects are not trivial or inconsequential aspects of pathology, but rather central to any systematic understanding of diseases, and of course, their treatment. Thus, the political and anti-competitive motivations behind the persecution of Revici has been an immense disservice to humanity.

**Redox Potential and the Redox-Buffering System**

Unlike pH, in which life stays pretty much in the middle, redox potential requires maintenance of an extreme state. The redox potential of living organisms is highly reduced compared to the oxygen-containing atmosphere.

In the schematic illustration at right, reduced redox potentials are at the bottom represented by cool and cold colors (green and blue, respectively) and oxidative redox potentials are at the top in increasingly hot colors (oranges, reds and magenta).

Green is where we live.

Notice how close this green zone is to the bottom.

The passive antioxidant defense system (the role that polyphenolic antioxidants play) operates in the warm and hot zones, intercepting reactive oxygen species that are generated (1) organically (by immune cells and metabolism, for examples), (2) pathologically (by hemochromatosis or sunburn, for examples), or (3) naturally by the environment (radioactive potassium, medical x-rays or cosmic rays, for examples).

The passive antioxidant defense system acts like an umbrella, protecting us from the “heat” or ultraviolet of direct sun exposure without actually cooling us off from the ambient air temperature. Antioxidants act like a bucket brigade, passing free radicals from antioxidant to antioxidant until they are de-energized enough to (1) be combined to pair up their unpaired electrons and (2) get reduced by the active part of the antioxidant defense system (vitamin E, vitamin C, glutathione, etc).

The active antioxidant defense system (the redox-buffering system) acts like an iced beverage on a hot day, cooling us off from the ambient temperature. While the passive antioxidant defense system keeps us from being “pulled up” into the hot zone, the active redox-buffering system pulls us down into the green zone.
The green zone is where redox defense takes place. This is characterized by active enzymes powered by ATP and pools of reducing equivalents, including vitamin C and glutathione.

Just below the green zone is the blue zone, the “coldest” part of the redox defense system. This is the “powered” part of the redox-buffering system where active NADH (by analogy, the ice in your drink) is generated, and the pool of NADPH is maintaining the “coldness” of the entire drink.

NADH and NADPH are used by enzymes to cool off (recycle, regenerate) oxidized (burnt, or fried) glutathione and vitamin C.

It’s a beautiful and fairly efficient system.

If maintained properly, it keeps us alive for a century.

But what happens when one or both falter? We get oxidized. By analogy, we heat up. If it’s a minor thing, we move from the bottom of the green zone to the top of the green zone, where we can stay alive but experience chronic oxidative stress that shortens our lives and gives us chronic diseases. Most pre-existing conditions put us there. If the oxidation is temporary, like from a viral infection, the temporary oxidation from the fever and immune defense is eventually overcome and we are restored to the bottom of the green zone. But if the oxidative stress is severe, we move up into the yellow zone where our lives are at grave risk. If this goes on for any significant length of time, we enter the bottom levels of the orange zone and die.

To be healthy, we need the levels of reduced antioxidants to greatly exceed their oxidized counterparts. Think about these as redox “couples,” where reduced glutathione (GSH) is temporarily oxidized into oxidized glutathione (GSSG), only to be rapidly recycled back to reduced glutathione. The redox couple is how we can measure our redox-buffering system. What is the ratio of the cool and cold partner in the redox couple to the warm and hot partner?

If we are healthy, it is higher than when we are sick.

It is lower when we are young and higher when we get older.

Vitamin C goes through the same process, and if we are robust, we recycle vitamin C as fast as gets oxidized. The degree of oxidative stress relative to the robustness of the redox-defense system can be described and measured by the GHS:GSSG ratio and the ascorbate:dehydroascorbate ratio.

But what if we are not robust? What if we are old? What if we have insulin resistance or diabetes? What if we have vascular inflammation and our vitamin C is being wasted trying to “mature” our vascular collagen over and over again? We call that cardiovascular disease.

What if we have inflammation and are sequestering copper with resulting ease of bruising and joint pain? What if we have a deficiency of zinc and our gut integrity is compromised? What if we have heavy metal toxicity from dental fillings or the burning of leaded gasoline for half of the last century? What if we have hypothyroidism or estrogen dominance? What if we eat regular meals with high carbs and/or high protein?

These pre-existing conditions compromise the ability of our redox-buffering system to recycle glutathione and vitamin C.

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251 Alcibey Alvarado and Isabel Arce. Antioxidants in respiratory disease: Basic science research and therapeutic alternatives. Clinical Research and Trials 3(1): 2-11, 2016. doi: 10.15761/CRT.1000163. A review article. From the abstract, “In many diseases, the balance between oxidants and antioxidants (redox balance) is altered causing severe consequences.”
As it turns out, some of the most dangerous effects of viruses on humans is their ability to cause oxidative stress and undermine the redox-buffering system.\textsuperscript{252} Maybe you have heard about “cytokine storms?” This is one way that oxidative stress can manifest during infections. And both high-dose C and selenium\textsuperscript{253} can restore redox-defense mechanisms shut down cytokine storms. There are many other redox-active compounds that can do the same thing.

The redox-buffering system is so important as a foundational aspect of cellular health that there may be many cellular homeostatic mechanisms piggybacked on redox-potential changes. One such identified mechanism is glutathionylation, where glutathione can bind to the reduced sulfur groups of proteins instead of to itself as it becomes oxidized. Normally, in oxidatively unstressed cells, reduced glutathione is at high concentration, 50-100 times greater than oxidized glutathione. In this state, the glutathione redox couple (the ratio of GSH/GSSG) is high. But as oxidative stress increases, the glutathione redox-couple drops, with more and more of the SH groups (GSH) forming SS links (GSSG).

It now appears that one way the cell “reacts” to this change in redox potential is by the formation of SS links between glutathione and proteins (and enzymes). So oxidative stress causes both GSSG and PSSG to build up. PSSG stands for protein-SSG, where the first S is on the protein surface and the second S is on glutathione’s surface. Because this redox-based research is relatively new, it is not yet clear as to the extent this kind of chemistry plays in the complexities of cellular defense and metabolic homeostasis. But it is clear that it has the potential to regulate enzymatic activity analogously to phosphorylation, a broadly critical aspect of cellular regulation, long-distance communication and structural stability.

While the phosphorylation mechanism adds or removes negatively charged phosphate groups from surface-exposed hydroxy (alcohol) groups (on serine and tyrosine amino acid residues), the glutathionylation mechanism adds oxidized glutathione to exposed sulfhydryl (thiol) groups, or removes reduced glutathione from exposed sulfhydryl groups. I hope to add more context to this as I learn more.

The basics for vitamin C (see page 78 and 90) and selenium (see pages 53, 57 and 67) have been discussed above in some detail, and vitamin C in great detail below in Appendix C (page 175).

**How Does This Tie into Coronavirus Infection?**

Some of the early responders to the 2002 SARS coronavirus outbreak noted that the initial presentation of symptoms for severe acute respiratory syndrome were not lung pathologies—or at least classic pneumonia-like symptoms. The initial presentation was hypoxia. Patients were “blue in the face” (very low oxygen saturation of hemoglobin in their blood) without respiratory distress. And without fever. The respiratory distress and fever were week-two symptoms, after their lungs filled up with fluid. Similar signs are present with Covid-19.

\textsuperscript{252} This is why the scientific foundation for the extensive discussion of vitamin C, which is an necessary and essential part of the antioxidant defense system and tissue-level redox control. It is the human inability to synthesize vitamin C that makes humans especially sensitive to catastrophic consequences from viral diseases. And this can be remedied by supplementing vitamin C. See page 17 for the therapeutic uses of vitamin C in viral diseases.

\textsuperscript{253} C K Tseng, C T Ho, H S Hsu, C H Lin, C I Li, T C Li, C S Liu, C C Lin and W Y Lin. Selenium is inversely associated with interleukin-6 in the elderly. J Nutr Health Aging 17(3): 280-4, Mar 2013. “Decreased serum selenium concentration with aging had been found in previous report. In this study, we aim to investigate the association between serum selenium and the inflammatory cytokine interleukin-6 in the elderly living in long-term care facilities in Taiwan.” And the conclusion: “Serum selenium was inversely associated with inflammatory cytokine interleukin-6 among elderly living in long-term care facilities in Taiwan. Monitoring serum selenium should be considered in these institutionalized elderly.”
So what did we learn, and what are we learning from this. First, placing people on ventilators does not work. Somewhere above 80% of people placed on ventilation died, with similar outcomes in 2020. With an understanding of hypoxia as the underlying pathology, this makes perfect sense. The lack of oxygen impairs energy-production symptoms, which results in a redox crisis, during which time the lungs lose the ability to handle the toxicity of oxygen. The re-introduction of oxygen to hypoxic tissues is well known to produce a pathology called hypoxic reperfusion injury, which is exactly what forcing oxygen into hypoxic lungs causes.

In Revici’s model of metabolic balance, catabolic-aerobic-acidifying influences are balanced by anabolic-anaerobic-alkalinizing influences to produce health. Although this is not static—aerobic processes are higher during the day and anaerobic processes are dominant at night—pH balance is still maintained 24/7 by the ability of the kidney to buffer the net imbalance of acid and alkaline influences during the day and night, respectively. But what happens in hypoxia? The catabolic-aerobic-acidifying influences are seriously compromised. The night-time anabolic-anaerobic-alkalinizing phase continues through the daytime, with its associated effects. Edema (tissue water retention). Alkaline stress at the blood level.

**Edema.** Fluid retention in the tissues. Fluid retention in the lungs that remains unbalanced, which leads to fluid accumulation in the lungs.

In normal healthy people, the circadian (daily) cycling of acid-to-alkaline and back again acts like a water pump, driving water into and out of cells, and out of and into tissues, respectively. This daily oscillation of cells from maximally hydrated to dehydrated is part of the cell detoxification process. During the day, water flows one way, for maximizing energy, work, immunity and mental attentiveness. At night, water flows the other way. This “pumps” accumulated waste products out of the cells for detoxification and promotes “healing” at night why we are sleeping.

With sustained hypoxia, this circadian oscillation falters, with unfortunate immune consequences. The augmentation of immune function that normally occurs with the daytime swing to catabolic-aerobic-acidifying dominance fails to take place. The accumulation of fluid in the lung tissues leaks out into the air spaces. The lungs fill with fluid without immune support. Bacteria, fungi and air pollution nanoparticles remain unchecked. The hypoxia now manifests as a classic pneumonia-like condition, but if treated like one, the underlying hypoxia remains entrenched and death is the typical outcome.

**Alkaline stress at the blood level.** The reason that the kidney has to be so efficient in dumping acid or alkaline stress that comes out of the tissues is that hemoglobin is an active protein that operates in a very narrow pH range (usually less than a twentieth of a pH unit!). Other tissues can withstand pH variations ten times higher, but arterial blood has to remain between pH 7.36 and pH 7.44 so that hemoglobin can “hinge” properly.

Hemoglobin has two structural states that depend on subtle pH differences. In the more acidic environment of deep tissues, the hemoglobin molecule hinges one way, and in the more alkaline environment of the

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254 Hypoxic reperfusion injury can also be called (1) reperfusion injury, (2) re-oxygenation injury, and (3) ischemia reperfusion injury. It is caused by the toxicity of oxygen under conditions when the redox-buffering system is compromised.

255 This mechanism also underlies brain health. The pumping of fluids into neurons and glia, and then out into (1) cerebral-spinal fluid, (2) lymphatic fluid and (3) sinus fluid via the cribriform plate is a critical mechanism of brain detoxification. This plays a role in the onset of Alzheimer’s disease. Many brain scans show excessive fluid and shrunken brains in patients with neurodegenerative diseases, which is the brain stuck in edema mode and NOT the result of wholesale loss of brain neurons. For information about preventing and reversing Alzheimer’s disease, watch my nine-part YouTube video series at: https://www.youtube.com/watch?v=j1FmK4582mA&list=PL620DC3CA557284EB.
lungs, hemoglobin hinges the other way. This pH-hinging action of hemoglobin alters the binding of oxygen and carbon dioxide.\textsuperscript{256} In the deep tissue state, hemoglobin has less affinity for oxygen and greater affinity for CO\textsubscript{2}. In the lungs, hemoglobin has decreased affinity for CO\textsubscript{2} and enhanced affinity for oxygen. So hemoglobin acts as a “pump” for oxygen and CO\textsubscript{2}, pumping oxygen from the lungs to the deep tissues, and pumping CO\textsubscript{2} from the deep tissues to the lungs. And this pumping depends on an optimal pH gradient between the subtle acidity of the deep tissues compared to the subtle alkalinity of the lungs.

So why do people with coronavirus-induced hypoxia not show any respiratory distress in this early phase? The answer is that the breathing reflex is triggered by the presence of CO\textsubscript{2} and not the absence of oxygen. We breathe to get rid of CO\textsubscript{2} not to obtain oxygen.

So as long as CO\textsubscript{2} is within ideal ranges, we do not experience the oxygen deficit. One possible pathology that might cause this is gradual blood pH stress altering the ability of hemoglobin to release oxygen. Another is viral infection of the respiratory center of the brain. A third might be viral infection of the kidney and loss of pH buffering capacity.

**Advanced Metabolic Balancing (towards genuine sustainability)**

The simplest model of metabolic balancing of anabolic and catabolic influences on viral disease is the grouping of influences into proviral and antiviral categories. This is, in essence, the table on page 158. Two categories is the simplest organizational schema. The proviral agents promote viral virulence and decrease viral host resistance, and the antiviral influences decrease virulence and increase host resistance.

But there is danger in simple models. Nature is rarely simple, and actions based on a simplistic understanding are rarely without collateral consequences. Take allopathic medicine and the traditional practice of prescribing pharmaceutical drugs to suppress symptoms. Although the side effects are considered “better than the disease,” the further prescription of another drug to control the side effects of a previous drug leads to a new round of collateral symptoms and further prescriptions. In institutions where this practice goes unchecked, like in intermediate-care facilities, polypharmacy-derived dementias are not an uncommon outcome.

For the nutritionally savvy, let me pick another example closer to home: the prescription of fish oil to people with hypometabolism, autoimmune disease, chemical sensitivity syndromes, asthma and migraine headaches. My point is not that the fish oil is necessarily recommended specifically for these reasons. Indeed, it often is recommended for general health reasons, because it is believed to be an “essential” nutrient and seemingly beneficial all around. But the dark side of fish oil is not nice. And some people end up taking amounts of fish oil equal to eating an entire salmon. Their underlying hypometabolism that gets better at the start can get worse due to inhibition of mitochondrial efficiency and peroxidation-induced inflammation. And new problems emerge: 1) accelerated mitochondrial aging, 2) increase wrinkling and aging of the skin, 3) systemic immune system suppression, and 4) strongly increased risk of cancer.

There are three points that I’d like to put on the table for your consideration.

First, failure to treat the underlying cause of disease is likely to boomerang on you. This is the central failure of allopathic medicine for chronic and degenerative diseases. Allopaths have no problem recognizing that a broken bone must be set for it to heal, but degenerative disease—they do not know what

\textsuperscript{256} If a teeter-totter analogy works better for you, by all means. The teeter-totter tips one way to let oxygen get on board, then tips the other way to let CO\textsubscript{2} get on board.
the causal mechanisms are, and resort to mythical views, like cholesterol causes heart disease (and statins are the remedy), or cancer is caused by wayward cells (which must be cut out by surgery or killed by poison or radiation).

A symptom is merely a biofeedback signal that something is wrong. Suppressing it is like turning off a fire alarm without putting out the fire.

Second, failure to treat the bottleneck of a metabolic process is likely to boomerang on you. If you have a deficiency of B12, taking folic acid causes serious problems. If you have generalized resistance to thyroid hormone, taking fish oil is counterproductive in the long term. So it may be wise to maintain some doubt about any decision you make regarding diagnoses and underlying causes.

Treating the wrong problem is like ordering more tires at a car factory that has a shortage of transmissions.

Third, and the critical point of this exposition on metabolic balancing, is that treating an anabolic-alkaline-anaerobic imbalance with just any catabolic-acidic-aerobic influence may have less-than-ideal results. Yes, selenium is a catabolic nutrient and an antiviral nutrient, but if selenium status is already high (above average), it may take a very large selenium intake to affect a small catabolic effect. The lack of proportion may be analogous to the above two examples. Furthermore, the taking of large doses of selenium may induce a compensatory adaptation in other catabolic mechanisms. For example, catabolic effects of fish oil at increasing cellular and mitochondrial membrane fluidity and permeability typically result in increased production of cholesterol to shut down membrane permeability and increase membrane rigidity. The opposite state, a selenium deficiency, might result a strong catabolic effect and produce a profound antiviral effect from a very much smaller dose of selenium.

I am not suggesting that the anabolic-catabolic continuum is as substance specific as metabolism is. With metabolism, none of the other B-complex nutrients will substitute for a B1 deficiency, and none of the other minerals will substitute for a zinc deficiency. Each cofactor/element is unique in its metabolic mechanism, and all must be present for metabolism to function. Just as pistons cannot substitute for rods in a car engine, and vinegar cannot be substituted for vanilla in a recipe, essential nutrients play a unique and indispensable role in metabolism. But among the anabolic-catabolic spectrum, the balance is not so specific. Anabolic and catabolic agents act in a generalized way.

But despite this non-specificity, there are characteristics that are not fully general. For examples, some anabolic nutrients tend to accumulate in specific compartments, like sodium in the blood and potassium in the cells. And there are water-soluble anabolics, like sodium and potassium, and fat-soluble anabolics, like vitamin E and cholesterol. Sodium does not substitute for potassium or cholesterol in the metabolic-balancing arena.

The image I’d like to suggest is a balancing scale. On the big-picture level, one side weighs the alkaline influences and the other side weighs the acidic influences. But on each side of the scale are other scales, which are weighing subsets of alkaline and acid influences—within the alkaline set and within the acidic set. Furthermore, each compartment of the body has its own scale. There are different aqueous scales for the blood, interstitial fluids, cytoplasm, and cellular organelles (nucleus, mitochondria, lysosomes, peroxisomes, etc.). The cell membranes and mitochondrial membranes have different lipid scales. There is much fine detail within the coarse acid-alkaline picture.

With this view, one can potentially distinguish differential effects from seemingly comparable influences. For examples, vitamin A and vitamin D are both acidifying, but their balancing potential may be quite different in different people.
Whether this difference is related to a nutritional deficiency that is corrected does not necessarily need to be discriminated. The difference between vitamin A and beta-carotene, for example, could be more about their vitamin activities (vitamin A is high and beta-carotene is low) than about their acidifying differences (vitamin A is moderate and beta-carotene is mild). Vitamin D supplementation could adversely compete with vitamin A absorption in the gut, thus unbalancing the vitamin A influence on the lipid-soluble acid-alkaline scale.

The take-home message is 1) be even handed rather than heavy handed in terms of chronic anti-viral interventions involving supplements, diets and lifestyle (don’t put all your eggs in one basket), and 2) pay attention to nuances of efficacy so as to be receptive to messages of differential benefit from things that may seem closely parallel (don’t put only eggs in your basket).

Regarding BHT, the message would be 1) use BHT with other, nutritional and/or hormonal modalities, and 2) if the BHT does not completely stop outbreaks by itself, add other modalities rather than push the dose up beyond that with which you are comfortable, or into the liver-toxic range.

If you learn something about the way your viral disease works, consider parallel approaches. For example, if you are an aerobic exercise enthusiast and pull a hamstring so that you cannot exercise, and have an outbreak, consider that the exercise is raising your metabolism and that some other method of raising your metabolism is needed, not only when you are sidelined nursing an injury, but possibly on a sustainable lifestyle basis. There may be a very good reason that you are an exercise enthusiast; your subconscious mind figured out long ago that exercise makes you feel better (and balances your metabolism).

As another example, you have outbreaks in synch with your menstrual period, which points to estrogen effects as an influence for viral susceptibility. Hormone replacement therapy with progesterone (an estrogen antagonist), estriol (an estrogen balancer), iodine (an estrogen transformer), or dietary phytoestrogens (estrogen competitors) might be considered. Then again, since estrogen’s proviral effect may be mediated through an inhibiting effect on basal metabolic rate, maybe your estrogen sensitivity is resulting from thyroid hormone inadequacy.

And in both males and females, estrogen influence could be increased by 1) food allergies, which might be most effectively mediated by dietary changes that reduce food allergies, 2) respiratory allergies, which might be most effectively mediated by air filtration that reduces exposure to mold spores, or negative ions, which cause mold spores and dust to stick to the walls, 3) infection, which could be something as simple and common as toe-nail fungus, 4) positive-ion pollution, which inhibits about 15% of aerobic metabolism, 5) leaky gut syndrome, which can be a foundational cause of food allergy reactions and systemic microbial infections.

Chronic inflammation might be caused by a lack of vitamin C, which is unable to facilitate the maturation of collagen infrastructure in vascular tissues.

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257 This book was originally written for people with chronic viral problem, like herpes, CMV, EBV and hepatitis. But in recent editions, my mission has expanded to include acute viral infections. I believe it is important to realize that whatever sustainability issues we may have for long-term management of chronic viral conditions do not apply to resolving short-term acute viral infections. In other words, survival is orders of magnitude more important than sustainability. In still other words, it is wise to burn tomorrow’s candle to survive today’s darkness.
Appendix B: Introduction to BHT

BHT (butylated hydroxytoluene) is a synthetic antioxidant with GRAS (generally recognized as safe) legal status in the United States. BHT has been widely used in the US to prevent rancidity in fat-containing foods, such as breakfast cereals, baked goods, potato chips, sausage, peanut butter, instant potatoes, and other commercially prepared foods. Even foods labeled “no preservatives” or “no preservatives added” may contain and often do contain BHT, which was present in the ingredients used in making the food and therefore does not need to be disclosed on labels.

You can thank the FDA for that.

The typical daily intake in the USA is estimated to be about 2 mg. The only apparent long-term effect from the small amount of BHT that most Americans get is a statistical reduction in the incidence of gastrointestinal cancer since this preservative first came into commercial use in 1947.

BHT is structurally related to the antioxidant features of (1) tocopherols (the vitamin E family of nutrients) and (2) many naturally occurring phenolic and polyphenolic antioxidants (flavones, isoflavones, flavonols, cyanidins, anthocyanidins, catechins, epicatechins, and quercetin analogs). BHT seems to have vitamin-like therapeutic properties in the body, possibly because it “preserves” the fat-soluble vitamins E (tocopherols and tocotrienols) and vitamin A from oxidative destruction.

For the past 40 years, many thousands of people have been taking from 50 to 2000 mg of BHT daily for its viral-protective and health-enhancing properties. Most of these people have used BHT for treating chronic viral infections, but rare cases of BHT’s use in treating acute viral infections have been noted.

Side effects are generally quite mild, especially at the lower dosages. A few people experience brief lightheadedness within a half an hour after taking BHT on an empty stomach. This can be minimized by taking it with meals, or by taking it lying down (i.e., just before going to sleep). Rarely, people have allergic reactions to BHT that manifest as skin problems, particularly rashes and dry, flaky skin. Such people should not take BHT. More commonly, people experience enhancement of the health of their skin. About half of the people who call me to share their experiences with BHT volunteer some comment about improvements in their skin appearance. I suspect that BHT helps the skin partly by direct enhancement of antioxidant protection in the outer dermis (what you see and touch) and partly by indirect preservation of vitamins A and E, both of which play a role in the health of the underlying dermis (the regenerating layer of the skin).

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258 For 50 years, BHT has been regarded as a synthetic-only substance. But in the last two decades, it has been reported as a natural plant compound (green-algae phytoplankton, three cyanobacteria and one fruit). Despite positive assertions that the BHT in these natural sources is natural, this has yet to be confirmed by identifying an enzymatic pathway for producing tertiary-butyl groups, a signature sub-structure in the BHT molecule.

259 In Japan, where BHT is not used, stomach cancer has remained high during the same period. Non-apparent (controversial) effects may include behavioral changes in children sensitive to BHT. This is reviewed in the toxicology chapter of The BHT Book. BHT can also produce sensitivity reactions in adults taking it in higher doses for medical reasons. This may be because “food grade” BHT is only 99.5% pure and the less-than-one-percent impurity increases with the dose. In other words, the dose of impurity from BHT’s use as a preservative is 100 times less than the impurity load from the 250 mg dose typically used to fight viruses. Nobody makes a “pharmaceutical grade” BHT.

260 This includes three cases of encephalitis progressing to coma, which reversed with BHT administration. This is especially relevant to coronavirus infection, where neurological symptoms have manifested in a minority of cases suggesting a neurotrophic capability for modified coronaviruses. The magnitude of this effect is not yet known, but over 2020, increasing emphasis has been placed on this possibility.
Some people still insist that BHT can cause cancer. The basis of this rumor is an old study in which rodents fed large amounts of BHT developed lung tumors. It was later revealed that the rodents’ feed had been contaminated with aflatoxin, a powerful natural carcinogen (cancer causing substance) produced by a mold called Aspergillus flavus, which commonly grows on grains and nuts. The carcinogenicity of peanut butter is primarily due to the traces of aflatoxin it contains. A subsequent bioassay performed by the National Cancer Institute showed BHT to be noncarcinogenic in rats and mice. Nevertheless, the FDA placed BHT on interim status and requested that further studies be done on its toxicity. These studies found BHT to be noncarcinogenic, non-mutagenic (doesn’t cause mutations), and nonteratogenic (doesn’t cause birth defects).

Part of the popular prejudice against BHT comes from the fact that it is a synthetic preservative which has been foisted upon an unsuspecting (or unwilling) public by the FDA and unscrupulous food vendors who do not appreciate the value of natural (and fresh) foods. While this may be true to a substantial extent, it is limited in its usefulness. Natural is not necessarily good and synthetic is not always bad. Aflatoxin and fatty acid peroxides (rancid fat) are quite natural and very toxic, while BHT and piracetam (the European “smart drug”) are 100% synthetic and safe enough to be on my lifestyle-supplements list.\(^{261}\)

Approximately 5% of the dry weight of most plants (i.e., vegetables and herbs) consists of chemicals that are specifically toxic to the predatory organisms which eat them, like nematodes (worms), insects and mammals. You can think of these chemicals as “natural insecticides,” for that is exactly their function. Plants cannot run away when something wants to eat them, so they defend themselves with “chemical warfare.”

Edible mushrooms contain hydrazine alkaloids, which are mutagens and carcinogens. Nightshade vegetables (tomato, potato, eggplant, green and red peppers, paprika, goji berries and tobacco) contain solanine, which can cause skin sensitivity and severe arthritis symptoms in susceptible people.\(^{262}\) Corn, peanuts and peanut butter frequently contains aflatoxin, which is a hundred thousand times more carcinogenic than alcohol. Celery contains psoralen (a photosensitizing chemical). The hypericin and pseudohypericin found in St. John’s wort are also photosensitizing chemicals in addition to being antiviral agents. Basil contains estragole (a mutagen). Alfalfa sprouts contain canavanine, an amino-acid “mimic” which causes a lupus-like autoimmune disease in monkeys—and probably humans. Wheat and soy contain estrogenic substances which interfere with the sexual function (and reproduction) of male mammals. And coffee, tea and chocolate contain caffeine and theobromine, both of which are natural insecticides.\(^{263}\) (there’s enough caffeine in used coffee grounds to discourage bugs from taking up residence in your vegetable garden or compost pit).

Contrast the toxicity of such natural chemicals with the “smart drug” piracetam. Despite being entirely synthetic, piracetam is considered the treatment of choice for newborn infants with myoclonic seizure disorders at dosages of 12-24 grams per day! That’s 1-2 heaping tablespoons of piracetam. Maybe vitamin C is less toxic.

\(^{261}\) In the last two decades, BHT has been found in a variety of plants. So it may turn out to be natural after all. But this remains unverified due to the presence of t-butyl groups in pollutants that might give rise to abnormal chemical processes driven by free-radical mechanisms and NOT biological enzymes [Dembitsky 2006, Bisel et al. 2008, Dembitsky et al. 2017]. To date, no t-butyl enzyme system has been identified.

\(^{262}\) Solanine alkaloids are the basis for the “eggplant skin-cancer cure.”

\(^{263}\) There’s enough caffeine in used coffee grounds to discourage bugs from taking up residence in your vegetable garden or compost pit.
Of course, there is no reason that BHT cannot easily be combined with nutrition, exercise, mitochondrial nutrition, metabolic balancing and hormone replacement as part of an integrated anti-viral program.

It is my hope that some readers and professionals may overcome significant prejudices against BHT to be able to use it effectively in cases where it is imperative. For examples, (1) herpes encephalitis is considered terminal when it progresses to coma, and (2) there is now evidence that the breathing difficulties associated with coronavirus may be caused by infections of the respiratory center of the central nervous system.264

There is now autopsy data in support of that hypothesis.265

Any CNS virus can be difficult to detect.266 The authors write, “...in cases of viral encephalitis involving the most prevalent viruses known to reach the CNS (mainly herpesviruses, arboviruses and enteroviruses), an actual viral presence can only be detected in 3 to 30 cases out of 100,000 persons.” This means that if it is happening, it’s not likely to be observed easily.

I'm now wondering whether this breathing pathology associated with coronavirus might be a “subclinical” encephalitis that is progressing towards respiratory coma. If so, a treatment that is able to cross the blood-brain barrier might be needed.

Over the last 40+ years, I have received three anecdotal reports of herpes encephalitis coma that were reversed by BHT (butylated hydroxytoluene).

In those three cases, BHT was given by the family and not the attending medical team. I believe that they reasoned that herpes encephalitis progressing to coma is invariably fatal, so why not try some heroic measure? But with coronavirus infection of the brain respiratory centers, the majority of those placed on ventilators do not survive. So is that a moral/ethical imperative to try BHT?

So the question I'm asking is two part, (1) is the respiratory suppression caused by coronavirus strictly limited to lung pathology, or is it partially mediated through coronaviral infection of the respiratory center in the CNS? and (2) would that suppression resolve with BHT administration?

BHT is well known to cross the blood-brain barrier, and it is well known to act against lipid-enveloped viruses and less well known to act against viral diseases. Although coronavirus is lipid enveloped, I know of no research testing its response to BHT. I cannot even find any research connecting the SARS and MERS viruses with BHT.


265 J Meinhardt, J Radke, C Dittmayer, et al. Olfactory transmucosal SARS-CoV-2 invasion as port of Central Nervous System entry in COVID-19 patients. BioRxiv doi: https://doi.org/10.1101/2020.06.04.135012, 6 June 2020. Abstract: “The newly identified severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) causes COVID-19, a pandemic respiratory disease presenting with fever, cough, and often pneumonia. Moreover, thromboembolic events throughout the body including the central nervous system (CNS) have been described. Given first indication for viral RNA presence in the brain and cerebrospinal fluid and in light of neurological symptoms in a large majority of COVID-19 patients, SARS-CoV-2-penetration of the CNS is likely. By precisely investigating and anatomically mapping oro- and pharyngeal regions and brains of 32 patients dying from COVID-19, we not only describe CNS infarction due to cerebral thromboembolism, but also demonstrate SARS-CoV-2 neurotropism. SARS-CoV-2 enters the nervous system via trespassing the neuro-mucosal interface in the olfactory mucosa by exploiting the close vicinity of olfactory mucosal and nervous tissue including delicate olfactory and sensitive nerve endings. Subsequently, SARS-CoV-2 follows defined neuroanatomical structures, penetrating defined neuroanatomical areas, including the primary respiratory and cardiovascular control center in the medulla oblongata.”

So, I'm putting it out there. If anybody tries BHT in coronavirus infection where respiratory difficulties are present, what happens? With the three cases of herpes encephalitis come mentioned previously, the response to BHT was rapid.

In situations where oral use is not practical, for mechanical or procedural restrictions, BHT can be applied transdermally (15% by weight dissolved in refined coconut oil) (1) to the sides of the neck, (2) to other skin surface less proximate to the brain, or (3) through a suppository or rectal infusion. All three of these transdermal approaches bypass the first-pass effect in which the liver can metabolize a substantial portion of the BHT before it gets to the brain.

And lastly, please pass on your observations.

For more information, you can download The BHT Book as a free PDF file from the Steve page at the Project Wellbeing website (http://projectwellbeing.com/steve/) or the 2020 version directly from my cloud account (https://drive.google.com/open?id=1k7uT5AKQYXt4LqX2jfWLRAxmeU2vpNEh).

**BHT Sources**

You can buy BHT capsules or bulk BHT from a number of mail-order or on-line dietary supplement companies. There are at least four companies that I know of that sell BHT capsules:

1) Lifelink, in California (www.LifeLinkNet.com),
2) Wholesale Nutrition, in Illinois (www.nutri.com),
3) Life-Enhancement products, in Nevada (www.life-enhancement.com)
4) SuperSmart, in Florida (us.supersmart.com)

Most of these products are also available through Amazon vendors.

Non-capsulated bulk BHT is harder to find. When sourcing BHT powder or crystals, be sure that it is food grade (FCC standard) and not feed grade or technical grade. Here are some sources:

1) www.myworldhut.com/search.php?search_query=BHT&x=0&y=0
2) https://pforlife.com/search.php?search_query=BHT (also selling on Amazon)

Some of these sources may stop selling BHT before you access them. Many of these companies operate within US jurisdiction, which means that their ability to sell BHT depends on the whims of FDA policy. Such policies are subject to change.

**BHT Administration**

BHT can be taken orally, in capsules, as powder, or dissolved in oil. If you use powder or granules, be aware that the squeaking sound echoing through the bones of your head from chewing BHT is completely normal. Oral BHT is absorbed in the intestine through the action of bile acids. For some who do not absorb lipids efficiently, the best results are obtained through pre-dissolving the BHT in oil. For others, taking BHT straight works best. So if one does not work well, try the other. All BHT taken orally passes through the liver before going to other organs. The liver metabolizes a substantial portion of oral BHT before it gets into general circulation.

BHT can also be taken transdermally to avoid the liver first-pass effect. This can be through rectal BHT-in-oil insufflation or suppositories, or skin application of BHT dissolved in oil. I recommend refined
coconut oil to avoid unpleasant smell, but any oil will do in a pinch. For somebody on a respirator, transdermal delivery to the sides of the neck may be the most efficient delivery option. But any thin-skinned area will absorb BHT fairly efficiently.

**BHT Dosing**

BHT can have adverse effects on liver enzymes. In 40 years of collecting anecdotes, I’ve never heard of a case of elevated liver enzymes in anybody taking between 50 mg and 1,000 mg. But there have been many reports of liver enzyme elevations from people taking 2,000 mg, but some report being able to tolerate two grams per day for months and years on end. So if you do go above one gram doses, get your liver enzymes tested to be sure.

**Lipid-Enveloped Viral Diseases**

The case for BHT is driven partly by the clinically troublesome nature of lipid-enveloped viruses. These viruses are often extremely difficult to treat, and the drugs and therapeutic strategies are widely considered inadequate and/or ineffective for the task. Calls for more research and better tools are common. Failed investigations into new antiviral compounds are common.

This is a list of known lipid-enveloped viruses:

1. coronavirus (several, including SARS, MERS and Covid-19 (SARS-CoV-2),
2. cytomegalovirus (CMV),
3. dengue fever virus,
4. Ebola virus (and Marburg virus; hemorrhagic-fever viruses),
5. Epstein-Barr virus (infectious mononucleosis),
6. hepatitis virus (types B, C and D, not A and E),
7. human immunodeficiency viruses (HIV),
8. influenza (all strains, including swine and bird flus),
9. norovirus (“winter vomiting bug,” causes gastroenteritis),
10. rubella virus (German measles virus),
11. SARS virus (a new coronavirus that infects humans),
12. varicella zoster virus (a herpes family virus causing chicken pox and shingles),

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267 Clinical effectiveness of pharmacist-led versus conventionally delivered antiviral treatment for hepatitis C virus in patients receiving opioid substitution therapy: a pragmatic, cluster-randomized trial. The Lancet 8 June 2020. Although this article postulates that “highly effective direct-acting antiviral drugs” provide the opportunity to eliminate hepatitis C infection, the actual efficacy of the program against hepatitis C was dismal. Only 7% of the patients achieved “sustained virologic response” on the drugs, which was considered a “high treatment success” by being compared to 3% of the non-pharmacist-led patients. And this is even worse when you consider that a “sustained virologic response” is only a surrogate marker actual clinical improvement, falling grossly short of the claimed opportunity to “eliminate” hepatitis-C viral infections.

268 S H Mehta, G M Lucas, L B Mirel, et al. Limited effectiveness of antiviral treatment for hepatitis C in an urban HIV clinic. AIDS 20(18): 2361-9, November 2006. Conclusions: “Although the potential for SVR [sustained virologic response] and the recent marked increase in access to HCV care are encouraging, overall effectiveness of anti-HCV treatment in this urban, chiefly African American, HCV genotype 1 HIV clinic is extremely low. New therapies and treatment strategies are an urgent medical need.”

11) variola virus (smallpox virus),
12) West Nile virus,
13) yellow-fever virus, and
14) Zika virus.

Animal lipid-enveloped viral diseases include:
1) bird flu (also infecting humans),
2) canine and feline distemper,
3) coronaviruses (infecting dogs, cats, birds, camels and bats),
4) cowpox (vaccinia, used to vaccinate humans against smallpox),
5) Newcastle disease (birds and horses),
6) pseudorabies (swine, crossing into rabbits, cattle, sheep, goats, cats, dogs and raccoons),
7) rabies virus (bats, raccoons, skunks, crossing into humans),
8) Semliki Forest virus (also infecting humans),
9) swine fever (pigs), and
10) swine flu (also infecting humans).

No serious effort has been made to make this list comprehensive.

**Common Non-Enveloped Viruses**

- Rhinoviruses (the primary cause of the common cold), and
- Poliovirus (the cause of polio disease).

**The BHT Book**

If you want more information on BHT, consider downloading *The BHT Book*, which devotes more of its content to BHT than nutrients, herbs and other natural agents. Nevertheless, part of the book is written for people who do not wish to use BHT against viruses, but are instead looking for a “natural” solution to viral infections. The natural options that will be discussed include herbal extracts, vitamins, amino acids, minerals, fats, mitochondrial nutrients and hormones. Examples include:

- **herbs** hypericin (an extract of St. Johns wort),
- **vitamins** A, D3, B6 and B12,
- **amino acids** cysteine, N-acetylcysteine (NAC) and glutathione,
- **minerals** selenium, magnesium, copper and strontium,
- **fats** polyunsaturated fatty acids (PUFAs) and medium-chain triglycerides (MCT fats),
- **mitochondrial nutrients** lipoic acid, B1, B2, B3, NADH, coenzyme Q10 and carnitine,
- **hormones** pregnenolone, progesterone, testosterone, low-dose cortisol, T3 and T4.

This book will also include discussion of lifestyle factors that influence viral virulence and viral susceptibility, like breathing, diet, exercise, detoxification and sun exposure. Not all this information is the typical stuff you may have heard from thousands of sources, in books, magazines, television and the web.

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For example, many herpes sufferers are aware from personal experience that sun exposure (sunburn, or even mild sun-induced skin redness) can trigger herpes flare-ups. However, not nearly as many people know that the vitamin D from regular mid-day sun exposure is a strong antiviral influence, and that avoiding sun completely (or automatically using sun-screen products) can *increase* the likelihood of having herpes flare-ups (and increase the risk of skin cancer).
Appendix C: Vitamin C Details

“Human beings are the only animal on the planet with an infinite capacity for rationalization.”

That statement goes to the heart of why vitamin C is so maligned, and so loved, depending on who you talk to. Human beings have an unfortunate difficulty in dealing with issues too close for comfort. What I’m going to attempt is to sort it all out for everybody. And with the full awareness that my “sorting” will piss off the majority of people reading this, who have sacred truths to lose.

Since I’m going to conclude that vitamin C is a critical part of anti-viral prevention and anti-viral therapy, let me start by pissing off Dr. Andrew W. Saul, one of the pioneers of vitamin C therapy and one of the most vocal critics of institutional resistance to using vitamin C in routine medical practice. Dr. Saul has said that “vitamin C is the most important antioxidant nutrient in your body” when the truth is that all antioxidant nutrients that play an essential role in the redox-defense system are important to the degree of their deficiency. If your vitamin C is robust and your selenium is not, it is selenium which is “the most important antioxidant nutrient” in your body.

The reason that I am being so picky here is that I am overcompensating for my bias. Having read information about vitamin C and health for more than four decades, I agree with Dr. Saul that vitamin C has been much maligned by scientists, bureaucrats, doctors and reporters with no care for fact or truth. Since only scientists can be held responsible for their knowing participation in this libel-and-slander campaign, I must be precise in my use of language to a degree that they are not. This will allow me to show you how they, the supposed scientists doing “science,” have deliberately mislead you, your friends, your neighbors, your politicians, your public health officials, your reporters and your doctors.

Dr. Saul’s statement, taken out of context, provides the exact illustration of this misuse of language, and the deliberate ignorance of context. The rules for understanding vitamin C are different because it is unique to humans in the context of a transplantation of circumstance: we no longer live in the environment where we lost the ability to synthesize vitamin C.

If the politics of vitamin D were the same as vitamin C, I’d be branded as “racist” for pointing out that vitamin D deficiency is a matter of skin color. It would not matter in the slightest that this is a statement of abject fact (see page 30). I would be accused of racism and my comments ignored, and those of others saying the same thing, ignored, all because of the superficial appearance of racism. The facts would never be part of the media process. But as point of fact, when human beings with dark skin adapted to high ultraviolet-B radiation exposure (latitude 10 degrees) are captured into slavery, shipped overseas to tend fields in the southern USA (latitude 33 degrees), then driven by brutal prejudice to more even further north (latitude 45 degrees) for the haven of “pretend” non-prejudice, their skin-color adaptation goes from adaptive, to slightly maladaptive, to seriously maladaptive.

The same thing has happened to vitamin C. Human beings now live in areas where the dietary vitamin C is an order of magnitude lower than it was where we lost the ability to produce it. And this is routinely ignored by doctors, public-health officials and the media. And, just as people with dark skin can take vitamin D3 supplements to correct for latitude, we can take vitamin C supplements to correct for our genetic disability. This is a core aspect of natural anti-viral self-defense. And it is a core aspect of treating viral infections, from the common cold and polio (protein-capsid viral diseases) to influenza, coronavirus infections and acute hemorrhagic fevers (lipid-enveloped viral diseases).

I do find it interesting that pharmacokinetics, the study of drug concentrations versus drug effects, is a respected and practiced scientific discipline where drugs are concerned, but not when vitamin C is
concerned. If there are two conflicting studies, one that that drug A is clinically effective at 2 mg/liter and the other that it is ineffective at 1 mg/liter, the immediate hypothesis is that 1 mg/L is subtherapeutic. What would not be done is declare drug A to be ineffective. Yet that is exactly what has been done to vitamin C. It may be hard to imagine that degree of malfeasance on the part of seemingly legitimate scientists and such incompetent peer review by top-ranked journals. But that is the case. As you read these in the footnotes (or not), you will see that the playing field is not level and that your life has been placed at risk for political and economic reasons.271

Vitamin C is the weak link in the redox-buffering and antioxidant-defense systems. To deny that fact is like saying that one should not change the oil in your car’s engine, or that you should not drink hydrogen monoxide because it is one of the most powerful solvents in the universe.

It is the strength of the antioxidant defense system, as backed up by the redox-buffering capacity of your body, that determines whether you live or die from a severe oxidative challenge. It really does not matter whether that challenge is cause by a virulent virus, second- or third-degree burns, or mercury poisoning. What matters is whether your redox capacity is strong enough to withstand the oxidative insult, or not.

What happens when it’s not? Your cells and tissues start to shut down. Your immune system goes into overdrive. The inflammation gauge “pins the needle.” There is massive free-radical damage to every important class of molecules in your body. DNA, enzymes, lipids in membranes, mitochondria. It is catastrophic before it becomes fatal.

For the immune system, we call this a “cytokine storm.”

In redox-sensitive infants, we call this “sudden infant death syndrome.”

So it is not hyperbole to say that your life has been placed at risk.

Vitamin C is the weak link in the redox-buffering system because we do not make it. Glutathione, an even more important antioxidant, is not as weak because we can make it. So when glutathione and vitamin C are both depleted by oxidative stress, it is the vitamin C that fails first. It is only supplied by diet or supplementation. To not give vitamin C to somebody in a redox

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271 The political reasons are overt. They deal with the ideology of the allopathic medical industry, from education in medical school to regulation of practice via the standard-of-care doctrine. The economic and anti-competitive motivations are covert, especially from the doctors themselves. When this went to court in Chicago, the American Medical Association lost. The court found that the AMA’s longstanding claims regarding “unscientific” chiropractic practitioners did not stand up to scrutiny and that the practice of excluding chiropractors from hospital access was illegally anti-competitive.
crisis is like not giving water to somebody with dehydration, not giving air to somebody suffocating or not food to somebody who is starving. Yet that is exactly what is happening to those with Covid-19 on ventilators.

**Vitamin C and Kidney Stones**

One of the biggest myths used against megadosing of vitamin C is that it causes kidney stones. This has been falsified to stronger and stronger degrees as time has passed. So why do doctors continue to make a false claim in opposition to the predominance of the evidence? It’s a good question. And the answer has both rationalization and conservatism explanations.

Rationalization is a character flaw of rational-dominant people. Because my own rational dominance is second place to intuition, I have escaped some of this character flaw. But only some. So I can provide testimony to those readers who are not rational dominant and cannot understand this mode of belief. Rationalization is the process of coming to a conclusion and then developing a rational explanation for that conclusion. Actually, the rationalization does not need to be rational at all, it just needs to appear to be rational to the rationalizer.

The eye of the beholder and all that.

This is not intrinsically bad because the rationalizer does not need to believe in the rationalization. Much of science is accomplished by rationalizations which were then proposed as theories. The contribution of imagination to scientific progress is vastly underappreciated. But proposing a theory for consideration by peers is not the same thing as believing that the rationalization is true. There is the process of falsification. If the theory is falsified, it must be modified or discarded. When the theory that vitamin C caused kidney stones because it was metabolized into oxalate, which was found in kidney stones, was tested in medium-

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This is based on Jungian personality traits: with intuitive-sensory dimension (N or S), thinking-feeling dimension (T or F), introversion-extroversion dimension (I or E) and judging-perceiving (J or P) dimensions, providing 16 personality polarities, eight of which can manifest in a single person. I’m the INTP type, but long believed I was an INTJ. My thinking-feeling split is quite marked, which gives me a significant tendency to rationalize everything around me. So I notice this in others. But my intuitive-sensory split is extreme, which gives me profound mental skills that do not make sense to the rational side of me (and impairs my ability to understand the those that are sensory types). For example, I can think about a problem before going to sleep and wake up with the answer or a design in my mind. And I feel totally at home doing abstract painting as a form of self expression. So I tend not to have the extreme arrogance of thinking types who go through school at the top of their class and escape most childhood criticisms, only to run into college criticism, when the brain is less plastic and adaptable to take such criticisms in a healthy emotional context, or worse, graduate-school criticisms, where the trauma of criticism is most traumatic to the thinking types. This arrogance is an adaptation to the fear of criticism and is one of the primary reasons why scientific advances take place “one funeral at a time” (as the adage goes). Rational types get “stuck” on their contribution to human progress and defend their creations at all costs, including preventing better creations from supplanting them. This is one of the human foibles that supports cronyism in academia and bureaucracies.
and high-dose vitamin C studies, the incidence of kidney stones went down. An honest scientist would then discard the idea, or modify it. Stanford professor and quackbuster Wallace Sampson did nothing of the kind. He continued to make the falsified assertion.

However, other scientists not so dogmatic and uneducable did modify the theory. They found that kidney stones were not formed by spontaneous precipitation (crystallization) of pure calcium oxalate, but rather that there was a gross substructure in the stone that looked like bacteria-shaped hollows. Sure enough, a new theory was postulated that kidney stones were formed around bacterial colonies and their biofilms.

The full theory is illustrated in the above graphic. Three factors influence kidney-stone formation. Oxalate concentration (which vitamin C often aggravates), bacterial infection at the stone formation site (which vitamin C minimizes), and a local abundance of calcium ions (which vitamin C mitigates).

Why zero kidney stone formation with 10-20 grams of vitamin C? One common explanation is that vitamin C enhances immune function, and this resolves focal infections. I suspect that the excretion of excess vitamin C through the kidney into the urine also has something to do with it. It makes sense to me. Immune incompetence due to redox abnormalities has been observed, and some practitioners believe that subclinical infections are far more common than is popularly believed.

But I’m going to also suggest that the dose of vitamin C also affects oxalate in a non-linear manner. In other words, oxalate conversion may not be proportional to ascorbate concentrations.

In situations where vitamin C is low, it tends to be more fully oxidized at any given level of oxidative stress. It’s like the proverbial sponge, for any given amount of spilled water, the smaller the sponge the wetter it gets.

It is only the fully oxidized vitamin C that degrades into oxalate. The half-oxidized ascorbyl radical anion (see below) is thermally stable. It is only dehydroascorbic acid (see below) which is thermally unstable.

When medium or high-dose vitamin C is taken, the sponge is larger. Many times larger. This means that there might be less dehydroascorbate to form oxalate. This would be particularly true if the oxidative stress did not change. Indeed, we know that oxidative stress is often not stable. Some people taking large doses of vitamin C do so because of increasing oxidative stress. Nevertheless, we can see a possible way that higher doses of C could actually lower oxalate levels. In addition, restoring redox buffering eases the

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![Vitamin C, Oxalate and Oxidative Stress](image-url)
dehydration that often accompanies elevations of oxidative stress. Dehydration is, itself, a risk factor for oxalate and urate precipitation.

I can also see a way that low-dose vitamin C could increase kidney-stone formation. If the dose of vitamin C is quite low or maybe intermittent, and accompanied by deficiencies of vitamin D3, K2 and/or magnesium, the slight elevation of oxalate could be accompanied by (1) a pronounced soft-tissue calcification that would facilitate biofilm calcification, and (2) an unchanged sub-clinical infection of the kidney and bladder. This may be the basis for the results found by a questionnaire-based Swedish cohort study273 in which those men taking supplements other than vitamin C were deliberately excluded from the study analysis. This exclusion would predictably optimize both infection and calcification contributions to stone formation by selecting for nutritionally and metabolically compromised Swedish men. The bottom line is that high-dose vitamin C does not cause kidney stones and can actually prevent them.

The evidence is not just early clinical reports from high-dose vitamin C studies for cancer where kidney stone formation was never observed, in men or women, in any age group. There have been multiple studies looking at the correlation of vitamin C intake with kidney-stone incidence. The Gary Curhan research team at Harvard did two large-scale food-questionnaire studies of people with no history of kidney stone formation. The first looked at 45,000 men over six years,274 and the second looked at 85,000 women over 14 years.275 Unlike the Swedish-men study cited earlier, both studies found no correlation of vitamin C intake with stone formation.

As this above research at Harvard was taking place, concurrent research at other institutions was taking place. In 1997, a re-analysis of the data found that people taking the highest intakes of vitamin C were found to have a lower risk.276 This study also identified flaws in the methodology of earlier studies in which stored samples continued to degrade vitamin C into oxalate. Some researchers may mistakenly believe that the oxalate pathway is enzymatically driven. It is not. It is strictly a thermal mechanism.

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275 G Curhan, W Willett, F Speizer and M Stampfer. Intake of vitamins B6 and C and the risk of kidney stones in women. Journal of the American Society of Nephrology 10(4): 840-45, April 1999. “A high intake of vitamin B6 was inversely associated with risk of stone formation. After adjusting for other dietary factors, the relative risk of incident stone formation for women in the highest category of B6 intake (> or =40 mg/d) compared with the lowest category (<3 mg/d) was 0.66 (95% confidence interval, 0.44 to 0.98). In contrast, vitamin C intake was not associated with risk. The multivariate relative risk for women in the highest category of vitamin C intake (> or =1500 mg/d) compared with the lowest category (<250 mg/d) was 1.06 (95% confidence interval, 0.69 to 1.64). Large doses of vitamin B6 may reduce the risk of kidney stone formation in women. Routine restriction of vitamin C to prevent stone formation appears unwarranted.”

This conclusion was contradicted in a later paper: E N Taylor, M J Stampfer and G C Curhan. Dietary factors and the risk of incident kidney stones in men: New insights after 14 years of follow-up. Journal of the American Society of Nephrology 15(12): 3225-32, December 2004. A new analysis using adjusted risk factors from diet-inferred potassium levels suggest a linear increased risk with low dose vitamin C intake. The age-adjusted risk ratio after age correction was 1.0, 1.0, 0.84, 0.97 and 1.01 for estimated vitamin C intakes of <90 mg, 90-249 mg, 500-999 mg, and 1000 mg and more, respectively. When adjusted for potassium intakes, the risk ratios rose to 1.0, 1.22, 1.20, 1.36, 1.41, respectively. Were diuretics confounding?

276 H Gerster. No contribution of ascorbic acid to renal calcium oxalate stones. Ann Nutr Metab 41(5): 269-82, 1997. “In the large-scale Harvard Prospective Health Professional Follow-Up Study, those groups in the highest quintile of vitamin C intake (> 1,500 mg/day) had a lower risk of kidney stones than the groups in the lowest quintiles.” This contradicts the earlier Harvard findings of no correlation.
Two years later, Simon and Hudes measured actual vitamin C levels in blood (rather than using food questionnaires) and quantified that reduced risk to 28% lower risk for every 1 mg/dL increase in blood ascorbate levels.277

This exactly illustrates how science is supposed to operate.

An excellent and comprehensive review of the many factors contributing to kidney-stone formation can be found in Thomas Levy’s classic book, Curing the Incurable (MedFox Publishing, 2002; pages 377-81.) The pathological role of calcium is fully reviewed by Levy in Death by Calcium (2013).

**Vitamin C Studies**

China was the first country to try intravenous medium-dose vitamin C for CoV-19 cases in a clinical hospital setting. This started in Shanghai, China and spread to multiple neighboring provinces.

The authors write,278 “Prevention and control of cytokine storms: High doses of vitamin C and ordinary heparin anticoagulant are recommended. High doses of vitamin C are intravenously injected 100 to 200 mg/kg per day. Continuous use time is aimed at a significant improvement in oxygenation index. It is recommended to apply high-dose broad-spectrum protease inhibitors, given 1.6 million units per 8 h 1 time, in a mechanical ventilation state, when the oxygenation index of 300 mmHg can be reduced to 1 million units/d. Anticoagulant therapy can be used to protect endothelial cells and reduce cytokine release, FDP s 10 sg/mL and/or D-diopolymers s5 sg/mL to ordinary heparin (3 to 15 IU/kg per hour) anticoagulant. The patient's clotting function and platelets must be reviewed at 4 h after first use of heparin. With ISVVH, 6 to 10 h per day.

**Wuhan University Zhongnan Hospital Study**

Chinese researchers operating in three Chinese hospitals in the Wuhan area conducted a small-scale “pilot” study of medium-dose IV vitamin C on a double-blind basis in severely ill Covid-19 patients.279 These

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277 J A Simon and E S Hudes. Relation of serum ascorbic acid to serum vitamin B12, serum ferritin and kidney stones in US adults. *Arch Inter Med* 159(6) 619-24, 22 March 1999. We have their Abstract Results quote, “We found no association between serum ascorbic acid level and prevalence of kidney stones in women or men (both P>.05).” And we have their Kidney Stone discussion where they write, “We were able to examine the relation of serum ascorbic acid level to history of kidney stones among a random sample of more than 10,000 Americans and found no evidence to indicate that high serum ascorbic acid levels increased the prevalence of kidney stones. On the contrary, among men, each 57-μmol/L (1.0-mg/dL) increase in serum ascorbic acid level was independently associated with an approximately 28% decrease in the prevalence of kidney stones (P =.06).” I find this odd in the extreme. Perhaps they failed to cite what they were referring to?


279 J Zhang, X Rao, Y Li et al. Pilot trial of high-dose vitamin C in critically ill Covid-19 patients. Research Square (pre-print publication): DOI: https://doi.org/10.21203/rs.3.rs-52778/v2. “Background: No specific medication has been proven effective for the treatment of patients with severe coronavirus disease 2019 (Covid-19). Here, we tested whether high-dose vitamin C infusion was effective for severe Covid-19. Methods: This randomized, controlled, clinical trial was performed at 3 hospitals in Hubei, China. Patients with confirmed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in the ICU were randomly assigned in a 1:1 ratio to either the high-dose intravenous vitamin C (HDIVC) or the placebo. HDIVC group received 12 g of vitamin C/50 ml every 12 hours for 7 days at a rate of 12 ml/hour, and the placebo group received bacteriostatic water for injection in the same way. The primary outcome was invasive mechanical ventilation-free days in 28 days (IMVFD28). Secondary outcomes were 28-day mortality, organ failure, and inflammation progression. Results: Only fifty-six critical Covid-19 patients were ultimately recruited due to the early control of the outbreak. There
were strictly patients with severe SARS-CoV-2 pneumonia admitted to the hospital ICU (intensive care unit) between the ages of 18 and 80, who did not have glucose-6-phosphate deficiency, nor end-stage pulmonary disease, nor pregnancy (or breastfeeding), nor a life expectancy of less than 24 hours. The placebo group was 28 of 29 and the medium-dose IV vitamin C group was 26 of 27 (one of each group was discharged in less than three days and never made it to seven days).

This was a follow-up to earlier research showing clinical benefits to IV doses of vitamin C at a 12-grams-per-day level, which because of its reported safety at much higher doses, was increased to 24 grams per day by these researchers. Given that this particular study was evaluating its clinical effects in end-stage Covid-19 disease, this was an excellent choice.

The IV vitamin C cut the mortality in half.

Intravenous vitamin C was administered twice per day for one week by slow central-vein pump, 12 grams each infusion. The placebo group received IV water in the exact same manner for the same seven days.

Other drugs and treatments were administered on a case-by-case basis by the blinded ICU team.

The illustration above shows a solid line for the mortality curves for the total patient populations. The researchers also examined the mortality curves for higher-risk patients (the dashed lines), who showed predictable increased mortality in the placebo group and possibly a decreased mortality in the vitamin C group.

Given that these patients were the most severely ill patients to study and there was a 50% decreased mortality makes vitamin C one of the most effective Covid-19 therapies ever investigated. At least potentially. It is important to keep in mind that this was a small study of just over 50 people where more than 120 would have been needed to provide sufficient statistical significance to make more definitive statements.

The researchers went on to measure multiple parameters during the week of vitamin C infusions (see data illustrated at right, following).

was no difference in IMVFD28 between the two groups. During the 7-day treatment period, patients in the HDIVC group had a steady rise in the PaO2/FiO2 (day 7: 229 vs. 151 mmHg, 95% CI 33 to 122, P=0.01). Patients with SOFA scores ≥3 in the HDIVC group exhibited a trend of reduction in 28-day mortality (P=0.06) in univariate survival analysis. IL-6 in the HDIVC group was lower than that in the placebo group (19.42 vs. 158.00; 95% CI -301.72 to -29.79; P=0.04) on day 7.

Conclusion: This pilot trial showed that HDIVC might show a potential signal of benefit for critically ill patients with Covid-19, improving oxygenation even though it failed to improve IMVFD28. Clinicaltrial.gov identifier and date: NCT04264533. Registered February 14, 2020."
Overall, the results were mixed, with three clinical aspects getting worse and four improving. Lung and kidney stress from the IV vitamin C seems an obvious interpretation, however arterial oxygenation (see P/F ratio) improved markedly while the other breathing difficulties manifested. A calculated P/F value of above 200 is considered mild ARDS (acute respiratory distress syndrome), whereas a value under 200 is considered moderate ARDS. So ARDS moved from moderate to mild in the vitamin C patients and moved from mild to moderate in the placebo patients.

It may be significant that the single patient on ECMO life support receiving vitamin C was able to be taken off life support, but the two not getting vitamin C stayed on life support. This parallels the cases of Allan Smith (see page 91) and Ryan Padgett (see page 93).

The possibly most significant clinical aspect was the effect of vitamin C on markers for organ failure, which is a huge issue with end-stage Covid-19 disease. The SOFA score (sequential organ-failure assessment) reversed itself during the week of vitamin C infusions, ultimately improving over baseline values by the end of the week, however, the placebo patients’ scores continued to get much worse during the week, going from a score of 2 to 6.

The illustration shows the vitamin C patient data in blue open circles and the placebo group in red solid circles, with positive findings in green and adverse findings in magenta. Where there is no Y-axis scale, numbers of involved patients are placed in large open circles.

**Vitamin C Can Shorten the Length of Stay in the ICU**

This meta study by two senior scientists with a long familiarity with vitamin C research found that time in intensive care (ICU) was significantly decreased by use of vitamin C.\(^\text{280}\) In one group of

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\(^\text{280}\) Harri Hemilä and Elizabeth Chalker. Vitamin C can shorten the length of stay in the ICU: A meta-analysis. *Nutrients* 11(4): 708, 27 March 2019. (This article belongs to the Special Issue Vitamin C in the Critically Ill, Effects on Oxidative Stress and...
a dozen primarily cardiac-surgery patients, the ICU stay was reduced by 7.8%. In another group of a half-dozen studies, vitamin C in doses between 1 and 3 grams per day reduced ICU stay by 8.5%. Given that ICU stay is thousands of dollars per day and three grams of vitamin C costs 30 cents per day, it’s a many thousand fold savings in money beyond the reduced pain and suffering.

(+ Metabolic summary reviews of C as well. Political summary, too. Chalmers!
(+ A triple-blind study, Zhongnan Hospital in Wuhan, China. Approx. 140 participants.
(+ Political comment: Dr. Richard Cheng shares “I was made aware that FB Fact Check claims “Shanghai did not officially recommend high-dose IVC for the treatment of Covid-19.”
Let me make it clear that not only Shanghai, but also Guangzhou, Guangdong Province, another major city in China, publicly endorsed high-dose IVC for the treatment of Covid-19.
Those who does Fact Check, please be more careful.”
47% reduction in mortality.
37% in the IV-C group versus 71% in the control group.
(+ add historical double-blind studies and anecdotal clinical reports.

## Vitamin C Effects on Pain

A long series of reports of vitamin C reducing or eliminating pain syndromes have regularly appeared in the literature over many decades. Many more have been passed from practitioner to practitioner. 

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281 Anitra C Carr and Cate McCall. The role of vitamin C in the treatment of pain: new insights. *Journal of Translational Medicine* 15: article 77, 14 April 2017. Abstract: The vitamin C deficiency disease scurvy is characterised by musculoskeletal pain and recent epidemiological evidence has indicated an association between suboptimal vitamin C status and spinal pain. Furthermore, accumulating evidence indicates that vitamin C administration can exhibit analgesic properties in some clinical conditions. The prevalence of hypovitaminosis C and vitamin C deficiency is high in various patient groups, such as surgical/trauma, infectious diseases and cancer patients. A number of recent clinical studies have shown that vitamin C administration to patients with chronic regional pain syndrome decreases their symptoms. Acute herpetic and post-herpetic neuralgia is also diminished with high dose vitamin C administration. Furthermore, cancer-related pain is decreased with high dose vitamin C, contributing to enhanced patient quality of life.

282 Anitra C Carr and Cate McCall. The role of vitamin C in the treatment of pain: new insights. *Journal of Translational Medicine* 15: article 77, 14 April 2017. Abstract: The vitamin C deficiency disease scurvy is characterised by musculoskeletal pain and recent epidemiological evidence has indicated an association between suboptimal vitamin C status and spinal pain. Furthermore, accumulating evidence indicates that vitamin C administration can exhibit analgesic properties in some clinical conditions. The prevalence of hypovitaminosis C and vitamin C deficiency is high in various patient groups, such as surgical/trauma, infectious diseases and cancer patients. A number of recent clinical studies have shown that vitamin C administration to patients with chronic regional pain syndrome decreases their symptoms. Acute herpetic and post-herpetic neuralgia is also diminished with high dose vitamin C administration. Furthermore, cancer-related pain is decreased with high dose vitamin C, contributing to enhanced patient quality of life. A number of mechanisms have been proposed for vitamin C’s analgesic properties. Herein we propose a novel analgesic mechanism for vitamin C; as a cofactor for the
Some deal with the kinds of severe, intractable pain from bone-cancer metastases. Others note the potentiation of opiate analgesia for improved pain control with lower doses of medications. And even more amazing, there is a repeating pattern of vitamin C blocking opiate habituation and addiction, facilitating the safer use of long-term prescription narcotics.

Regarding the latter, the conspicuous absence of mention of vitamin C in the national, media and worldwide public-health debate over the supposedly unacceptable death rate from “the problem” of prescription pain-killing drugs. I’d be baffled if it weren’t for my extensive experience with government bureaucracies. They never forget. The FDA never forgot nor forgave the popularization of the amino acid L-tryptophan by Psychology Today. Two decades later, they got their revenge when Showa Denko introduced contaminated GMO-derived tryptophan into the world market and caused EMS (eosinophilia myalgia syndrome), which gave the FDA the excuse to ban uncontaminated tryptophan, too.\(^{283,284}\)

Actually, they lied to the public and media; they did not actually ban it, they just said they did, put out press releases that declared that they did, and used that lie to arrest (!) uncontaminted tryptophan from those who were using safe tryptophan to treat EMS.

Regarding the enhanced pain-killing effects of vitamin C, it was Ewan Cameron and Linus Pauling who first noticed it in their studies of high-dose vitamin C treatment for cancer. So no forgetting nor forgiving after four decades—and untold narcotic-mediated deaths.

The graph at left shows pain levels in two women with shingles pain receiving 15 grams of IV vitamin C every other day.\(^{285}\) In one week, one was free of shingles pain. At two weeks, both women were pain free.

Vitamin C not only potentiates longer-term efficacy of opiates and other pain drugs, it ameliorates much of the effects of withdrawal in heroin addiction.\(^{286}\)
The effect has also been studied in rat self-administration of morphine where intraperitoneal injection of vitamin C decreased the frequency of self-administration. The compulsion to use higher and more frequent dosing of morphine was almost completely blocked by vitamin C.

The intraperitoneal injection of vitamin C in these rats is like oral intake of vitamin C in humans. The IP injection puts the vitamin C in the spaces between the folds of the intestine, where it is absorbed into the blood stream exactly like how oral vitamin C is absorbed.

Interestingly, IP vitamin C also interfered with the rat’s ability to learn the difference between pressing the morphine-dosing lever (red) and pressing a placebo saline-dosing lever (blue). While control rats and vitamin C rats could both slightly distinguish between morphine and saline, the morphine-only rats could readily distinguish the difference.

Here is another report on vitamin C effectively treating herpes and shingles pain.

Personaly, I’ve heard many dozens of anecdotes of BHT resolving shingles pain in less than a day (see Appendix B).

**Vitamin C and Cancer**

There is a long line of papers presenting data that vitamin C is effective at treating cancer in a variety of symptom] ranging from 10% to 16.6%, in contrast to the untreated subjects (control group), who expressed a major [withdrawal symptom] in 56.6% of the cases.”


288 J Y Chen, C C Chu, E C So, C H Ching and M L Hu. Treatment of postherpetic neuralgia with intravenous administration of vitamin C. *Anesthesia & Analgesia* 103(6): 1616-17, 2006. doi: 10.1213/ane.0000246396.64010.ee. A 78-year old man suffered from intermittent, spontaneous, shooting chest pain on his right side for 8 months. Low-dose intravenous vitamin C (2.5 grams on days 1, 3 and 5) resolved that pain. Dietary recommendations for increased fruits and vegetables was sufficient in preventing the return of the pain at 3 months follow up.
capacities. These include improved quality of life, increased duration of survival and some rare cures.

289 Chang Hwan Yeom, Gyou Chul Jung and Keun Jeong Song. Changes of terminal cancer patients’ health-related quality of life after high dose vitamin C administration. *Journal of Korean Medical Science* 22(1): 7-11, February 2007. Thirty-nine terminal cancer patients were given two 10-gram intravenous vitamin C doses separated by three days, with 4 grams of oral vitamin C for a week. “In the global health/quality of life scale, health score improved from 36+/–18 to 55+/–16 after administration of vitamin C (p=0.001). In functional scale, the patients reported significantly higher scores for physical, role, emotional, and cognitive function after administration of vitamin C (p<0.05). In symptom scale, the patients reported significantly lower scores for fatigue, nausea/vomiting, pain, and appetite loss after administration of vitamin C (p<0.005).”


292 Claudia Vollbracht, Berthold Schneider, Van Leendert, Gabriele Weiss, Leo Auerbach and Josef Beuth. Intravenous vitamin C administration improves quality of life in breast cancer patients during chemo-/radiotherapy and aftercare: results of a retrospective, multicentre, epidemiological cohort study in Germany. *In Vivo* 25(6): 983-90, November-December, 2011. “A total of 53 of these patients were treated with i.v. vitamin C (supplied as Pascorbin® 7.5 g) additional to standard tumour therapy for at least 4 weeks (study group) and 72 without this additional therapy (control group).” From Results: “IV vitamin C administration resulted in a significant reduction of complaints induced by the disease and chemo-/radiotherapy, in particular of nausea, loss of appetite, fatigue, depression, sleep disorders, dizziness and haemorrhagic diathesis [bleeding events]. After adjustment for age and baseline conditions (intensity score before adjuvant therapy, chemotherapy, radiotherapy), the overall intensity score of symptoms during adjuvant therapy and aftercare was nearly twice as high in the control group compared to the study group. No side-effects of the IV vitamin C administration were documented.”

293 A Murata, F Morishige and H Yamaguchi. Prolongation of survival times of terminal cancer patients by administration of large doses of ascorbate. *International Journal of Vitamin and Nutrition Research* Supplement 23: 103-23, 1982. Abstract: Clinical trials administering supplemental ascorbate to terminal cancer patients were conducted at two hospitals in Japan. During the period 1973-1977 there were 99 patients with terminal cancer at the Fukuoka Torikai Hospital. The average times of survival after the date of designation as terminal were 43 days for 44 low-ascorbate patients and 246 days for 55 high-ascorbate patients. Three of the high-ascorbate patients were still alive, their average survival being 1550 days, on April 1, 1980. Similar effectiveness of ascorbate was also observed at the Kamioka Kozan Hospital. There were 31 patients with terminal cancer during the period 1975-1979. The average survival times were 48 days for 19 control patients and 115 days for 6 high-ascorbate patients. One of the high-ascorbate patients was still alive, his survival being 215 days. In addition to the increase in survival times, the administration of large doses of ascorbate seemed to improve the quality of life.

294 Ewan Cameron and Linus Pauling. Supplemental ascorbate in the supportive treatment of cancer: Prolongation of survival times in terminal human cancer. *Proceedings of the National Academy of Science USA* 73: 3685-89, 1976. “The ascorbate-treated patients were found to have a mean survival time about 300 days greater than that of the controls. Survival times greater than 1 year after the date of untreatability were observed for 22% of the ascorbate-treated patients and for 0.4% of the controls. The mean survival time of these 22 ascorbate-treated patients is 2.4 years after reaching the apparently terminal stage; 8 of the ascorbate-treated patients are still alive, with a mean survival time after untreatability of 3.5 years.”

295 Christoph S Nabzydk and Edward A Bittner. Vitamin C in the critically ill – indications and controversies. *World Journal of Critical Care Medicine* 7(5): 52-61, 16 October 2018. Abstract: “Ascorbic acid (vitamin C) elicits pleiotropic effects in the body. Among its functions, it serves as a potent anti-oxidant, a co-factor in collagen and catecholamine synthesis, and a modulator of immune cell biology. Furthermore, an increasing body of evidence suggests that high-dose vitamin C administration improves hemodynamics, end-organ function, and may improve survival in critically ill patients. This article reviews studies that evaluate vitamin C in pre-clinical models and clinical trials with respect to its therapeutic potential.”


Vitamin C and Risk of Dying from All Causes

Yes. Vitamin C plasma concentration was inversely related to mortality from all causes.

Sepsis and Acute Respiratory Failure

This is an interesting study because the authors presented their data as a failure, yet the mortality data was statistically significant for a decrease in mortality from vitamin C. The vitamin C was infused over 96 hours (the first four days) and patients were followed for the next four weeks. Mortality was sharply reduced in the first four days, slightly increased for the next 4-5 days (see black arrow), and sustained for the duration of the study. The authors did not report this in their “results” summary. They conclude only that the “96-hour infusion of vitamin C compared with placebo did not significantly improve organ dysfunction scores or alter markers of inflammation and vascular injury.” Given the four-day timeline, this is not particularly edifying.

The standard of care regarding vitamin C among the mainstream medical profession does not include any contraindication for sudden discontinuation of vitamin C. Among functional medical societies, sudden discontinuation of vitamin C is not sanctioned. However, the reason that vitamin C was suddenly discontinued in this particular study protocol may not have been because of ignorance on the part of this medical team, but rather the

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298 K T Khaw, S Bingham, A Welch et al. Relation between plasma ascorbic acid and mortality in men and women in EPIC-Norfolk prospective study: A Prospective population Study. European Prospective Investigation into Cancer and Nutrition. *Lancet* 357(9257): 657-63, 3 March 2001. Findings: “Plasma ascorbic acid concentration was inversely related to mortality from all-causes, and from cardiovascular disease, and ischaemic heart disease in men and women. Risk of mortality in the top ascorbic acid quintile was about half the risk in the lowest quintile (p<0.0001). The relation with mortality was continuous through the whole distribution of ascorbic acid concentrations. 20 micromol/L rise in plasma ascorbic acid concentration, equivalent to about 50 g per day increase in fruit and vegetable intake, was associated with about a 20% reduction in risk of all-cause mortality (p<0.0001), independent of age, systolic blood pressure, blood cholesterol, cigarette smoking habit, diabetes, and supplement use. Ascorbic acid was inversely related to cancer mortality in men but not women.”

ignorance of the oversight group from the FDA which was supervising this CITRIS-ALI study.

In the adjacent graph, the difference between the mortality in the vitamin C group and the control group (the net lives saved) is graphed to better illustrate the three-phase response to IV vitamin C. The efficacy of vitamin C seen in the first 4-5 days is a direct effect of the intravenous vitamin C being administered (see cyan color). Within 48 hours of the last IV dose of vitamin C, there is a significant downturn in the survivability advantage seen in the first phase, which halved the survival from the IV vitamin C administration.

48-72 hours is the rebound timeframe for sudden withdrawal of vitamin C to produce “rebound scurvy.” This study is one more suggestion that sudden cessation of vitamin C is not wise.

The take-home message is that likely twice as many people would have survived if the IV vitamin C infusions had been continued until the treatment group was fully recovered. Instead of 70% survival, it might have been 80-90%. See the light blue extrapolation of the vitamin C group from day four towards the end of the study with the same shape as the curves of the control group and the IV vitamin C group from day-7 on.

Vitamin C and Respiratory Infections

Since we are talking about Covid-19, why not respiratory infections in general.\textsuperscript{300,301}

\textsuperscript{300} C Hunt et al. The clinical effects of vitamin C supplementation in elderly hospitalised patients with acute respiratory infections. \textit{International Journal of Vitamin and Nutrition Research} 64: 212-19, 1994. Abstract: “A randomised double-blind trial involving vitamin C/placebo supplementation was conducted on 57 elderly patients admitted to hospital with acute respiratory infections (bronchitis and bronchopneumonia). Patients were assessed clinically and biochemically on admission and again at 2 and 4 weeks after admission having received either 200 mg vitamin C per day, or placebo. This relatively modest oral dose led to a significant increase in plasma and white cell vitamin C concentration even in the presence of acute respiratory infection. Using a clinical scoring system based on major symptoms of the respiratory condition, patients supplemented with the vitamin fared significantly better than those on placebo. This was particularly the case for those commencing the trial most severely ill, many of whom had very low plasma and white cell vitamin C concentrations on admission. Various mechanisms by which vitamin C could assist this type of patient are discussed.” https://www.ncbi.nlm.nih.gov/pubmed/ 7814237.

\textsuperscript{301} H C Gorton and K Jarvis. The effectiveness of vitamin C in preventing and relieving the symptoms of virus-induced respiratory infections. \textit{Journal of Manipulative and Physiological Therapeutics} 22(8): 530-33, October 1999.
Chronological Citations for Further Reading on Vitamin C


Achille Kalokerinos. *Every Second Child*, Thomas Nelson, Australia, *1974*. A book about sudden infant death syndrome (SIDS) caused by Australian “official policy” vaccinations of aboriginal infants and its 99% effective treatment by vitamin C. What I find most damming is that the officials did not conduct any follow-up and were utterly unaware of the results of their vaccinations.


E. Cheraskin, MD. *Vitamin C: Who Needs It?* Atticus Press, 1993. This is a reply to those who refuse to examine ascorbate science and then dismiss the claim that ascorbate is an anti-viral medicine on the grounds that it has not been “proven” by “randomized, controlled trials.” It includes 22 pages of references to reports in the science of ascorbate, including some RCTs. For witty humor, see also the detailed meta-analysis of the RCTs for the clinical use of parachutes in saving lives from the trauma of gravity challenges (see page 228).


J A Simon and E S Hudes. Serum ascorbic acid and other correlates of self-reported cataract among older Americans. *J Clin Epidemiol* 52(12): 1207-11, December 1999. “Serum ascorbic acid level was inversely
associated with prevalence of cataract in multiple logistic regression analyses; each 1 mg/dl increase was independently associated with a 26% decrease in cataract (p = 0.03).”

**Post 2000**


Thomas E Levy, MD, JD. *Curing the Incurable: Vitamin C, Infectious Disease and Toxins.* MedFox Publishing, 2002. This book includes over 1,200 scientific references to published reports. Viral diseases are said to be “incurable” only because the speaker does not know about the vitamin C literature.

Steve Hickey and Hilary Roberts. *Ascorbate: The Science of Vitamin C.* Lulu.com, 2004. This is a careful, understated review of real science with 1,000 references.


Steve Hickey and Andrew W. Saul. *Vitamin C: The Real Story.* Basic Health Publishers, 2008. See especially chapter 2, “The Pioneers of Vitamin C Research” a review of the work of Linus Pauling and his reply to the false replication claim of the Mayo Clinic. Their tests of small doses of ascorbate did not show a strong benefit. He countered that often only large doses are effective and that most animals produce multigram amounts.


S Chambial, S Dwivedi, K K Shukla, P J John and P Sharma. Vitamin C in Disease Prevention and Cure: An Overview. *Indian Journal of Clinical Biochemistry* 28(4): 314–28, October 2013. doi: 10.1007/s12291-013-0375-3. Linus Pauling “was the first to introduce the concept of high doses of vitamin C for the treatment of various conditions from common cold to cancer. Since then mega doses of vitamin C have been widely used in the treatment and prevention of a large number of disorders like diabetes, atherosclerosis, common cold, cataracts, glaucoma, macular degeneration, stroke, heart diseases, cancer and so on.”

Jeffrey Dach, M.D.  Vitamin C saves dying man of viral pneumonia. 21 July 2013.  
http://truemedmd.com/vitamin-c-saves-dying-man/


N A Mikrova and Ronald Hunninghake.  Effect of high dose vitamin C on Epstein-Barr viral infection.  

Nick Lane, PhD.  Oxygen.  Oxford University Press, 2016.  See chapter 9 on vitamin C, which includes a rare discussion of Fenton chemistry (see pages 136 and 138).  Fenton chemistry is one way the energy barrier between electron-paired oxidants and electron-paired reductants (reverse oxidants) is reduced to become biologically useful (constructive redox reactions) or biologically damaging (pathological free radicals).  This production of superoxide, hydrogen peroxide and hydroxyl radicals only became possible after the evolutionary advancement of plant photosynthesis, which first killed off 99% of all species on the Earth and then enabled the evolution of multicellular life forms, fish, amphibians, reptiles, mammals, primates, apes and humans.  These Fenton-derived oxidants have been harnessed by the immune system to respond to infections.


L Ried, N Travica and A Sali.  The acute effect of high-dose intravenous vitamin C and other nutrients on blood pressure: A cohort study.  


Henry Hemilä.  Vitamin C and infections.  

Melissa Prier, Anitra C Carr and Nicola Baille.  No reported renal stones with intravenous vitamin C administration: A prospective case study.  


Margreet C M Vissers and Andrew B Das.  Potential mechanisms of action for vitamin C in cancer: Reviewing the evidence.  

Won-Young Kim, Eun-Jung Jo, Jung Seop Eom, et al.  Combined vitamin C, hydrocortisone, and thiamine therapy for patients with severe pneumonia who were admitted to the intensive care unit: Propensity score-based analysis of a before-after cohort study.  
Journal of Critical Care 47: 211-18, Oct 2018.  Results: “In the propensity-matched cohort (n = 36/group), the treated patients had significantly less hospital mortality than the control group (17% vs. 39%; P = 0.04).  The vitamin C protocol associated independently with decreased mortality in propensity score-adjusted analysis (adjusted odds ratio = 0.15, 95% confidence interval = 0.04–0.56, P = 0.005).  Relative to the control group, the treatment group had a significantly higher median improvement in the radiologic score at day 7 compared with baseline (4 vs. 2; P = 0.045).  The vitamin C protocol did not increase the rates of acute kidney injury or superinfection.”

John Costello, PhD. *Vitamin C Cures or Treats Many Illnesses*. 2nd edition, **2019**. Short and sweet.


Henry Hemilä. Vitamin C may reduce the duration of mechanical ventilation in critically ill patients: a meta-regression analysis. *J Intensive Care* 8: 15, **7 February 2020**.


I’ll add more to this list for the next version. It seems to be one of those never-ending stories.
Appendix D: Pharmaceuticals

Because the title of this book is *natural* self defense, the non-natural pharmaceuticals and generics are relegated to this appendix. It is my opinion that there is value in these options, especially in combination with the natural elements mentioned above like vitamin D and selenium.

Chloroquine and Hydroxychloroquine

This might be a suitable topic for the crony-science appendix due to the scientific malfeasance surrounding chloroquine and hydroxychloroquine research following US President Donald Trump’s public announcements regarding their use in treating Covid-19 cases. Several of the rapid-released “official studies” claiming lack of efficacy (or unacceptable toxicity) for chloroquine and hydroxychloroquine, which were frequently cited by Trump detractors (Democrats) as evidence of Trump’s public-health delusions, have since been withdrawn by their publishers. False data. Unsupportable conclusions. Very embarrassing. But since the fraud side of this science-fraud scandal is one that I have not yet bothered to investigate in depth, I will merely present the case for chloroquine’s and hydroxychloroquine’s efficacy as therapeutic agents.

For those who need some kind of scientific context, let me start with Simpson’s paradox. This is an explanation of how statistically significant data can be hidden by aggregating it with a larger data set. In this case, researchers have come to the erroneous conclusion that hydroxychloroquine has no efficacy in Covid-19 by aggregating the data from all studies of hydroxychloroquine. But if those researchers had done their job properly, and statistically analyzed subsets of those studies, they would have found that (1) early intervention with hydroxychloroquine with zinc is highly efficacious, that (2) early intervention with hydroxychloroquine without zinc is moderately efficacious, that (3) prophylactic dosing with hydroxychloroquine might be somewhat effective, and (4) late-stage intervention and (5) toxic dosing of hydroxychloroquine is not efficacious. This is real science, not the fake science motivated by political sentiments against President Trump and economic conflict-of-interest bias against generic pharmaceuticals.

This section is in Appendix D because chloroquine and hydroxychloroquine are drugs, not natural substances. This is in keeping with my promise to devote the bulk of this content to natural modes of anti-viral treatment, despite the fact that these drugs may work through a zinc-nutriture mechanism.

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French researchers have now joined the anti-hydroxychloroquine scandal. V Dubée, P-M Roy, B Vielle et al. A placebo-controlled double-blind trial of hydroxychloroquine in mild-to-moderate Covid-19. medRxiv doi: https://doi.org/10.1101/2020.10.19.20214940. Although they say that “patients treated with hydroxychloroquine did not experience better virological outcomes than those receiving the placebo,” their actual data did show better outcomes. They also manipulated their data by combining mortality data (deaths) with tracheal intubation data (mechanical ventilation), which allowed them to state that “the primary endpoint occurred in nine patients in the hydroxychloroquine group and eight in the placebo group.” Yet their data show that none of the three hydroxychloroquine patients on mechanical ventilation died and three of four patients on placebo died. And on day 28 of the study, 5 hydroxychloroquine patients had died compared to 9 placebo patients. Like the IV vitamin C study in China where mortality was decreased by half, this study failed to recruit the intended number of patients needed to reach statistical significance because of a slowdown in new Covid-19 patients in France during the summer. Yet the lack of statistical significance does not excuse an outright falsification in the conclusions by the authors. This falsification might simply be admitted to be a mis-statement on their part were it not for their second admission that “relative risk” could not be calculated for a subgroup of patients because “zero of ten patients” receiving hydroxychloroquine with azithromycin reached the primary endpoint (death, intubation) and “three of eleven patients” receiving placebo and azithromycin reached the primary endpoint. Zero versus three? This cannot be calculated? It actually can, but the mathematical answer is infinity. So, hydroxychloroquine was actually *infinitely* less risky than placebo, although the finding has minimal statistical significance.
I'd like to also point out that institutional resistance to these drugs from the pharmaceutical industry is based on the same, simple, straightforward anti-competitive motivations commonly applied to nutrients due to their mutual generic status. As the pharmaceutical industry's economic foundation of new-drug candidates has been contracting over the last 50 years, they have become increasing vocal against both nutrients and generic drugs. In fact, the over-the-counter availability of nutrients in higher-than-RDA levels in many countries of the world has been criminalized as part of the “globalization” movement. Much of what I talk about here as over-the-counter is actually not available without a prescription in many countries, many of which consider themselves progressive.

**Zinc Ionophores**

Chloroquine and hydroxychloroquine are zinc ionophores, meaning that they facilitate the bioavailability of zinc within cells. For this reason, the chloroquines are best administered with zinc to optimize this feature. One way that crony scientists have deliberately skewed their research designs is to use the chloroquines without zinc, analogous to the NIH studies falsifying high-dose vitamin C research by using low doses, and by giving vitamin C to chemotherapy and radiation patients, a protocol violation.

Chloroquine and hydroxychloroquine are used in treating malaria throughout the world, which is one reason that the market availability of these drugs could not be restricted by official actions, despite their attempts to do exactly that. The market is huge, and now projected to reach a half-billion dollars per year for a low-cost drug. But there are side effects when the dose of drug is pushed too high, which is another way to demonstrate that the chloroquines are too dangerous for widespread use. As it turns out, the zinc-ionophore feature of chloroquine and hydroxychloroquine is quite effective at low doses if administered early in the infectious process, so the only reason to push high doses is to induce unnecessary side effects and cause a study to fail. Hydroxychloroquine has a very long half-life and only has to be administered at a level of one tablet every three weeks.

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303 J Xue, A Moyer, B Peng et al. Chloroquine is a zinc ionophore. *PLoS ONE* 9(10): e109180, 1 Oct 2014. Abstract: “Chloroquine is an established antimalarial agent that has been recently tested in clinical trials for its anticancer activity. The favorable effect of chloroquine appears to be due to its ability to sensitize cancerous cells to chemotherapy, radiation therapy, and induce apoptosis. The present study investigated the interaction of zinc ions with chloroquine in a human ovarian cancer cell line (A2780). Chloroquine enhanced zinc uptake by A2780 cells in a concentration-dependent manner, as assayed using a fluorescent zinc probe. This enhancement was attenuated by TPEN, a high affinity metal-binding compound, indicating the specificity of the zinc uptake. Furthermore, addition of copper or iron ions had no effect on chloroquine-induced zinc uptake. Fluorescent microscopic examination of intracellular zinc distribution demonstrated that free zinc ions are more concentrated in the lysosomes after addition of chloroquine, which is consistent with previous reports showing that chloroquine inhibits lysosome function. The combination of chloroquine with zinc enhanced chloroquine's cytotoxicity and induced apoptosis in A2780 cells. Thus chloroquine is a zinc ionophore, a property that may contribute to chloroquine's anticancer activity.”

304 WHO “Solidarity” clinical trial for Covid-19 patients using high-dose hydroxychloroquine and two antiviral drugs. World Health Organization. Study was discontinued because of “no benefit,” but that was changed to “little or no reduction in mortality” in a press release. The study is reported to be in process of peer review.

305 http://www.francesoir.fr/politique-monde/oxford-recovery-et-solidarity-overdosage-two-clinical-trials-acts-considered “In the Recovery and Solidarity clinical trials, it is not excluded that patients died as a result of a therapeutic overdose of hydroxychloroquine (HCQ). Hydroxychloroquine continues to be the subject of many conversations. Brazilian researchers are now facing legal charges for overdosing HCQ, resulting in the deaths of 11 patients.”

306 See the Silicon Valley Health Institute interview with Professor Dolores Cahill, Ph.D. (https://www.youtube.com/watch?v=hlZCzqW6ywY&t=533s) (starting at 24:10).
Like vitamin D and zinc, chloroquines promote autophagy mechanisms,\textsuperscript{307} which are one of the cells innate immune responses to viral infections. As such, this feature is most effective when chloroquine treatment is begun earliest in the viral-infection process. So we have one more way to make a trial fail: start treatment late in the disease process.

Here’s a worldwide review of chloroquine and hydroxychloroquine studies in 2020 in Covid-19.\textsuperscript{308} They conclude that early treatment with hydroxychloroquine is effective, “while late treatment shows mixed results.”

The zinc-ionophore property of hydroxychloroquine may be shared by a variety of bioflavonoids and polyphenolic compounds. However, this has not been investigated systematically. Quercetin has been cited as a zinc ionophore,\textsuperscript{309,310} and it is being used clinically to treat Covid-19, but this has not been studied as well as hydroxychloroquine has for this particular mode of action.

There is another aspect to consider. The mechanism of action of chloroquines is likely divergent from quercetin and other zinc-chelating substances; neither chloroquine or hydroxychloroquine has a strong binding capacity for zinc. So we may be dealing with parallel phenomena.

An interesting historical aside; chloroquine drug development started with the clinical observations of the efficacy of methylene blue (see below, page 198) against malaria back in the 1890s. Prior to that time, natural quinine extracted from the bark of the cinchona tree was in widespread use. Those medical practitioners in tropical countries where malaria is endemic started making drug analogs that exploited structural features of both drugs and found hundreds of candidates worth consideration, dozens that were clinically tested and many that were found to be superior to both quinine and methylene blue.\textsuperscript{311}

It is also interesting that (1) the antiviral properties of chloroquine drugs were well known to the practitioners of the regions affected by malaria but were not considered noteworthy until 2020, and (2) even the researchers in those regions did not consider the zinc ionophore properties of chloroquines to be noteworthy of mention when discussing mechanisms of action.\textsuperscript{312}

The role of zinc mobilization and zinc bioavailability in innate immune defense is biologically intrinsic. Ignoring it as a mechanism of action is a scientific mistake with human-welfare consequences. I believe it

\textsuperscript{307} J P Liuzzi and C Yoo. Role of zinc in the regulation of autophagy during ethanol exposure in human hepatoma cells. \textit{Biological Trace Element research} 156: 350-56, 2013. “Zinc depletion caused a significant suppression of autophagy in cells. Conversely, zinc addition to medium stimulated autophagy in cells. Moreover, cotreatment with ethanol and excess zinc (40 μM) had an additive effect on the induction of autophagy. 3-Methyadenine treatment decreased labile zinc, but this effect was more pronounced in cells exposed to ethanol. Lastly, ethanol and 3-methyladenine caused significant changes in the expression of metallothionein and zinc transporters. The results from this study support the hypothesis that zinc is critical for autophagy under basal conditions and during ethanol exposure.” This is especially interesting in that the first metabolite of ethanol is acetaldehyde, a substantial inhibitor of glutathione. 3-methyl adenine is a known inhibitor of autophagy.

\textsuperscript{308} see c19study.com and hcqtrial.com.


is likely that future research will find that hydroxychloroquine improves zinc functionality at the cellular level and that this is a primary mechanism of action by which chloroquine drugs exert their beneficial effects for malaria, lupus erythematosus, rheumatoid arthritis, psoriatic arthritis, prostate cancer, porphyria cutanea tarda (PCT), ulcerative stomatitis, hepatic amoebic abscess, chronic urticaria (hives) and possibly a host of other skin and autoimmune conditions (dermatomyositis, sarcoidosis, Sjögren’s syndrome, granuloma annulare, erosive lichen planus, frontal fibrosing alopecia, chronic actinic dermatitis, Kikuchi–Fujimoto disease, graft-versus-host disease, chronic erythema nodosum, morphea and systemic sclerosis and pemphigus vulgaris).311

**Lysosomotropic Agents and Ivermectin**

One of the exciting breakthroughs of 2020 is the recognition that several efficacious pharmaceuticals for Covid-19 treatment belong to a class of drugs called lysosomotropic agents. This is claimed to subsume ivermectin, chloroquine and hydroxychloroquine. In actuality, this is not a new finding, scientifically. These drugs have been so well studied in areas of the world where malaria is endemic that they are chapters in old pharmacology textbooks published many decades ago.

Ivermectin is an anti-parasitical drug that is now showing high efficacy towards SARS-CoV-2 disease. Although medical resistance to its use in the USA is still extreme, several cases have become legal matters where medical refusal to use ivermectin has been overturned by court order resulting in rapid recovery of those medicated with ivermectin. These stories are exactly parallel to the vitamin C stories of people on death’s door or in comas on life support who recover rapidly when the forbidden treatment is administered. And in a parallel fashion, those doctors who have been over-ruled by the courts on behalf of the patient’s family continue to maintain that the treatment had no effect and the recovery was not related to the treatment. This is just one more way that beliefs prevent learning and progress.

The number of doctors using ivermectin is growing rapidly, to the point that there is now an informal “medical society” for which ivermectin is considered not only a standard of care but also a treatment of choice for all levels of Covid-19 severity.

Although there have been several bureaucratic attempts to legally restrict ivermectin availability by federal agencies and state governors, their efforts have been obstructed by the generic availability of veterinary ivermectin for treating parasitical infections in horses.

A similar state of affairs exists in the USA for hydroxychloroquine. Only five states have refrained from restricting hydroxychloroquine availability. Again, idiocy and politics are found hand in hand.

**Corticosteroids**

The ability of SARS, MERS and Covid to induce acute immune reactions (cytokine storms) would predict that immune suppressing and modulating therapies might prove efficacious. That has proven to be the case with corticosteroids.

It is, however, not clear whether this is in any way necessary in situations where vitamin C and glutathione are used in sufficient doses to restore redox control. One way or another, an out-of-control immune system needs to be shut down. Cortisosteroids can do this, and redox-buffering can do this. Given the polarity of opinions about this issue, further research will be needed to convince everybody that one way is superior to the other.
Multiple studies of Covid-19 therapeutics which have attempted to isolate different therapeutic agents from each other have found less-than-ideal results from corticosteroid use.

**Methylene Blue**

Dr. Deepak Golwalkar has gone public with his methylene-blue approach to Covid-19. One of his considerations is the extreme low cost of methylene blue for widespread distribution in countries that cannot afford expensive treatments. Methylene blue is easily and conveniently delivered in water or by nebulization.\(^{313}\) It is quick, cost effective and efficacious treatment for Covid-19 as far as he is concerned.

Dr Golwalkar is a pulmonologist (lung specialist) with 42 years of clinical experience in treating tuberculosis, pneumonias and other respiratory ailments. His experience as a pulmonologist has given him a unique perspective on the hypoxia-associated effects of Covid-19 infections. Methylene blue has been remarkably effective in relieving hypoxic stress, with nebulized methylene blue being particularly effective in “clearing alveolar block.”

He notes that the hypoxia of Covid-19 particularly resembles methemoglobinemia, a blood condition in which the iron in hemoglobin is in its oxidized state (ferric iron) instead of its reduced state (ferrous iron). Only ferrous iron in hemoglobin can carry oxygen. Methylene blue is considered an approved drug of choice for methemoglobinemia.

Without treatment, this hypoxia tends to induce a cytokine storm, where patient goes into irreversible respiratory distress.

The effect of methylene blue on reducing ferric iron in heme is part of its core biological functionality as a redox-buffering agent. Methylene blue has a very large, distributed aromatic orbital system which can (1) easily delocalize an oxidizing free radical, and (2) easily donate an electron to iron, vitamin C or glutathione.

Methylene blue is also a fast acting anti-fibrotic agent that mimics heparin and nattokinase.

Dr. Golwalkar concludes, “Methylene blue can be used as a treatment to all Coronavirus symptomatic patients and as a prophylactic drug to all vulnerable population. Used in low dosage (as prescribed below) it shows no significant side effects (less than 2% of patients complained of irritation in the nose/nausea for a day which subsided on its own). Given its low cost, it is also a viable regime for poorer countries like India. A number of patients (with varying levels of respiratory distress) have been treated using MB in nebulised/sub-lingual over the course of my years of practice.”

Methylene blue is normally handled and administered in its blue, oxidized form, but for optimal treatment of oxidative stress, the reduced form (colorless leucomethylene blue) may be preferred. Concomitant use of vitamin C as a reducing agent can convert blue methylene blue to clear in a few minutes.

In a preliminary study of five patients in an Iranian ICU, four responded positively to a combination of methylene blue, vitamin C and N-acetyl-L-cysteine (NAC).\(^{314}\)

\(^{313}\) Primary course of administration is nebulized form and sublingually together. For patients with severe cyanosis (oxygen saturation less than 85%), intravenous use may be required. The ideal concentration is 0.1% (1 gram of methylene blue powder dissolved in one liter of distilled water). If using the 1% concentration solution, dilute ten to one with distilled water.

These three agents act as a reducing cocktail, which counteracts the oxidant effects of an out-of-control immune system and its production of NO, nitrite, peroxynitrite and nitrate, all of which are oxidants and can convert ferrous heme to ferric heme and drive methemoglobin pathologies.

Methylene blue has been in clinical use for over 100 years in a wide variety of therapeutic applications. But because of its generic status, it gets little respect in Western medical circles. Yet its clinical use has persisted despite such prejudice. In a recent cell study, methylene blue was found to mitigate the adverse aging effects in progeria cells, in which children develop the symptoms of advanced age early in life.\(^{315}\)

Just like with vitamin C, there is a potentially serious complication for methylene blue use in those with glucose-6-phosphate dehydrogenase deficiency, where it can cause hemolytic anemia at lower doses than in everybody else. There is also a warning about potential serotonergic side effects in those taking serotonin reuptake inhibitor (SRI) drugs.

One of the arguments advanced for methylene-blue contraindication is that it can temporarily increase the challenge to NADPH (1) when it is administered in its blue, oxidized form and (2) when it is administered in high doses, before the reduced leucomethylene blue can act therapeutically on NADP+ and oxidized iron. This NADPH-depleted state is likely already present when symptoms of methemoglobinemia and RBC membrane damage are manifesting.

A 2010 review noted methylene blue applications to vasoplegia (catastrophic low blood pressure), septic shock, hepatopulmonary syndrome (low O2 saturations), malaria (especially chloroquine-resistant strains), ifosamide toxicity (a NADH-accumulation toxicity), tissue staining for aiding surgeries and imaging studies, neutralization of heparin overdosing, and possible treatment for Alzheimer’s disease.\(^{316}\)

### Antibiotics

(+) research metal-chelating and transition-metal interactions with azithromycin.

### Ebselen

Ebselen is a synthetic, small-molecule drug with interesting anti-viral effects and antioxidant effects.\(^{317}\) It is not yet FDA approved for anything, but it is being studied for treating viral diseases, *Clostridium* bacterial infections, stroke, bipolar disorders, osteoporosis, fungal infections, cardiovascular conditions, arthritis, atherosclerosis and cancer.\(^{318}\)

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\(^{318}\) G K Azad and R S Tomar. Ebselen, a promising antioxidant drug: mechanisms of action and targets of biological pathways. Mol Biol Rep 41(8): 4865-79, August 2014. From the abstract: “Ebselen, an organoselenium compound, mimics glutathione peroxidase activity. It is a multifunctional compound, which catalyzes several essential reactions for the protection of cellular components from oxidative and free radical damage. Based on a number of in vitro and in vivo studies, various mechanisms are proposed to understand the biomedial actions of ebselen in health and diseases. It modulates metallo-proteins, enzymatic cofactors, gene expression, epigenetics, antioxidant defenses and immune systems.”
Ebselen seems to interact strongly with cysteine residues, transferring their reducing power to reduction of hydrogen peroxide and organic peroxides. Ebselen shows strong antioxidant and reducing effects analogous to those of glutathione peroxidase. But whereas glutathione peroxidase is a large protein-based enzyme, ebselen is a very small molecule using unique selenium-sulfur redox chemistry similarly to the action of thioredoxin, which also alters the redox-buffering properties of sulfur systems by using selenium-sulfur analogs.

It remains to be seen whether a chemical with generalized redox-active chemistry will find a place in the pharmacological-oriented therapeutics industry, or in public-health agencies with a single-minded interest in new-chemical-entity pharmaceutical solutions at the expense of every generic therapeutic of utility.

Regarding Covid-19, scientists have modeled the binding of ebselen to M\textsubscript{pro}, the primary protease of SARS-CoV-2 that regulates its gene expression and replication.\textsuperscript{320} High throughput


\textsuperscript{320} C A Menendez, F Bylehn, G R Perez-Lemus et al. Molecular characterization of ebselen binding activity to SARS-CoV-2 main protease. Science Advances 14 August 2020. “We examine at a molecular level the potential of Ebselen to decrease Mpro’s activity. We find that it exhibits a distinct affinity for the catalytic region. More importantly, our results reveal a higher-affinity, previously unknown binding site localized between the II and III domains of the protein. A detailed strain analysis indicates that, on such a site, Ebselen exerts a pronounced allosteric effect that regulates catalytic site access through surface loop interactions, thereby inducing a reconfiguration of water hotspots. Taken together, these findings highlight the promise of Ebselen as a repurposed drug against SARS-CoV-2.”
screenings by other researchers have identified ebselen as “an especially promising” inhibitor of M\textsuperscript{pro} at concentrations 2-10 times lower than other candidates (see adjacent illustration).\textsuperscript{321}

The structures of these six M\textsuperscript{pro} inhibitors are displayed, with their “aromatic centers” outlined by gray dashed lines, along with the vitamin C redox pair.

**Shikonin**

The shikonin candidate for Mpro inhibition is a redox-active napthoquinone with some notable similarities to the structures of vitamin C (meta- and para-positioned hydroxy-ketone groups, and meta- and ortho-positioned hydroxy-ketone groups, respectively). It is being investigated for its antimicrobial effects on skin Staphylococcus infections and treatment of some cancers.\textsuperscript{322,323} It also has notable antithrombotic properties.

The cellular toxicity of ebselen is significant enough that it may need to be used in low doses and under very specific conditions. However, its ability to engage in sulfur-based redox chemistry makes it a unique therapeutic that may have application to oxidative stress, redox collapse and cytokine storms.

Among the candidate structures which passed the rapid throughput screening for Mpro inhibition, more than half feature sulfur-based chemical groups that would have redox activity.\textsuperscript{324}

**Disulfiram**

Disulfiram is a long-approved generic drug most well known as a treatment for alcoholism. But it also has been investigated for treatment of cancer and HIV infection.\textsuperscript{325}

Disulfiram prevents obesity in mice on high-fat diets. Insulin sensitivity was restored for both males and females. No data was collected on leptin.\textsuperscript{326}

\textsuperscript{321} Zhenming Jin \textit{et al.} Structure of M\textsuperscript{pro} from Sars-CoV-2 and discovery of its inhibitors. \textit{Nature} 582: 289-93, 2020. Abstract: “Coronaviruses infect humans and other animals and cause a variety of highly prevalent and severe diseases, including severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS). The SARS-CoV-2 genome comprises about 30,000 nucleotides: the replicase gene of SARS-CoV-2 encodes two overlapping polyproteins—pp1a and pp1ab—that are required for viral replication and transcription. The functional polypeptides are released from the polyproteins by extensive proteolytic processing, predominantly by the 33.8-kDa Mpro (also known as 3C-like protease). M\textsuperscript{pro} digests the polyprotein at at least 11 conserved sites, starting with the autolytic cleavage of this enzyme itself from pp1a and pp1ab8. The functional importance of M\textsuperscript{pro} in the viral life cycle, combined with the absence of closely related homologues in humans, identify M\textsuperscript{pro} as an attractive target for the design of antiviral drugs.”


\textsuperscript{324} The candidates are (1) ebselen, (2) disulfiram, (3) carmofur, (4) shikonin, (5) tideglusib, (6) PX-12 and (7) TDZD-8, the last of which was discarded as a candidate. Ebselen has an extended aromatic system with which the selenium atom can selectively interact through an aromatic nitrogen atom. Disulfiram has an aromatic disulfide linkage. Tideglusib has a sulfur-nitrogen linkage between two aromatic systems. And PX-12 is an assymetric disulfide with an aromatic system on only one side. Only shikonin and carmofur are not sulfur or selenium based, but both have extended aromatic systems that can delocalize free radical electrons.


Disulfiram is known to strongly interact with copper and zinc.\textsuperscript{326}

**Expectorants**

Bromhexine is an expectorant that increases fluid in mucous secretions. Similar to guaifenesin, bromhexine is a generic treatment for improving the productivity of coughing. A randomized pilot study in China involving only 18 patients suggested that bromhexine was clinically helpful, and a larger, randomized study of 78 patients in Iran reached statistical significance. The placebo group (39 patients) had eleven ICU admissions and the bromhexine group (also 39 patients) had two ICU admissions. A similar effect was seen for intubations (nine versus one), and death (five versus zero).\textsuperscript{327}

It is not clear at this time that the benefits are strictly related to improved coughing efficiency.

**SARS-CoV-2 Vaccines**

Vaccine side effects are not uncommon, but the vast majority of them are reasonably tolerable. Headaches, temporary fatigue, nausea, insomnia, itching, dizziness, brain fog, balance problems, aches and pains, etc. The most common non-lethal SARS-CoV-2 “vaccine” side effect (see next heading for the explanation) reported so far appears to be Bell’s palsy, a condition of acute neurotoxicity and significant facial paralysis that is often temporary. During the early stages of the Pfizer-BioNTech vaccine trials, three people developed Bell’s palsy. For the Moderna vaccine, it was four people. This appears to be a classic vaccine side effect.

The significance of Bell’s palsy side effects is being downplayed by both industry and public-health spokespeople as a temporary condition. However, some people who develop Bell’s palsy remain affected for the rest of their lives. And it is widely believed that Bell’s palsy is associated with later development of Parkinson’s disease, although the hard evidence for this is still statistically thin.\textsuperscript{328} This suggests the possibility that those experiencing Bell’s-palsy side effects might have a pre-Parkinson’s pre-existing condition or that the vaccine reaction may be increasing Parkinson’s disease risks.

There has been one well-publicized death 16 days following the vaccination of an obstetrician in Miami, who reportedly died of thrombocytopenia purpura, a condition characterized by immune destruction of blood platelets, and one previously linked to other vaccines. The report identifies the Pfizer BioNTech vaccine as the vaccination administered. Although Pfizer spokespersons deny that the vaccine was responsible, the attending physicians disagree. It is clinically clear in Dr. Michael’s case of thrombocytopenia purpura was autoimmune in nature, induced suddenly, and failed to respond to massive, repeated platelet infusions.


\textsuperscript{328} R Savica, J H Bower, D M Maraganore, et al. Bell’s palsy preceding Parkinson’s disease: a case-control study. Movement Disorders 24(10):1530-3, July 2009. doi: 10.1002/mds.22616. Six of the 196 patients with Parkinson’s disease had been diagnosed with Bell’s palsy years and decades earlier (2-54 years, mean time delay was 27.5 years). None of the age and sex-marched controls were diagnosed with Bell’s palsy. These results “are not statistically significant.”
The take-home point is that some degree of inflammation, oxidative stress and systemic toxicity are involved in SARS-CoV-2 vaccines, despite claims that these vaccines are fundamentally different than typical vaccines.

There are unconfirmed reports of up to 25 severe adverse reactions to Covid-19 vaccines. Given (1) the many millions of doses already administered, (2) the political and economic motives to deny adverse vaccination events, particularly for Covid-19, and (3) the sensationalism value for the media to over-report such events, I’m not surprised with such a figure, nor would I be if it was hundreds.

**The SARS-CoV-2 Neo-Vaccine Boomerang**

What is not being appreciated is that this new style of vaccine is such a radical departure from regular vaccines that it is not really proper to call it a vaccine. All historical vaccines have been comprised of an antigen and an adjuvant, the antigen being an exposed part of the infectious organism being targeted, and the adjuvant which challenges the redox-buffering system of the body to potentiate the immune system reaction to the antigen. This is the working definition of all vaccines prior to 2000.

But the mRNA “neo-vaccines” being distributed against Covid-19 do not contain the antigen that the body’s immune system can react to for the SARS-CoV-2 virus. These neo-vaccines contain the mRNA (messenger RNA) that “codes” for the antigen (stated to be “part of the spike protein”). While this is a very elegant concept, it is deeply flawed in a crucial way, and it has failed to work as intended in animal studies so far. This latter aspect makes this an exceedingly troublesome “medical experiment” on the world’s human population. The animals that have been given these mRNA neo-vaccines (cats and ferrets), when challenged later with a subsequent infectious agent, have all died. Rather, suddenly. This phenomenon has been described for the SARS-CoV-1-virus mRNA vaccines in cats and the MERS-virus mRNA vaccines in ferrets as a kind of focal immune enhancement. It is now called antibody-dependent enhancement. This induces an immune reaction which is so strong that is kills the animals in short order. It’s like a cytokine storm, but much more surgical, much more focused, and much more targeted.

The political rush to develop *any kind* of Covid-19 vaccines has purposefully bypassed the animal-test safety requirements for the neo-vaccines that are being distributed now (late 2020 and early 2021). It is possible that the developers of these neo-vaccines have solved the antibody-dependent-enhancement problem, but there is no sign of this in the emerging scientific literature, or in pre-prints of papers in the editorial and peer-review process.

The “flaw” that I mention above is that the mRNA causes the antigen to be produced inside of all the cells of the body. So the antigen is no longer restricted to the fluids of the body, as is the case for (1) historical vaccines, and (2) natural bacterial infections. These mRNA injections have every cell in the body making the antigen as if every cell in the body is infected with the virus. So the antigen to which the immune system is reacting is coming out of every cell in the body, which makes every cell in your body look like an infectious organism or infected tissue.

Another reason that this mRNA approach is flawed is that the risk enabled by the vaccine is not seen by vaccine safety surveillance currently being employed. In other words, the risk is not from the antigen sensitization itself, it is from the immune system’s antibody over-reaction at some future time. So it is, in essence, a “sleeping” side effect. I think of it as a new kind of “vaccine interference” that applies not to another virus or infection (an adverse cross reaction that is not intended), but to the infection that is actually being targeted (a spike-protein-containing viral infection of any kind).

These mRNA viruses have the potential to a bigger disaster than racemic thalidomide.
There is some good news. Theoretically, enhancement of the cellular immune system and induced autophagy could eliminate the mRNA making the spike-protein segment that these neo-vaccines induce and actually digest the spike-protein segments that have been produced. So this may be a future strategy for correcting the above flaws in these neo-vaccines if they blow up in our faces.

The correct functional-medicine approach is not to enhance humoral immunity with vaccines and neo-vaccines, which only get involved in fighting viral infections after the innate immune system has failed, but rather to augment innate immunity so that the humoral immune system never has to become critical.

**Colchicine**

Many countries are investigating older drugs for their anti-Covid-19 potential. One that has been reported to be quite successful is colchicine, a natural, plant-sourced, generic medication used to treat a wide range of inflammatory conditions. Structurally, it is closely related to bioflavonoids and polyphenolic antioxidants, which are named after “phenol,” a six-membered carbon ring with an alcohol sticking off the ring. The best polyphenolic antioxidants tend to have more than one alcohol groups, and more than one six-membered ring. Colchicine has a seven-membered ring, which gives it unique antioxidant properties.

Colchicine also has a nitrogen group, which is also not found on most polyphenolic antioxidants. This makes it a potential lysosomotropic agent, like chloroquine and hydroxychloroquine.

Colchicine is not commonly used due to NSAID popularity, but it is used in treating gout, Behçet’s disease (an autoimmune condition), heart inflammation, pulmonary fibrosis, and the prevention of atrial fibrillation after cardiac surgery. Research is currently underway for its use in treating atherosclerosis, coronary artery disease and Covid-19.

Canada is running the COLCORONA study, one of the largest studies of colchicine by mail, allowing people to participate from home by registration through the Internet with the colchicine being delivered to their registered address. The study results are not yet published, but news reports claim 21% less risk of hospitalizations or death from people taking colchicine at home. In confirmed Covid-19 patients, hospitalization was reduced 25%, ventilation was reduced 50% and death was reduced by 44%. Although the authors claim that colchicine is “the only effective oral medication for treating non-hospitalized patients,” this is not true at all. But these numbers move colchicine to the top of the approved medications list for Covid-19.

The COLCORONA protocol that should be adopted for testing natural antiviral generic substances on a wholesale level without the risks associated with hospitals and medical offices. It was described as a “contactless, randomized, double-blind, placebo-controlled clinical trial” where the participants entered the study at home—and stayed there if they did not need hospitalization. However, this study was partially funded by the Gates Foundation and Pharmascience, the company distributing colchicine. These funding sources would be unlikely to step forward for investigating generic nutrients that would be likely to be end runs around their financial and ideological incentives.

The GRECCO-19 study of colchicine cited by the above authors is not particularly helpful towards their case. Claims of survival are not for survival but rather for “event-free survival,” where the meaning of the

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event is not clarified.\textsuperscript{330} The primary endpoint is not actually survival, but rather two blood-test markers that tend to be associated with inflammation, which colchicine reduces. So these results could be entirely artifactual regarding alleged survival.

However, the results of an Italian study\textsuperscript{331} comparing 262 hospitalized patients, 122 of whom received colchicine plus the hospital’s standard-of-care regimen (lopinavir/ritonavir, dexamethasone or hydroxychloroquine) and 140 of whom received only the standard of care. The colchicine group had 84% survival, versus 64% in the controls.

This definitely deserves further study.


Appendix E: The Elitist Politics of Crony Science

I’ve been reporting fake science for 40 years. There’s some good news and bad news. The good news is that it does not appear to be getting worse from a volume perspective. The bad news is that it has become blatantly institutionalized. It’s hard for me to maintain a scientific perspective in the face of what appears to be gross malfeasance to undermine the general health and welfare of citizens. But when bad science becomes a “unified” basis of public policy, the risk of catastrophic results becomes unacceptable. How many lives lost from lack of vitamin C treatment for acute viral infections? It’s dozens of millions. Compare that to Covid-19 which is roughly one million.

As an AIDS activist in the 80s and 90s, I witnessed Robert Gallo’s malfeasance regarding HIV science, and Anthony Fauci’s selective non-funding of AIDS research that was not aligned with his personal agenda at the expense of science that would, ultimately, expose AIDS as an immune disorder precipitated by conditions beyond the “mere” presence of HIV infection.

As a Down’s syndrome activist of the 90s, there was no such institutional resistance against use of dietary supplements to minimize adverse health consequences from trisomy21 and prevent the majority of mental retardation of infants born with Down’s syndrome. One physician was disciplined for doing a retrospective study of his private practice without IRB approval, but that can be attributed to simplicity of mind. To lend credence to such a hypothesis, one volunteer scientist working on the consensus team for a commercial supplement was a full-time FDA researcher.

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332 A good example from long ago was the gross manipulation of the market by the FDA, which falsified a study of the Swiss-owned artificial sweetener cyclamate, in anti-competitive preparation for the pending approval of Monsanto’s aspartame. The FDA banned cyclamate in the USA because it was a “suspected carcinogen” based on an FDA study of the combination of cyclamate and saccharine. Saccharine, a known carcinogen, would have caused the cancer by itself. Nevertheless, this was sufficient “evidence” of risk to the American consumer for their administrative ban of cyclamate. When criticized, their bureaucratic argument was that there was insufficient evidence to prove that cyclamate was not a carcinogen. If this was not criminal enough, Monsanto arranged for one of their executives to become temporary head of the FDA, who then over-ruled the committee charged with aspartame’s approval, who had recommended against its approval based on carcinogenic data! As it turned out, there were twelve cases of cancer in the aspartame-rat group and none in the control group, which was statistically significant based on the small rodent population used. But, mysteriously, the study data disappeared for a period of time, which when it was fortuitously found somewhere, listed eleven cancers in the aspartame group and one in the control group. Since this was deemed not statistically significant, aspartame’s approval was not considered bureaucratically unsound. For all the details of this episode of crony capitalism, read the two-part series “Aspartame” by Mark Gold, Ward Dean and myself in Smart Drug News, 4(1): (https://drive.google.com/file/d/0B22v0N-O32jUY3NDVW/pvbZU3Rza/view) and 4(2): (https://drive.google.com/file/d/0B22v0N-O32jUODJIZEkzeldkY1E/view) including the following Senate testimony by John W. Olney, “There were other problematic aspects of the brain tumor data. In the pre-1975 records that I reviewed, it was clear that several competent pathologists had carefully examined the original microscopic slides from the first study and agreed that there were 12 brain tumors in the NutraSweet-fed rats and zero brain tumors in the controls. When the FDA conducted a task force investigation of these laboratories in 1975, they singled out those studies for further investigation and ordered that all laboratory records, including microscopic slides et cetera, be impounded under FDA seal. Several years later, when a group of pathologists (UAREP) was sent to authenticate these studies, they could not find the microscopic slides. The UAREP pathologists were finally taken to a laboratory where the slides were not supposed to be and there they found some but not all of the original slides. Clearly, they had not been kept under FDA seal, and by mysterious coincidence the slides that were finally presented to the UAREP pathologists contained evidence for 11 brain tumors in the NutraSweet-fed rats and one tumor in the controls. It is important to recognize that if there were zero in the controls, it is very difficult to argue that the tumor incidence in the control and NutraSweet-fed rats is the same. But if there is one tumor in the control rats, it is possible with statistical acrobatics to reach the conclusion that the incidence is the same. And, indeed, this is exactly the argument that the manufacturer and the FDA bureau of Foods pressed at the Public Board of Inquiry. ...Even more seriously, I wonder why FDA allows microscopic slides to disappear (while supposedly impounded) and why they do not question the de novo emergence of a brain tumor among the controls when the slides reappear.”
So what explains the divergent institutional responses? It might be as simple as public relations. The FDA can abuse HIV infected citizens because they can be castigated as gay and/or intravenous-drug users. Whereas “motherhood and apple pie” is something the FDA will not risk touching with a ten-foot pole.

But the conspiracy view is that there is no money at stake with the treatment of children and adults with Down’s syndrome. But anti-HIV drugs marketed to prevent AIDS is a trillion-dollar industry affecting the bottom-line profit margins of the pharmaceutical industry. This makes just as much sense to me seeing Fauci’s unfortunate research-funding decisions that have resulted in millions of people on expensive (and toxic) drugs that might otherwise be on inexpensive (and safe) dietary supplements.

As unfortunate as that might be for those who have died with AIDS, those HIV+ people still living still have the option to integrate natural anti-viral self-defense into their pharmaceutical program. This kind of end-run around institutional malfeasance is still open to us, despite current plans to “militarize” both vaccine testing and vaccinations.

**Viral Science versus Viral Politics**

One of the hardest things to do in a time of crisis is to maintain a big-picture vision while being bombarded with propaganda and scare tactics. According to the propaganda, the SARS-CoV-2 virus is an unprecedented virus for which we have dire need of a vaccine. But is that true?

The average human is exposed to tens of thousands of new viruses during their lifetime. Viral novelty is the norm rather than the exception.

When a population of over 8000 normal healthy humans (free of any viral diagnosis) were blood tested for 19 human viruses by analysis for viral DNA and RNA sequences unique to those 19 viruses, 42% were positive for one or more of those viruses. The truth is that we live in an ocean of viruses. If we had the capability of testing for more than those 19 documented viruses, the results would be dramatically higher. Adapting to new viruses is part of being alive. We do it all the time, and our pets and livestock do it all the time, too. We’re just not doing a good job of adapting to viruses because of pre-existing conditions caused by neglect, and we are panicking because the death rate is scarier than that from influenza viruses, which we accept without any panic whatsoever.

Do we need a vaccine? Hardly. We have never needed a vaccine before. For coronavirus or for influenza. Flu vaccines are openly promoted, but do not work well.

During the SARS coronavirus epidemic of 2002, there was a similar panic. There was no vaccine then, and SARS was gone by the middle of 2003 due to herd immunity (and the seasonal transition from winter to summer). A large effort to make a vaccine failed.

During the seasonally atypical MERS coronavirus epidemic of 2012, it was basically gone by 2013. Both of these coronavirus outbreaks have transitioned from epidemic to seasonally recurring viral outbreaks just like the flu. Attempts to make MERS vaccines have consistently failed, with some attempts actually boomeranging, like flu vaccines causing increased risks for adverse Covid-19 outcomes.

Creating an expectation that this coronavirus outbreak is different is the epitome of media pandering. And it remains to be seen whether the new (and unprecedented) quarantine policies will make the problem

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[333] https://journals.plos.org/plospathogens/article?id=10.1371/journal.ppat.1006292
worse rather than better. We’ll know this by the end of 2020, when the flu season hits, and herd immunity has been prevented by official policy.

Moving on. Let us also consider the US government’s official dietary recommendations, set by the National Food and Nutrition Board “expert committee” of academics, industry representatives and bureaucrats as an example of crony science. They have, for decades, recommended consumption of high carbohydrate diets rich in whole grains. While this may be the backbone of the American farming and processed-food industries, this diet directly causes obesity, insulin resistance and diabetes, and promotes the development of cardiovascular disease and cancer. The scientific evidence contradicting this unfortunate advice is literally overwhelming, yet the 2020 expert committee was able to discount it all by excluding “weight-loss studies” from the evidence they had to consider, and concluding that they “couldn’t find a single study with carbohydrates below 25% of calories.”

The Low-Carb Action Network, a group of citizens, published a list of 52 such studies. Let’s look at a few.


**Findings:**
- (a) weight loss was due to deficit in energy intake.
- (b) 24-hour glucose normalized.
- (c) HbA1c, insulin sensitivity, triglycerides and total cholesterol improved.

*These are all pre-existing conditions for increased Covid-19 mortality.*


**Findings:**
- (a) in two weeks, dietary intake dropped to what was appropriate to their height.
- (b) weight loss was completely accounted for by reduced caloric intake.
- (b) much improved 24hr blood glucose profiles, insulin sensitivity and HbA1c.
- (d) decreased triglyceride and total cholesterol levels.

*Again, broad decrease in Covid-19 risk factors.*


**Findings:**
- (a) low-fat meals elevated glucose and exaggerated the insulin response.
- (b) low-carb meals gave higher metabolic rate in the late post-prandial period.

*Low metabolic rate is a risk factor for adverse Covid-19 outcomes.*

Study 4. L R Saslow, A E Mason, S Kim. An online intervention comparing a very low-carbohydrate ketogenic diet (VLCK) and lifestyle recommendations versus a plate method diet in overweight individuals with type 2 diabetes: a randomized controlled trial.

Jessica Wharton. 52 low-carb studies the US Dietary Advisory Committee says it can’t find. [lowcarbaction.org/low-carb-studies], 31 March 2020. Dishonest (“rigged”) exclusion criteria included, (1) lack of specific descriptions of foods eaten, and (2) all studies focusing on weight loss. When assessing food-composition aspects of health and wellbeing, the specific foods eaten are not important. As long as they are low carb, the scientific conclusion regarding carbohydrate impact on health and wellbeing is valid. The exclusion of weight-loss studies is blatant bias. The stated mission of the Board includes counteracting obesity and insulin resistance. Weight loss IS exactly the result that anti-obesity research would be finding in a successful dietary protocol. This is a perfect example of how crony science operates.

**Findings:**
(a) type-II diabetics improved glycemic control and lost more weight on a VLCK diet compared to a low-fat American Diabetes Association (ADA) diet.
(b) online programs: dropout rate 1 of 12 (VLCK) and 6 of 13 (ADA).
(c) improved mood, vitality and depression for VLCK diet.
(d) some measures worsened on ADA low-fat diet.


**Findings:**
(a) carb-restriction diets improve clinical risk factors for metabolic syndrome better than low-fat, calorie-restricted diets.
(b) carbohydrate restriction targets a range of risks with a single intervention.
(c) low-carb diets are a strategy for general health beyond weight regulation.
(d) ApoB:ApoA ratio worsened on low-fat diet.
(e) low-carb diet decreased leptin resistance more than low-fat diet.
(f) total saturated fatty acids reduced 57% on low-carb vs 24% in low-fat.
(g) low-carb increased size of LDL and reduced small and very small LDL particles.

The effect of crony science on adults is a matter of choice. You can choose to ignore the American Diabetes Association’s crony science and malfeasance. The crony academics and corporate scientists of the US Food and Nutrition Board cannot enforce their advice on you, either. However, the effect on our children is not a matter of choice. The school-lunch programs operate under the guidance and recommendations of the US Food and Nutrition Board. Their bad judgment is putting children, adults and seniors at risk.

And we wonder why autism is on the rise, and why so many kids are dying from Covid-19 infections.

The fox is administering the henhouse.


**Findings:**
(a) doubling of saturated fat intake did not increase plasma saturated fatty acids in any lipid fraction.
(b) dietary saturated fat is efficiently metabolized in the presence of a low-carb diet.


**Findings:**
(a) subjects adapted to a very low carbohydrate diet showed no evidence of vascular dysfunction in response to a high-fat meal.
(b) flow-mediated dilation improved on the carb-restricted diet from 5.1% to 6.5%.
(c) on the low-fat diet, flow-mediated dilation declined from 7.9% to 5.2%.
(d) a 12-week low-carb diet improves postprandial vascular function more than a low-fat diet in individuals with atherogenic dyslipidemia.

*This paper contradicts the widely held belief by the medical profession that dietary fat sabotages endothelial function. Quite the reverse.*

**Study 8.** C E Forsythe, S D Phinney, M L Fernandez, *et al.* Comparison of low-fat and low-carbohydrate diets on circulating fatty acid composition and markers of inflammation.

**Findings:**
(a) The very low carbohydrate (ketogenic) diet resulted in reduced inflammation.
(b) Ketogenic diet increased arachidonate (20:4n-6), while inflammatory biosynthetic metabolic intermediates of arachidonate were decreased.
(c) The ketogenic diet decreases in cytokines (TNF-alpha, IL-6, IL-8).
(d) The ketogenic diet decreased MCP-1, E-selectin, I-CAM, and PAI-1.
(e) Ketosis lowered saturated fatty acids, including palmitoleic monounsaturate.

This paper illustrates that low-fat diets do not enhance redox-buffering capacity and that low-carb diets inducing ketosis do restore redox control.

This is a good place to stop and assess the effects of the US Food and Nutrition Board on the general health of US residents and the susceptibility of people to Covid-19 infection. I hope this minimalist presentation of studies indicates just how damaging the Board’s recommendations for high-carb diets has been towards the health and welfare of its citizens over the last half-century, and in the present time frame, how this malfeasance has set the stage for increased Covid-19 morbidity and mortality. This continuing dismissal of low-carb diets is perpetuating the underlying risk factors that are described as pre-existing conditions for poor Covid-19 outcome, both of the blatant, visible kind (diabetes and heart disease) and the invisible, iceberg-below-the-surface kind (insulin resistance, lack of beta-oxidation and ketosis ability, shallow antioxidative defensiveness, and inability to recycle the key elements of the redox-buffering pool under oxidative load).

These are some of the same people who tell us that vitamin C is only needed in 60 mg/day quantities.

**More Crony Science for Vitamin C**

University of Helsinki researcher Harri Hemilä writes, “A large number of placebo-controlled studies have shown that vitamin C supplementation alleviates the symptoms of the common cold, but widespread skepticism that vitamin C could have any significant effect remains. One of the most influential common cold studies, published in 1975, was carried out by Thomas Karlowski, et al., at the National Institutes of...

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335 The abstract: “Abnormal distribution of plasma fatty acids and increased inflammation are prominent features of metabolic syndrome. We tested whether these components of metabolic syndrome, like dyslipidemia and glyceremia, are responsive to carbohydrate restriction. Overweight men and women with atherogenic dyslipidemia consumed ad libitum diets very low in carbohydrate (VLCKD) (1504 kcal:%CHO:fat:protein = 12:59:28) or low in fat (LFD) (1478 kcal:%CHO:fat:protein = 56:24:20) for 12 weeks. In comparison to the LFD, the VLCKD resulted in an increased proportion of serum total n-6 PUFA, mainly attributed to a marked increase in arachidonate (20:4n-6), while its biosynthetic metabolic intermediates were decreased. The n-6/n-3 and arachidonic/eicosapentaenoic acid ratio also increased sharply. Total saturated fatty acids and 16:1n-7 were consistently decreased following the VLCKD. Both diets significantly decreased the concentration of several serum inflammatory markers, but there was an overall greater anti-inflammatory effect associated with the VLCKD, as evidenced by greater decreases in TNF-a, IL-6, IL-8, MCP-1, E-selectin, I-CAM, and PAI-1. Increased 20:4n-6 and the ratios of 20:4n-6/20:5n-3 and n-6/n-3 are commonly viewed as pro-inflammatory, but unexpectedly were consistently inversely associated with responses in inflammatory proteins. In summary, a very low carbohydrate diet resulted in profound alterations in fatty acid composition and reduced inflammation compared to a low fat diet.”

336 This is increase in arachidonic acid without a corresponding increase in gamma-linolenic acid (GLA) or eicosapentaenoic acid (EPA) is routinely considered to be a marker of increased inflammation. But it is clear here that this is not the case and that it is actually reversed from expectation. Arachidonic acid proportionately increased, but its oxidative metabolites decreased markedly. This means that the low-carb, pro-ketosis diet was shutting down inflammation by an underlying mechanism (redox-buffering) which is an earlier step in the inflammatory cascade. Because of improved redox control, arachidonic acid was not being oxidatively diverted into prostaglandin pathways, and cytokine regulation improved dramatically. This is one more way in which the lack of understanding of redox mechanisms puts humans at risk.
Health. Their placebo consisted of lactose, which can easily be distinguished from ascorbic acid by taste. Karlowski, et al. found a 17% decrease in the duration of cold episodes in the group administered vitamin C (6 g/day); however, they suggested that the decrease was entirely due to the placebo effect.\(^{337}\)

This is a perfect example of how inuendo and a bad study design can be used to color the “reporting” of a finding unacceptable to the powers that be (the National Institutes of Health, in this case). By botching the placebo, the positive results could then be blamed on a placebo effect, which could then be extended by inuendo to each and every other study that also found that vitamin C decreased the duration and symptoms of colds, none of whom botched their placebos. The influence that this crony-science publication had in deflecting then-significant attention on vitamin C among young professionals was profound. It was conducted by the NIH, used NIH personnel for subjects, was published in the prestigious New England Journal of Medicine, and was well publicized in the media.

The reality was that this study verified and validated previous studies which used proper placebos. Yet the mere suggestion that this study’s flaw was an explanation for false findings in those previous studies was accepted without any scientific evidence whatsoever.

Other signs of cronyism are also evident. First is the participation of Thomas Chalmers in this study, who built his professional reputation on discrediting vitamin C, and who wrote a concurrent review in the American Journal of Medicine (1995) expounding on the guilt by association of previous studies. And finally, this “finding” for vitamin C was used as an example to discredit other vitamin C studies in a textbook (Principles and Practice of Infections Disease, 1995). This is a perfect example of cronyism in action, (1) falsify a finding, (2) publicize that finding, (3) embed that finding in your peer group, (4) repeat the finding in other contexts (medical school), and finally (5) review that finding as exemplary.

A nearly identical scientific fraud was perpetrated on the scientific community by the Parkinson Disease Research Group of the United Kingdom. They had botched their study design by administering deprenyl (Eldepryl) without lowering the dose of Sinemet (L-dopa plus carbidopa). Plus, they had the bad fortune of using imported Eldepryl from eastern Europe that was contaminated. Because of these two mistakes, they had 60% increased mortality among the Eldepryl plus dopa group. But rather than report this honestly and risk civil lawsuits, they blamed the increase deaths on deprenyl, despite the fact that six of six previous studies solidly concluded that deprenyl increased survival and delayed the progression of Parkinson’s disease. Their explanation of a delayed toxicity effect of short duration was unprecedented in the history of toxicology, then and now, yet because the group was prestigious, the researchers prestigious and the British Medical Journal prestigious, they got away with it.\(^{338}\) To this day, deprenyl (selegiline) is not popular in the USA despite it being the best drug available for the treatment of Parkinson’s disease. And none of the families of the victims of contaminated Eldepryl were able to sue within the statute of limitations.

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Official Mayo on Your Baloney Sandwich?

“Chances are you’ve heard about a food, drug or other method that claims to prevent, treat or cure coronavirus disease 2019 (COVID-19). But while it might be tempting to use a questionable product or method to stay healthy during the pandemic, it’s extremely unlikely to work and might cause serious harm.”

As you have read for more than a hundred pages, all of this is “extremely unlikely” to work. They go on to say...

“Many people take vitamin C, vitamin D, zinc, green tea or echinacea to boost their immune systems. While these supplements might affect your immune function, research hasn't shown that they can prevent you from getting sick. The supplement colloidal silver, which has been marketed as a COVID-19 treatment, isn't considered safe or effective for treating any disease.”

While I have my doubts that colloidal silver has any direct antiviral effect, I do not doubt for a second that it is amazingly effective against bacteria and fungi. To the degree that people have chronic infections other than viral, colloidal silver could easily have clinical benefits beyond the capacity of Mayo clinicians to comprehend.

Indeed, most of the nutritional and metabolic factors discussed in these pages are well correlated with keeping you from getting as sick. Half as sick. A quarter as sick. A tenth as sick.

Even their statement on use of antibiotics is ill informed.

“Antibiotics kill bacteria, not viruses. However, people hospitalized due to COVID-19 might be given antibiotics because they also have developed a bacterial infection.”

Actually, antibiotics have a long history of efficacious accidental use in treating viral diseases. This has long been a puzzle to medical academics because “antibiotics only kill bacteria.” This type of all-or-nothing thinking pervades the medical community without any evidentiary basis. There are mechanisms beyond what is known, and as Revici showed almost 100 years ago, most antibiotics have catabolic-aerobic-acidifying activity, which is indeed antiviral in clinical reality. Accidental antibiotic use in treating viral infections has shown significant clinical efficacy, despite their ignorance of the mechanism.

Similar ignorance is involved in official public-health policy.

**NEJM with Mud on their Faces**

The supposedly peer-reviewed and prestigious *New England Journal of Medicine* got caught twice in the anti-Trump scientific fraud campaign against hydroxychloroquine. Both *NEJM* and *Lancet* were forced to withdraw two papers where allegedly fictitious data was used to question the safety of hydroxychloroquine and chloroquine.

Now a second paper published by *NEJM* is now under question, but rather than withdraw the paper, they went through a long correction which was as incomprehensible as the questionable conclusions of the
It seems impossible that the dozen authors of the paper could have been oblivious of such error and misinformation. Perhaps the NEJM editors and staff played a role in this scientific blunder and that’s why they will not withdraw it?

Anyway, it’s likely worse than the editors of NEJM admit. 341

340 Hydroxychloroquine with or without Azithromycin in Mild-to-Moderate Covid-19 (Original Article, N Engl J Med 2020. DOI: 10.1056/NEJMoa2019014). The report as published did not provide accurate and complete information on the frequency and duration of previous use of hydroxychloroquine or azithromycin among the trial participants. In the Participants subsection of Methods (page 2), the first sentence should have begun, “We enrolled patients who were either actively screened by the trial team or referred to us who were 18 years of age or older and had been hospitalized …,” rather than “The trial included consecutive patients who were 18 years of age or older and who had been hospitalized ….” In the second sentence, the phrase “previous use of chloroquine, hydroxychloroquine, azithromycin, or any other macrolide for more than 24 hours before enrollment (and since the onset of symptoms)” should have been omitted. In the third sentence, the phrase “, including criteria regarding previous use of hydroxychloroquine or azithromycin,” should have followed the term “exclusion criteria.” In the second paragraph of the Randomization, Interventions, and Follow-up subsection of Methods (page 2), the sentence beginning “The administration of hydroxychloroquine or chloroquine” should have been omitted. In the final footnote below Table 1 (page 4), the phrase “during the 24-hour period” should have been omitted, and the footnote should have ended, “Details are provided in the Supplementary Appendix.” In the Statistical Analysis subsection of Methods, in the final sentence of the paragraph beginning “We also performed …” (page 5), the phrase “during the randomized treatment period” should have been added after “medications received.” In the first paragraph of the Characteristics of the Patients subsection of Results (page 8), “randomization on May 17, 2020” should have been “… on May 18, 2020.” At the end of that subsection, “Tables S5 and S6, respectively” should have been “Tables S5 through S7.” In the Primary Outcome subsection of Results (page 8), the mentions of Tables S7, S8, and S9, should have been Tables S8, S9, and S10, respectively. The final sentence of that subsection should have ended with “(Table S11) or in three post hoc subgroups defined according to the date of trial enrollment or according to previous use of hydroxychloroquine or azithromycin (Table S12),” rather than “(Table S10).” In the second paragraph of the Secondary Outcomes subsection of Results (page 8), the mentions of Table S11 and Table S12 should have been Table S13 and Table S14. In the final sentence of the Safety subsection of Results (page 9), the mention of Tables S13 and S14 should have been Tables S15 and S16. In the first sentence of the first footnote below Table 3 (page 11), the expression “during the randomized treatment period” should have been added after “according to the medications received.” In the penultimate paragraph of the Discussion (page 11), the sentence beginning, “The enrollment of patients with no previous use of these medications was challenging …” should have been replaced by, “We did not specify in our protocol the exclusion of such patients until late in the course of the trial, and as a consequence, 9.3% of the trial participants had previous use of hydroxychloroquine and 36.1% had previous use of azithromycin. However, in most instances, the duration of previous use was only 24 to 48 hours before enrollment, primarily because, before May 13, we required that patients be enrolled in the trial within 48 hours after hospital admission and because outpatient use of these drugs (before admission) was infrequent. After May 13, we specified that use of these drugs for more than 24 hours was an exclusion criterion.” The Supplementary Appendix was also affected. The article is correct and the Supplementary Appendix has been replaced at NEJM.org.

341 The online report: “I’ve probably looked back at the Cavalcanti study more than any other. Why? I predicted from the moment I saw it that it was a fraud, but it was not nearly as obvious as the Surgisphere fraud. Recently, the research team admitted that a lot of the patients in the study were previously on HCQ or ATH, and that's obviously a huge point of controversy probably worse dismissing the study over, but I want to finally talk about some of the other things wrong with the study, some of which have been pointed out and some of which haven't. It gets worse. Nearly 10% of the control group actually used HCQ *during* the trial on a daily basis, recorded in the patient data. How are these people considered a control?!!

Next (and others have pointed this out around the internet), a lot of the patients are clearly not “mild or medium” cases of Covid-19 as the student title claims, and the moment you see a title purport to be something that it isn't, you should look for
First, some of the control group patients who were not supposed to be taking any hydroxychloroquine were taking hydroxychloroquine for the duration of the study, as evidenced by the patient records? A control group that is not actually a control group? This got through peer review?

Second, the study claims to be a study of mild-to-moderate Covid-19 patients, yet 14% were recruited from the intensive care unit of the hospital? And NEJM did not notice?

Third, control patients were reported to be taking “quinolone” as an “additional” medicine. There is no such medicine. There are several categories of drugs referred to as quinolone antibiotics or fluoroquinolone antibiotics. But which of these was or was not involved in the study is not made clear in the slightest. This happens to be a bid deal because some quinolones may act like chloroquine and hydroxychloroquine regarding zinc ionophore properties, and other quinolones are known to be copper chelators that induce catastrophic copper deficiencies and ligament and tendon damage. Zinc is deeply tied to Covid-19 disease, and copper is well known to be antagonistic to zinc bioavailability. So the lack of clarity in this lack of disclosure calls their entire conclusion into question.

NEJM should be deeply ashamed of their participation in this scientific travesty.

The Mis-Regulation of Medicine

Crony science in the US Food and Nutrition Board is not the worst of it. Medical misregulation likely affects many more people in a much more life-and-death manner. The #1 cause of human mortality in the United States is not heart disease and cancer. It’s iatrogensis. Iatrogenesis means doctor-caused deaths. The death-by-mistake data is so blatant that official sources allow that it is the third-ranked cause of death, which is damning enough. But what they will not admit is that there is a huge under-reporting problem with iatrogenic deaths. Neither doctors nor hospitals want to admit their mistakes, especially when somebody dies. Those who have made serious efforts to assess under-reporting come to the conclusion that iatrogenesis kills more people than heart disease or cancer. And because some of these people admitting to the magnitude of the under-reporting problem are themselves embedded in the crony-science machine, they are likely under-estimating the degree of under-estimating going on.

How could the practice of medicine be that far off base?

There are many reasons that could be presented, with ample data to justify them. But what I’d like to do is draw your attention to the rotting foundation of medicine: the standard of care doctrine. This is the legal rationale for the way medicine is regulated, and why bad medicine persists. This doctrine says that “good” medicine is defined by what most doctors do in practice (the “standard”), and “bad” medicine is defined as deviation from that standard of care. It’s not hard to see that any new-and-improved medical technique starts out as a deviation from the standard of care. So there is no legal difference between bad medicine

suspect information. In the 41 page supplement, we see that 13.8% of patients were recruited from the ICU. Huh. And then they weren't even given medicine immediately! And these are probably NOT the patients previously taking HCQ or ATH as previously mentioned because the apologetic update in the NEJM talks about patients taking these drugs outside of the hospital. So, it is likely that the strict majority of patients in the control group were either ICU patients or *also* treated with the medications tested.

It gets even worse. I hadn't noticed this before probably because I hadn't fully cataloged all the drugs of the cinchoa tree when I first read the Cavalcanti paper, but almost 10% of the patients in the study were given quinolone, which is, like HCQ, derived from quinine and has the isomeric (symmetric) chemical structure believed to make it a successful zinc ionophore. Quinolone is itself the subject of COVID-19 trials in some countries.

And this study got published in the NEJM.”
and really great medicine. And just as bad, any bad medicine that is mainstream is legally good medicine, even if it kills and maims people, or costs a thousand times as much as an equivalently effective treatment.

What this standard-of-care doctrine does is (1) legally sanction the suppression of medical innovation that challenges the crony system, and (2) legally protect bad medicine from any liability litigation. Some of the examples here are the tip of the iceberg regarding crony-suppressed treatments. Here is a link to a TED talk by Sharyl Attkison on how special interests coopt media and Wikipedia, with their consent, giving them free reign to disparage more effective and less costly treatments while promoting bad-medicine practices.

★★★★★  https://www.youtube.com/watch?v=-bYAQ-ZZtEU

Investigative journalist Sharyl Attkison tells the story of how new drugs are promoted by pharmaceutical companies in mainstream media and social media. TEDx. She also exposes the corrupt practices of Wikipedia editors.

What we get is mediocre medicine. At a time when scientific knowledge is advancing faster than it ever has in the past. The gap between medical discoveries and medical practice is expanding at an unprecedented rate, which has caused the United States to be ranked #1 in worldwide medical care expense and 17th in health results. According to the Commonwealth Fund, the US ranks last among the highly industrialized countries (Australia, Canada, Germany, the Netherlands, New Zealand and the United Kingdom). The Netherlands spends $3837 per capita for first place, while the US spends $7,290 per capita for last place.

Cronyism is expensive.

So we get bad medicine as a matter of fact. If you are smart and listen to a low-carb guru that says cutting carbs will help your diabetes or heart disease, and your doctor measures your triglycerides in the first two or three weeks of your new diet, you will be scolded, demeaned, and told in unequivocal terms that this is going to kill you. Utter rubbish. But because such horrendous advice is the standard of care, the doctor never learns. You have no recourse if you do not know any better and are intimidated into relying on such deadly advice.

However, if you are lucky enough to know better, and you schedule your next physical and CBC blood test 6-10 weeks after starting the diet, your triglycerides will be substantially lower than they were on the carb-based diet. There is even a faint chance that you will be praised instead of denigrated. Your risk of dying from cardiovascular disease, neurodegenerative diseases, cancer and Covid-19 are decreased. But it was because you circumvented mainstream medical advice, not because you followed it.

The Deep State

Along with crony science is crony capitalism,342 crony media, crony academia and crony nGOs. They work together. This is how the oligarchy works.

If somebody in academia starts to ask questions about something that is against the crony position, they are gently persuaded to stop, pointing out that their careers (and family livelihoods) depend on following the

342 Crony capitalism is the term for the economic system used by socialists. But crony socialism is equally apt. It all comes down to your definition of our economic system, whether you consider it a perversion of socialism (crony capitalism) or a perversion of capitalism (crony socialism). Like beauty, this is entirely in the mind of the perceiver.
popular lines of research. And if that does not work, they are threatened with loss of funding, bureaucratic retaliation, bad performance reviews, social ostracism, termination, and character assassination.

In today’s media institutions, investigative reporters are routinely instructed as to what stories not to cover.

**Oligarchy in the USA**

The term for this kind of government is oligarchy. Rule by “the few.” Students in the United States may be taught by crony teachers and the US media propaganda proclaims that we live in a democracy, but it just is not so. There may be a thousand messages proclaiming democracy as the design principle of our governmental system, but in reality we were set up as a republic. But that republican organizational system has lost the battle with the forces of corruption and we now live in an oligarchy, with a popularized fiction that we are “a democracy.” And it is the crony influence in science, bureaucracy, education and media that empower this decidedly non-republican, non-democratic system.

Rule by the few.

And the nice thing about this new system is that we do not know who the few are. That’s the innovation behind the power of cronyism; influence flows down through the chain of command free of the constraints of titles and ranks, so only the bosses know who their bosses are.

Anonymity is a perfect strategy for unaccountability.

**Oligarchy in the World Health Organization**

Rather than itemize all the political corruptions operating in the WHO, let me single out their focused support of the pharmaceutical industry and vaccine-oriented public-health policy. Less than one year after the SARS-CoV-2 epidemic became a pandemic, the WHO has coopted “herd immunity” as a vaccine-only term.

The actual definition of herd immunity is the level of immunity in a population that shuts down transmission of the disease through the population. This transmission is herd member to herd member (person to person), so in the early stages of an epidemic, there are more than enough contagious herd members to spread the disease to others not yet infected. But as the numbers of infected grow, the number of herd members not yet infected diminish. This slows down the spread of the disease. And at a particular point, the spread drops to zero. That is the point at which “herd immunity” has been reached—for that particular disease.

Herd immunity varies with the disease. Some diseases are difficult to spread and herd immunity might be reached with 60-80% of the herd having immunity by past infection. Other diseases might require 85-95% of the herd to have been infected. But the definition is not variable; it applies to the herd population and the dynamics of the infection process taking place.

With the advent of vaccination technology, the definition of herd immunity was modified to include vaccination. This certainly makes sense. Resistance to infection can be natural (from infection itself) or technological (from vaccination). Since herd immunity is all about the transmission vector within a herd, vaccination is equivalently effective at diminishing transmission, so the WHO definition prior to Covid-19 was:
“Herd immunity is the indirect protection from an infectious disease that happens when a population is immune either through vaccination or immunity developed through previous infection.”

And late in 2020, the definition is now:

“Herd immunity, also known as population immunity, is a concept used for vaccination, in which a population can be protected from a certain virus if a threshold of vaccination is reached.”

They go on to add that:

“Herd immunity is achieved by protecting people from a virus, not by exposing them to it.”

This is Propaganda 101, the manipulation of language to establish a political position that is not supportable by the language itself. Despite their protestations, the entire history of viral vaccines is exposing people to viruses to achieve herd immunity. So what they are saying is not only a corruption of the English language, it is false to their own case.

Their political position is that vaccination is the only “populational” defense against SARS-CoV-2.343 Such an assertion is blatantly false to fact. The truth is that the vast majority of herd immunity has been established by near asymptomatic Covid-19 cases. We also know with certainty that supplementation of vitamin D drastically decreases Covid-19 severity at every level of symptomatology, even in people with well known risk factors (seniors). The fact that mortality is decreased by half, hospitalization is decreased by half and near asymptomatic cases are increased by more than a factor of three among 87-year-old seniors indicates that vaccination is absolutely not the only way to achieve herd immunity. And this remarkable efficacy was achieved by a very low dose of vitamin D costing less than a half dollar.

If the vitamin D’s therapeutic efficacy scales with dose (it does prophylactically) and if other innate immunity therapeutics are added for their multi-fold synergies, the functional medicine alternative to vaccination will prove to be better than the hoped-for vaccine. It’s better in efficacy (resistance to ALL viruses, not just SARS-CoV-2). It’s better in cost ($2-8 versus $35). It’s better in availability (now, versus later). It’s better in infrastructure (no refrigeration or freezing needed). And it’s better in mitigating a broad range of pre-existing conditions, all of which are ignored by the vaccine.

And the WHO wants to dismiss all of this, in favor their vaccine-only agenda and the profits of their cronies.

**Covid-19 and Gain-of-Function Research**

Although initially denied, overwhelming evidence has established that the US government funded gain-of-function research into coronaviruses for many years. Congress, the media and the Governmental Accountability Office have confirmed the details, and that monies were spent after the Congressional ban. Anthony Fauci was a key advocate for that research, which was argued to be necessary for understanding future pandemic and biowarfare risks for American citizens.

One of the most highly censored topics on the Internet today is the argument that the SARS-CoV-2 virus was not a natural virus that crossed over from bats and pangolins, that it was an accidental (or deliberate?) release from a Chinese coronavirus research laboratory in Wuhan, China (or some other military

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343 The WHO definition also specifies that the “protection” must not expose people to SARS-CoV-2, even though the new-generation of Covid-19 vaccines do exactly that.
Laboratory). Although many mainstream researchers have published papers in support of the natural-emergence theory, their arguments have been feeble at best and not backed up with any data that could be tested, verified or falsified.

As I read each of these papers making the case for natural emergence, I was overcome by disappointment. Although I am not a virologist, I understand enough chemistry to know how DNA and RNA operate to make proteins and how genes and proteins change over time due to mutations and cross-over events. But none of these papers had the slightest content about the fine structure of natural coronavirus RNA and its comparison to the fine structure of SARS-CoV-2 RNA. Not one. I wondered how these researchers could be so incompetent—or lazy.

It was not that they did not talk about genetics. It’s that they did so by gross analysis (e.g., 93% homology between SARS-CoV-2 and horseshoe-bad coronavirus). That’s it. This is like comparing two books based on their titles without reading them. Utter disappointment.

What I wanted was a side-by-side comparison of the natural viral RNA sequences with the SARS-CoV-2 RNA sequence with discussion of how the differences would arise by mutation and cross-over events. Simple. To the point. Meaningful content relating to the actual titles of these papers.


Finally, I saw one. And it was damning. It caused me to shift my position from a natural-emergence-is-likely hypothesis as best guess, to an accidental-release-is-likely from a gain-of-function laboratory. By this time (June), the natural crossover theories involving bat species, pangolins and the Wuhan wet market were all scientifically falsified, and officially abandoned by Chinese scientists, who I assumed to have the highest incentive to misrepresent the possible negligence and carelessness of accidental release.

But, oddly, the natural emergence idea was staunchly defended everywhere else, and especially in the west in the Unites States and the United Kingdom, all the way from sleazy news reports to supposedly prestigious scientific institutions. It did not seem possible to me that anti-Trump sentiment (his trashing of China) was so high as to corrupt the integrity of academic, professional and journalistic institutions.

At the time, it made sense to me that the US complicity in the gain-of-function research that directly led to the accidental release was a realistic motive for cover-up. After all, the US NIH had samples of all the super-coronaviruses they had engineered, and they could easily know that the SARS-CoV-2 RNA sequence was (or was not) their creation. Even if it was released accidentally by careless bio-security, if the source was a US laboratory strain, the US would be partly blamed for the crime by the media and public alike.

After the publication of the RNA sequences in a side-by-side array, better and more methodological arguments for a laboratory source for SARS-CoV-2 have been made, while the volume of official denials has risen exponentially, the official counter-strategy has advanced from (1) financial support of denial publications to (2) overt censorship, and (3) outright character assassination.

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344 H Zhou, X Chen, T Hu et al. A novel bat coronavirus reveals natural insertions at the S1/S2 cleavage site of the spike protein and a possible recombinant origin of HCoV-19. bioRxiv preprint. doi: 10.1101/2020.03.02.974139. The same evidence, but an unsupported presumption of natural recombination which is used to argue the case for natural recombination. Even the homology differences of the highly conserved spike protein (61%) with two other “similar” bat coronaviruses (93% and 96%) was “evidence” of natural recombination. In later publications, the 96% homologous virus was questioned as being natural and was not found in the claimed animal species from which it was reported to be extracted. Nevertheless, this paper is a perfect example of crony science: how to get published by arguing for the sun-revolves-around-the-earth theory.
In September, a very thorough analysis and argument has been independently published by a team led by Li-Meng Yan, a Chinese-defector viral scientist who escaped to London while others in China disappeared.\textsuperscript{345}

Not only is this newer analysis exceptionally detailed and well reasoned, it is falsifiable in multiple ways. So far, none of the critics have actually falsified any of the arguments detailed in the paper, merely continuing the older arguments made by previous apologists that the idea is preposterous and citing viral-homologies data out of context.

One of the most amazing arguments advanced in the paper is a detailed proposal for replicating how the SARS-CoV-2 virus was made in a laboratory. While this may not be the way it was actually made, it is a step-by-step proposal for how it could be made in a normally equipped viral gain-of-function laboratory setting. It is definitively falsifiable. But was more interesting to me was that it explained several odd parts of the SARS-CoV-2 virus as remnants of genetic splicing methods in current use.

Contrary to the detractor’s allegations, the paper makes a good case that each of the cross-over theories advanced to explain the natural emergence of Covid-19 are unlikely in the extreme. As an example, the parts of the virus that are so similar to a naturally occurring bat virus are only selective parts of the virus. Those other parts of the virus that are utterly different from that “natural source” species of bat virus would have to have come from another natural coronavirus that could simultaneously infect the same animal.

This is the scientific/biological basis for the cross-over phenomenon, where two viruses exchange pieces with each other. For this to happen both viruses need to be replicating in the same animal, in the same cell, at the same time. The problem with the bat-virus natural cross-over theory is that the novel spike-protein RNA sequence is in a virus that cannot infect pangolins, the proposed candidate for cross-over.

Oops!

Consider a hypothetical analogy: It would be like claiming that this particular paragraph is not plagiarism when it includes a discussion of the virus to be or not to be, that is the question: we are dealing with the assessment of the fine structure of the SARS-CoV-2 virus.

So, what do you think? The sentence structure kind of makes sense. But the phrase “to be or not to be, that is the question:” is a quote from Shakespeare (Hamlet). And not only is it intact, which suggests plagiarism, but the colon between “question” and “we” is conspicuously wrong for the sentence but is part of the original quotation. It’s a “smoking gun” piece of evidence.

To insert that phrase into a paragraph and claim that it is mine and not Shakespeare’s is plagiarism. For a scientist to say that a bat virus naturally picked up the SARS-CoV-2 spike sequence from a virus that does not infect that bat species or any pangolin is scientific sophistry. Those that do not know any better

\textsuperscript{345} Li-Meng Yan, Shu Kang, Jie Guan and Shanchang Hu. Unusual features of the SARS-CoV-2 genome suggesting sophisticated laboratory modification rather than natural evolution and delineation of its probable synthetic route. https://zenodo.org/record/4028830#.X2VZ0JNKhqe “The Covid-19 pandemic caused by the novel coronavirus SARS-CoV-2 has led to over 910,000 deaths worldwide and unprecedented decimation of the global economy. Despite its tremendous impact, the origin of SARS-CoV-2 has remained mysterious and controversial. The natural origin theory, although widely accepted, lacks substantial support. The alternative theory that the virus may have come from a research laboratory is, however, strictly censored on peer-reviewed scientific journals. Nonetheless, SARS-CoV-2 shows biological characteristics that are inconsistent with a naturally occurring, zoonotic virus. In this report, we describe the genomic, structural, medical, and literature evidence, which, when considered together, strongly contradicts the natural origin theory. The evidence shows that SARS-CoV-2 should be a laboratory product created by using bat coronaviruses ZC45 and/or ZXC21 as a template and/or backbone.”
(reporters) might be easily fooled, but respectable scientists are embarrassed when such improbabilities are mentioned outside of a stand-up comedy routine.

While RNA cross-over insertions do occur in nature, they occur in specific ways that can be named and characterized. I’ve already mentioned that they have to occur in an animal that can be simultaneously infected by both viruses.

But they also require the presence of two natural viruses.

How do we know whether a virus is natural? We collect it from an animal in the wild, and it is comparable to different strains of that virus already collected, (1) from that species, (2) from closely related species, and (3) from other, genetically non-related species occupying the same ecosystem, which could infect each other. This is why a few dogs and cats have been found to carry SARS-CoV-2 caught from their Covid-19 humans.

Or zookeepers.

There are hundreds of coronaviruses that have been collected and sequenced, and the rates of mutation of different parts of these viruses have been studied carefully. And published.

In other words, viral mutations take place in different parts of the viral genome at different rates with predictable frequencies. In other words, some parts of the virus can experience a high mutation rate without adversely affecting the viral virulence. This has been the case for the 8+ SARS-CoV-2 variants that I have seen published so far; the mutations are not having a clinically significant effect. The list of variants is now much longer, and few show any significant clinical effect.346

And other parts of the natural coronaviruses are critical to virulence and cannot tolerate mutations. These parts are known. The mutation rates for these parts have been studied extensively and are similar across the entire range of coronavirus strains. The ORF1a and ORF1b parts of the SARS-CoV-2 virus are tolerant of mutations and the spike protein part is not tolerant of mutation. Yet the SARS-CoV2 virus has few mutations in the ORF1a and ORF1b parts and extensive mutation in the spike protein part. This is entirely backward from anything natural.

But it is exactly what you would see in a gain-of-function laboratory setting. Just like the “pointy end” is the dangerous part of the spear, the spike-protein is the “dangerous” part of the SARS-CoV2 virus. For gain-of-function study, the focus must be on the potential changes to the spike protein. There is little reason to expend serious effort on the non-essential parts of the viral genome.

If this were not damning enough, there are two special regions in the spike protein that are even more genetically altered. One of these deals with the receptor affinity of the spike protein to human ACE-2 receptors, arguably the primary route of human infection of SARS-CoV-2. This is the genetic sequence that codes for the amino acids in direct contact with the ACE-2 receptor surface. These amino acids are singly altered, which in a natural environment would be an extended series of point mutation event and not a cross-over event. This would never happen naturally without extensive point mutations elsewhere in the virus. And that is clearly and unequivocally not the case.

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346 At least one strain is claimed to be much more transmissible, which has many people panicking. However, the SARS-Cov-2 primary strain is already so spectacularly transmissible for humans that the increase is like raising the speed limit on a viral freeway from 65 miles per hour to 70. This has little practical effect and no effect on clinical factors.
Single-point *spike* mutations are less frequent in natural viruses, and here they are not merely more frequent, they are an order of magnitude more frequent. To argue that this could be natural is specious.

The other special region deals with the “activation” of the *spike* protein after successful binding to ACE-2, to split the S1 and S2 subunits of the *spike* protein so the virus can merge with the cell membrane and infect the cell with viral RNA. In SARS-CoV-2, and in no other natural beta-coronavirus, there are four additional amino acids (12 different nucleotides) inserted between S1 and S2 which happen to be splittable by *furin*, one of the most common peptidase enzymes found in *human* cells.

This is, again, a feature totally divorced from nature yet perfectly aligned with gain-of-function research.

Two highly modified features, both specific to humans.

And both in a highly conserved region for natural beta-coronaviruses.

This is either a bizarre set of coincidences that are unprecedented in the entire multi-billion-year history of biology, or it is a straightforward restructuring of bat beta-coronavirus RNA to study potential risks to humans.

This paper makes the case that SARS-CoV-2 is a laboratory-made virus. The evidence and arguments are sound, and the evidence and arguments are falsifiable. So it is now up to the opponents of this hypothesis to actually falsify it.

It remains to be seen whether the opponents will rise above character assassination and censorship to actually deal with the evidence and arguments scientifically. This is a new publication, built upon a previous finding published only a few months ago, so there has not been much time to mount a meaningful challenge. But I think it is likely to the point of certainty that the current political environment of censorship and retaliation is unlikely to change. These scientific insights into the SARS-CoV-2 genome could easily remain a conspiracy theory for some time to come. So why should any scientist risk their reputations in responding meaningfully?

I can see why they would choose not to engage.

The powers that be do not want to be held responsible for the biggest health and economic disaster of recent history. The programs ostensibly begun to protect Americans from risk caused the precise scenario that they were charged with preventing. Personally, I think they are right to hide this. I very much doubt that the American public or world-wide community will forgive such a blunder despite the widespread ethical and religious sentiments that good intentions are a moral justification for unintended consequences.

Too many people will assume that the gain-of-function research was a euphemism for biological warfare research and that the multiple stated claims to protect the public were merely a spin-doctor, public-relations campaign to legitimize illegal bio-warfare research.

So what’s happening?

Twitter has suspended Li-Meng Yan’s account. She had 60,000 followers before Twitter acted on behalf of the cronies.

*Natural Geographic* has responded with a blatant, utterly unsupported statement that “the coronavirus wasn’t made in a lab,” making it all out to be a conspiracy against science and public truth.

This is the Catholic Church defense tactic for the earth-is-the-center-of-the-universe dogma.
Vox published an article by Alex Ward making claims that the article was backed by conservative Trump supporter Steve Bannon, and citing multiple prejudicial statements by academics as inuendo, calling the analysis “fact challenged” and casting aspersions on Li-Meng Yan’s character for writing a paper “so bad” that her “once-sterling” credentials are now irreversibly stained.

Character assassination 101, conducted by Democrat cronies who decry the identical tactic by Republicans. Inuendo by academics: “similar coronaviruses have been found in labs as early as 2013.” (italics in the original). No such evidence exists. And the furin feature, claimed by Chinese military researchers to be found in nature, was unable to be found in the reported animal by non-military scientists.

Columbia’s Dr. Angela Rasmussen has taken a front-line position in the media storm was quoted as saying that the three pieces of evidence Yan offers are “easily disproven.” But has not done so. Instead, she has engaged in specious statements that pander to media mentality, sidestepping the specifics of the arguments. “These are found in other coronaviruses” is offered as evidence for falsification, when it is nothing of the kind. There is no way to tell if such high-school level errors being made by a Ph.D. scientist are real errors made by careless over-simplifications of the complexities of science, or errors created by incompetent reporting. But, hopefully, Dr. Rasmussen will actually back up her media strategy with an actual scientific publication that does not replicate the regrettable lack of quality of previous lab-debunking efforts. So far (January 2021), she has failed to back up even one of her assertions, but she is repeating them frequently as if they were true. She is now suddenly getting published in the top journals (Cell, Nature and Nature Medicine). Does this look like cronism?

There is only one statement that was reported that could be evidence against Yan’s case, that the furin cleavage site that Yan says is not found in any natural beta-coronavirus does occur in the 2012 MERS beta-coronavirus. It will be interesting to see if (1) the future comparison of the MERS virus RNA shows the same RNA-editing and differential-mutation signs of laboratory creation, or (2) any possible natural-crossover mechanism of how the MERS sequence could have crossed-over into the SARS-CoV-2 genome as an alternative to having it spliced there purposefully in a gain-of-function laboratory. It is obvious that the furin-cleavage feature would be of critical interest to any gain-of-function research program. So Dr. Rasmussen’s argument, advanced to disprove Yan’s case, may turn out to be further evidence for a laboratory genesis for SARS-CoV-2.

It is not possible that MERS virologists would have missed the furin-cleavage locus during the five years of MERS research between the 2012-2013 initial MERS epidemic and the 2017-2018 latest possible time frame for the laboratory creation of the SARS-CoV-2 virome. It would have been one of the first things they would have noticed, such a conspicuous feature that is not found on any other natural beta-coronavirus. That would be comparable to a teen not noticing a humongous zit on their face while looking into a mirror. In any case, the scientific side-by-side analysis that Yan and colleagues used to identify the fingerprints of laboratory manipulation of SARS-CoV-2 can easily be applied to MERS to see if it is likely or unlikely to be a natural virus. Either way, the other spike modifications remain compelling evidence of gain-of-function genetic meddling.

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347 If you are a serious scientist looking for irony, please read Dr. Rasmussen’s latest publication in Nature Medicine on the natural evolution of SARS-CoV-2. It’s an impressive political diatribe totally lacking in any scientific content but with numerous appeals to authority and repeated character assassination of people who are presenting highly detailed scientific data and cogent interpretations of its meaning that have undermined her public position. It is now quite obvious to me that she is not just unwilling to address the issues scientifically, that she is also incapable of doing so. doi: 10.1038/s41591-020-01205-5. She is the poster child for scientific cronism.
I look forward to appending this chapter in future releases.

The Role of Crony Science in Politics

What has amazed me the most about the current SARS-CoV-2 epidemic is the chilling of scientific opinion regarding political process. For example, for the first time in the history of civilization, we have coherent social engineering taking place that is supposedly justified by science, but is actually not based on science at all. And the public believes that it is scientific because crony scientists proclaim it as scientific.

The other side of the story, which is always a necessary and essential aspect of scientific process, is not is not being heard because of censorship, intimidation and retaliation directed against anybody not towing the party line. Most of those scientists criticizing the six-foot social distancing rule and the wearing of masks rule have been censured by their institutions, or censored by their Facebook, YouTube or Twitter service providers.

The Six-Foot Social-Distancing Rule

There is no clear evidence that 6 feet of separation is in any way protective. Nobody has ever gone out and studied the relationship between how far apart people are and their infection risk. And any simple understanding of air-flow patterns makes it all meaningless. The “contact tracing” studies that have been done of audiences in buildings with central-air systems show high infection of those people downwind at distances way above six feet and near zero infection of people upwind at far less than six feet.

Duh. Respiratory viruses like colds and flus are “shed” in the lungs, mouth, throat and sinuses and carried on the air in tiny aerosol droplets. So they go where the air goes. So how did a male-bovine-soil-additive rule like six-feet-of-separation become public policy? The kind answer is: we needed some kind of protection policy and this is “better than nothing.” But as most wise people know, the we-have-to-do-something mentality usually backfires. In fact, the doing-nothing strategy often leads to a meaningful solution that is hidden by doing something unproductive. Socially, it looks and feels like doing something is a step forward, but that’s just an appearance of functionality, which is often entirely dysfunctional.

The not-so-kind answer is that the bureaucratic cronies actually wanted to impose a form of social conformity that would make the non-conformers stand out. This is a path to intolerance that has caused many societies to fracture and become divisive, usually leading to massive social injustice against a minority at the hands of the majority. This puts the cronies in the driver’s seat for such injustices. By creating something to fear, people are encouraged to act out their fears against their neighbors. During prohibition, it was boozers and alcoholics. In Nazi Germany, it was the Jews.

Singling out the Jews in Nazi Germany and in US universities, singling out the black former slaves in the southern United States, singling out whites in the black-lives-matter campaign; these manifest in many ways from the subtle to the blatant. But it is the oligarchs that benefit from the distraction of social divisiveness. It appears that we need them more than ever.
So, how far can SARS-CoV-2-containing aerosol droplets actually disperse into the air with the virus remaining infectiousness? One answer is over ten meters for indoor air. In other words, SARS-CoV-2 virus aerosols are being found in hospital air that was nowhere near any infected person.

Nowhere is this more obvious as a politically motivated tactic than in the mask-wearing policy.

**The Pros and Cons of Wearing Masks**

Do masks actually help prevent the spread of diseases? Most Democrats think so and most educated Republicans agree. But is there evidence to support this? If you go looking for it, you are likely to be disappointed.

While it is true that Covid-19 disease is significantly lower in some countries where mask-wearing is an aspect of popular culture, most of the people wearing masks are motivated by social politeness; it’s an act of courtesy towards others. It’s like saying, “I’m trying not to infect you with my germs.” But does it work?

Some pretty well-designed studies say otherwise. So maybe the country-wide differences in statistics are artifacts of other factors that have nothing to do with wearing masks, *per se*. And maybe there are other consequences of wearing masks that are being ignored by only looking at one aspect of disease management?

For example, one group of researchers studied masks using salt aerosols, figuring that the particle size was viral like. They found that many N95 respirators and all “surgical” masks did not perform in conformity with OSHA regulations. Given that these masks represent the medical standard for such masks, with both fitting and one-time use, the use of masks by the general population, which are home-made, unfitted and used repeatedly, would be dramatically less effective. They conclude, “N95 filtering facepiece respirators may not achieve the expected protection level against bacteria and viruses.” They even point out that the “exhalation valve” on some N95 respirators “does not affect the respiratory protection” of the

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348 L Setti, F Passarini, G De Gennaro et al. Airborne transmission routes of Covid-19: Why 2 meters / 6 feet of inter-personal distance could not be enough. Int J Environ Res Public Health 17(8): 2932, April 2020. “Based on the available knowledge and epidemiological observations, it is plausible that small particles containing the virus may diffuse in indoor environments covering distances up to 10 m from the emission sources, thus representing a kind of aerosol transmission. On-field studies carried out inside Wuhan Hospitals showed the presence of SARS-COV-2 RNA in air samples collected in the hospitals and also in the surroundings, leading to the conclusion that the airborne route has to be considered an important pathway for viral diffusion. Similar findings are reported in analyses concerning air samples collected at the Nebraska University Hospital. On March 16th, we have released a Position Paper emphasizing the airborne route as a possible additional factor for interpreting the anomalous COVID-19 outbreaks in northern Italy, ranked as one of the most polluted areas in Europe and characterized by high particulate matter (PM) concentrations. The available information on the SARS-COV-2 spreading supports the hypothesis of airborne diffusion of infected droplets from person to person at a distance greater than two meters (6 feet). The inter-personal distance of 2 m can be reasonably considered as an effective protection only if everybody wears face masks in daily life activities.”

349 S A Lee, S A Drinshpun, and T Reponen. Respiratory performance offered by N95 respirators and surgical masks: human subject evaluation with NaCl aerosol representing bacterial and viral particle size range. Annals of Occupational Hygiene 52: 177-185, 2008. Results: “About 29% of N95 respirators and approximately 100% of surgical masks had PFs <10, which is the assigned PF [protection factor] designated for this type of respirator by the OSHA. On average, the PFs of N95 respirators were 8-12 times greater than those of surgical masks. The minimum PFs were observed in the size range of 0.04-0.2 mum. No significant difference in PF results was found between N95 respirators with and without an exhalation valve.” Conclusions: “The study indicates that N95 filtering facepiece respirators may not achieve the expected protection level against bacteria and viruses.”
mask wearer. However, the exhalation valve offers zero protection to anybody in the vicinity of the mask wearer, who is exhaling unfiltered breath.

The conclusion of Setti, et al.\(^ {350} \) is that “the inter-personal distance of 2 m [meters] can be reasonably considered as an effective protection only if everybody wears face masks in daily life activities” [emphasis added]. Without such stringent compliance, “there is reasonable evidence” that “small particles containing the virus may diffuse in indoor environments covering distances of 10 m from the emission source.”

Indeed, one hospital found surfaces testing positive for SARS-CoV-2 fifty meters away from their Covid-19 ward. They did NOT test to see if the samples were actually infective.

One can only assume that mask policy is about social conformity. Even if we go beyond the requirement for scientific data and presume that masks do limit risk of spreading SARS-CoV-2 either way (for the wearer, or from the wearer), is it not better to be safe than sorry? This might be defensible if we did not take into account the health costs of wearing masks—hypercapnia, for example. This, also has not been scientifically assessed for the general population. So it is possible that the possible benefit of some is paid for by the known cost to others, who are at known risk of hypercapnia (impaired ability to exhale waste carbon dioxide).

So six feet of separation is a number pulled out of a hat. So how can an imaginary number become law?

Stupidity meets policy.

How about the compulsory wearing of masks? It would seem to make sense that masks prevent cross-infection of respiratory infections, but is there any evidence that it is so? According to the published studies, the evidence is weak at best. Even studies ignoring the bad-fit problem of masks, assuming that 100% of each exhale and inhale goes through the mask fabric show that N95 masks are unable to stop coronaviruses, influenza viruses and rhinoviruses from passing through the fabric. The fabric is able to stop larger droplets containing viruses in fluids temporarily, but a coating of virus-containing particles builds up on both the inner and outer surfaces of the fabric and renders them ineffective for re-use. Lesser masks and cloth-fabric masks are hugely less efficient.

There are some propaganda campaigns for masks that show some countries with high mask use have much lower death rates than countries that do not wear masks as part of their culture, but in the USA, states and counties that do or do not wear masks do not have differential death rates. Some areas where mask use is minimal have much lower death rates than other areas where mask use is mandatory.

Why are these areas different? Politics. Democrats versus Republicans? Liberals versus conservatives? Population density? Air pollution? There is no science being applied. It’s strictly a political decision that claims scientific justification on both sides of the aisle. Wearing a mask means you are an idiot democrat in some areas. An in other areas, not wearing a mask means that “you are killing people.” (That’s intended literally and not figuratively.) There is science that establishes that mask-wearing causes hypercapnia (toxic levels of CO\(_2\) and acidity in the blood), which is worse in older people than younger people (documented) and worse in hypometabolic people (my own observations).\(^ {351} \)

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\(^ {351} \) If you want to test this in yourself, measure how long you can hold your breath, before putting on the mask, and after wearing it for 30 minutes. And then two hours. For real-life context, test your breath-hold time any time you can take off your mask. Human breath-hold time is inversely proportional to how much CO\(_2\) is found in your blood. Long breath hold means low CO\(_2\) and short breath hold means high CO\(_2\).
So maybe wearing masks is also “killing people,” just in a different way. Or at the very least, giving them headaches, risk of stroke, brain fog, anxiety, edginess, stress and panic attacks.

Ambient outside air has CO₂ levels between 250 and 350 ppm (parts per million). Indoor air can vary from 350 to 1200 ppm depending on how well the airspace is sealed (closed doors and windows tend to make it the higher range, and open windows [and leaky construction] make it towards the lower number). The exhale breath is about 35,000 to 50,000 ppm CO₂. With a normal breathing pattern, only the residual air in the lungs, throat, mouth and sinuses retain that 35-50,000 PPM of CO₂, which is inhaled at the start of the next breath. With a mask, that volume of CO₂-laden air can be up to twice as high, depending on the shape of the mask and the volume of air it traps. This means more CO₂ recycled back into the lungs.

Since breathing is based on CO₂ levels and not oxygen levels, the breathing rate differs in those wearing masks. This has been scientifically measured in mask-wearing professions and it is a health risk that is now well documented. It is especially problematic in surgeons conducting extended surgeries requiring exceptional manual-dexterity skills. And it is more of a problem in older mask wearers.

https://www.youtube.com/watch?v=p6h2JBr179o

Dr. Frank Shallenberger making the case against mask wearing as a matter of personal and public-health policy. Several citations to the primary literature are provided.

**Crony Mask Science 2021**

There is now legislation pending in Congress to make mask wearing mandatory. Given the democratic Presidential victory, the past alignment of Joseph Biden to public-health officials and the pharmaceutical industry (vaccines AND drugs), there is popular expectation that this legislation will pass, as it has elsewhere. To offer support of this legislation, advocates cite a fraudulent study published by *Nature Medicine* in October which claims that increasing mask wearing from 49% to 95% will save an estimated 130,000 lives by February of 2021.

It is exceedingly odd that *Nature Medicine*’s peer review for this paper was conducted in just seven days and that they did not notice that mask wearing in the USA had already peaked at 80% back in July, the time when they allegedly collected their data and four months before the publication of that data, cited as current.

So this is not just incompetent peer review, it is bad science. How could the authors (the Institute for Health Metrics Evaluation, at the University of Washington) make such an egregious mistake, and how could the editors and peer reviewers of *Nature Medicine* not notice such a mistake? It’s an academic glitch.
that a graduate student would not be allowed to make. Yet here is *Nature Medicine* refusing to withdraw the paper, The Editors stating that bad data correctly cited is not a matter for peer review, and both President-elect Biden and Anthony Fauci citing the study as clear evidence for a national mask mandate.

Take a look at the fraud. The red and blue lines represent ongoing data being collected by YouGov surveys of American and Canadian citizens, respectively. Look at how many data points are being collected. Now notice the IHME data collected (in magenta), where it was collected (May and June) and where it was reported as current data (late in September). And finally, look at other sources of mask-wearing behaviors of Americans (California and Pennsylvania data from Carnegie Mellon University, in orange), the CDC data (in green), and the Brookings Institute (80%, not shown). The IHME data is utterly divergent, falsely reporting lower values in support of the idea that mask wearing is lower than it really is.

After this fraud was exposed by several groups and publicized by Retraction Watch, the estimate by the IHME team was revised. However, that revision was only to their model and not based on any new data. The correction was not published, nor was the *Nature Medicine* paper corrected.

As a point of scientific clarity, this misleadingness was pre-arranged by the IHME team who asked the question, “who wears masks in public at all times?” This political slant on the question excludes people who wear masks in public where other people are present but do not wear masks, for example, while driving alone in a vehicle. Or take off their masks while walking on a street alone, or walking with a member of their shelter-in-place household. So they can hide behind a scientific technicality that can then be purposefully misreported by reporters (a thousand reports in this case, at least) or deliberately misunderstood by lazy politicians and misrepresented by politicians making speeches. However, such discretionary mask wearing has nothing to do with public risks. So excluding those who wear masks responsibly is entirely misleading for assessing genuine risks OR predicting lives lost by non-mask wearing that does not actually cross-infect anybody. So the pretense of science can be maintained even if the facts are false.

**Crony Science Coopts Evidence-Based Medicine**

One of the crony science campaigns that has gone horribly wrong is the “evidence-based medicine” campaign. There is certainly nothing wrong with evidence being used in conjunction with the evolving practice of medicine, but that’s the English-language meaning of the phrase and not the meaning of the campaigners.

Somehow, the only legitimate evidence that is considered “evidence” is double-blind placebo studies. No other evidence need apply. Given that this kind of evidence is routinely invalidated by subsequent studies (see extensive research of John Ioannides, a world-class expert in evidence-based medicine) and most medical progress has been based on observational studies in clinical settings, this has to be one of the more idiotic campaigns in modern history.

Given the above context, there is no rational basis for such prejudices regarding evidence. All evidence should be evaluated on its own merits.

Follow the evidence advice suggested by Stanford Professor John Ioannidis.
Cost Differentials

One way to see cronyism and oligarchy in action is to follow the money. Since money drives politics and commerce alike, it shows when the slanted playing field causes low-cost generic therapies to be ignored in favor of expensive proprietary therapies.

For the services industries, we see low-cost preventive therapies being ignored while expensive disease-management therapies thrive. For example, many countries in Europe have long-ago switched to the M2PK test as a safer, less invasive, lower cost and better efficacy (lower false positives and false negatives) for colon cancer screening compared with colonoscopy, which is still the overwhelmingly favored procedure in the USA.

Such cost differences illustrate the mostly hidden economic downside of cronyism.

Here are some cost figures for the natural self-defense substances mentioned in these pages.

<table>
<thead>
<tr>
<th>Supplement</th>
<th>Cost per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>vitamin D3</td>
<td>$0.04 for 7,000 IU/day administered weekly.</td>
</tr>
<tr>
<td>selenium</td>
<td>$0.001 for homemade selenite (what I make).</td>
</tr>
<tr>
<td></td>
<td>$0.01 for commercial, over-the-counter selenite (Nutricology-brand).</td>
</tr>
<tr>
<td></td>
<td>$0.10 for selenocysteine or selenomethionine.</td>
</tr>
<tr>
<td>vitamin C</td>
<td>$0.05 for above-RDA supplementation.</td>
</tr>
<tr>
<td></td>
<td>$0.20 for 2 grams of ascorbic acid in capsules.</td>
</tr>
<tr>
<td></td>
<td>$0.40 for 4 grams of ascorbic acid in capsules.</td>
</tr>
<tr>
<td></td>
<td>$2. for bowel-tolerance dose without infection.</td>
</tr>
<tr>
<td></td>
<td>$8. for bowel-tolerance dose with viral infection.</td>
</tr>
<tr>
<td></td>
<td>$200. for out-patient vitamin C IV infusion (estimate).</td>
</tr>
<tr>
<td></td>
<td>$400. for four in-patient vitamin C infusions with IV line already in place (est).</td>
</tr>
<tr>
<td>zinc</td>
<td>$0.10 50 mg veggie caps, as monomethionine and citrate chelates, one.</td>
</tr>
<tr>
<td></td>
<td>$0.04 50 mg tablets, gluconate chelate, one.</td>
</tr>
<tr>
<td></td>
<td>$0.75 23 mg lozenges, one every two hours while awake.</td>
</tr>
<tr>
<td>copper</td>
<td>$0.05 2 mg veggie caps, one per day.</td>
</tr>
<tr>
<td></td>
<td>$0.50 topical copper-peptide cream. $60/2oz (AcheRelief). 1/4 ml/day.</td>
</tr>
<tr>
<td>vitamin E</td>
<td>$0.45 mixed tocopherols, 360 mg, gelatin pearl.</td>
</tr>
<tr>
<td></td>
<td>$0.40 mixed tocopherols and tocotrienols, gelatin pearl.</td>
</tr>
<tr>
<td>IV heparin</td>
<td>$15. 500 ml, 50 units/ml, $14.82. 1000 units per hour = 480 ml/day.</td>
</tr>
<tr>
<td>nattokinase</td>
<td>$0.50 four 2,000 FU veggie caps, one upon waking, before bed, between meals.</td>
</tr>
<tr>
<td>lumbrokinase</td>
<td>$1.45 two 20 mg veggie caps, one upon waking and one before bed.</td>
</tr>
<tr>
<td>vitamin A</td>
<td>$0.07 15,000 IU retinyl palmitate in gelatin pearl.</td>
</tr>
</tbody>
</table>

Links

Rather than discuss all the pros and cons of the public-health messages that twist science and subvert medical care, I’ll provide a list of links for those with cast-iron stomachs who can listen to (or read) a “lone voice of reason” without upset. Some of these links reiterate information in this book. Others not. But if you have faith in the official reporting and believe the governmental propaganda, do not follow these links.

Abstract: **Objectives:** To determine whether parachutes are effective in preventing major trauma related to gravitational challenge. **Design:** Systematic review of randomised controlled trials. **Data sources:** Medline, Web of Science, Embase, and the Cochrane Library databases; appropriate internet sites and citation lists. **Study selection:** Studies showing the effects of using a parachute during free fall. **Main outcome measure:** Death or major trauma, defined as an injury severity score > 15. **Results:** We were unable to identify any randomised controlled trials of parachute intervention. **Conclusions:** As with many interventions intended to prevent ill health, the effectiveness of parachutes has not been subjected to rigorous evaluation by using only observational data. We think that everyone might benefit if the most radical protagonists of evidence-based medicine organised and participated in a double blind, randomised, placebo controlled, crossover trial of the parachute.

Ron Rosedale MD. “Was Otto Warburg Wrong?” mTOR and low-protein diet.

Nina Teicholz is the author of *The Big Fat Surprise* (Simon and Schuster, 2014), which challenged governmental dietary recommendations and shook up the public’s perception of fat as the “bad guy” for diseases of civilization. This is the link to The Nutrition Coalition (non-WSJ) version of Nina’s article due to the WSJ only allowing access to registered readers.

This article asserts that the dietary advice by the US government to eat carbs as the foundation of the food pyramid has failed to stem the tide of obesity, diabetes and other pre-existing conditions which are now known risk factors for adverse Covid-19 outcomes. It also uncovers malfeasance on the part of the 2015 “expert committee” that formally reviewed the scientific evidence for low-carb diets reversing diabetes and obesity, yet failed to publish alongside the other formal reviews that had been conducted.

It’s now 2020 and the expert committee is again charged with revising official governmental dietary recommendations. Their new report is due this month.


This excerpt edited to fit space: “What does this all mean at the present time? We have in China: (1) a record of laboratory escape of the SARS virus in 2004 from a premier Chinese research institute; (2) a record of poor biosafety in some of its high-containment facilities,
including the Wuhan institutes; (3) a record of suppression of information in general, and in the case of SARS-CoV-2 in particular; (4) the initiation of a disinformation campaign in regard to the origin of SARS-CoV-2, targeting US biological laboratories; and (5) a record of gain-of-function research at the Wuhan Institute of Virology, including passage of a bat coronavirus construct through experimental animals.”

https://www.hormonesmatter.com/thiamine-deficiency-causes-intracellular-potassium-wasting/

“Animal research in rats showed that chronic thiamine deficiency increases sodium tissue content in heart, liver and skeletal muscle by 18-35%, while also decreasing potassium content by 18-25%. Interestingly, although tissue levels were altered, plasma levels of these electrolytes remained unaffected and stayed within the normal-high range (sodium at 141.6 and potassium at 4.8). This means that blood measurements did not reflect tissue content.” In humans, potassium-utilization variations caused by metabolic factors cause serum potassium to be independent of cellular potassium.

http://orthomolecular.org/resources/omns/v10n13.shtml

This is a page written by Steve Hickey, Hillary Roberts and Damien Downing, all vitamin C experts, which discusses the general use of vitamin C and the potential for its use in Ebola.

https://fee.org/articles/how-states-turned-nursing-homes-into-slaughter-houses-by-forcing-them-to-admit-discharged-covid-19-patients/?fbclid=IwAR2xFc8ZXvIugmSTguGmg_DISaHMrg223iz-EqGtCvP_GrrHEayXFrQ_k

An excellent example of governmental “good intentions” going horribly wrong. Mmanslaughter is the term if governmental bureaucrats were citizens making the same decision.


Dr. Shiva Ayyadurai interviewed by Stefan Molyneux, self-proclaimed anarcho-capitalist and philosopher, and interviewer on Freedomain Podcast. A very fast-paced interview with many excellent points and some incompletely ideas. Short attention-span theater, but fascinating.
John P A Ioannidis. The infection fatality rate of Covid-19 inferred from seroprevalence data. medRxiv preprint. From the abstract: “Results: 23 studies were identified with usable data to enter into calculations. Seroprevalence estimates ranged from 0.1% to 47%. Infection fatality rates ranged from 0.02% to 0.86% (median 0.26%) and corrected values ranged from 0.02% to 0.78% (median 0.25%). Among people <70 years old, infection fatality rates ranged from 0.00% to 0.26% with median of 0.05% (corrected, 0.00-0.23% with median of 0.04%). Most studies were done in pandemic epicenters and the few studies done in locations with more modest death burden also suggested lower infection fatality rates. Conclusions: The infection fatality rate of COVID-19 can vary substantially across different locations and this may reflect differences in population age structure and case-mix of infected and deceased patients as well as multiple other factors. Estimates of infection fatality rates inferred from seroprevalence studies tend to be much lower than original speculations made in the early days of the pandemic.”

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John P A Ioannidis is professor of medicine and professor of epidemiology and population health, as well as professor by courtesy of biomedical data science at Stanford University School of Medicine, professor by courtesy of statistics at Stanford University School of Humanities and Sciences, and co-director of the Meta-Research Innovation Center at Stanford (METRICS) at Stanford University.


The best, short, spoken review of the status of vaccine safety and testing that I have ever seen, and ethical condemnation of public-health officials, too. Six minutes to understand the issues.

A very thorough review of the potential involvement of vasopressin and oxytocin in neuromodulation of “social neurocircuits,” which are likely disrupted by social-distancing policies and practices.

A broad-ranging and exceedingly detailed review of therapeutic strategies with 380 references to the primary literature.

Professor Johan Giesecke, MSc, MD, PhD speaks out against draconic measures, which at the time they were implemented throughout the world were not evidence-based, and to this date are questionably documented. All about the Swedish experience; the mistakes they made with
the AIDS epidemic and how they decided not to repeat their mistaken policy with the Covid epidemic.

★★★★★  https://www.youtube.com/watch?v=UQcCIjz9_s

Investigative journalist Sharyl Attkison tells the story of how the “fake news” media campaign launched by Democratic supporters of Hillary Clinton was ironically coopted by Donald Trump and the Republicans.

★★★  https://earthheroestv.com/programs/special-free-live-broadcast-5th-jan-2021-focus-on-fauci-46120-a90064

Very uneven production values, but plenty of insights into Anthony Fauci’s abject negligence regarding public-health policies, his termination of scientists conducting research that undermines and contradicts his public and private statements, and his use of federal funding to influence decisions on drug and vaccine approval committees.

★★★★★  https://www.bitchute.com/video/7nhmZi6rbKLu/?fbclid=IwAR0lqorSYSKWqXuVvaVglWMqEz1b15TRuzNQJgoQozYc36DPzjPMW-BNBo

An interview with Lee Merrit, M.D., conducted by Alex Newman, Senior Editor of The New American and published on BitChute to prevent censorship by Facebook and Google. Dr. Merrit talks about her experiences in the trenches of public-health research and delivery and puts what is being done—and not done—in the context of medical ethics. There are several questions that I would have liked to ask of Dr. Merrit that Newman did not, but overall, it is a scathing indictment of public-health officials overtly failing to do their jobs and the illogical and unjustifiable public-health policies that are being imposed by ignorant politicians. She provides excellent perspectives on (1) the medical utility of vaccines, (2) the availability of antimicrobial drugs (lysosomotropic agents, like ivermectin and hydroxychloroquine) that effectively treat acute viral infections like Covid-19, (3) the level of vitamin D is the best predictor of Covid-19 ICU risk, (4) that nobody getting the mRNA neo-vaccines are being given informed consent, (5) that posting a direct quote from the vaccine insert sheet can get you censored and your Facebook account suspended, (6) that no mRNA vaccine for SARS or MERS has ever made it through animal testing, including those being given to humans for SARS-CoV-2, (7) the change in worldwide survivability (99.992% before Covid-19, to 99.991% with Covid-19), and (8) cites her emergency Covid-kit at the office contains NAC, vitamin C, vitamin D, zinc, selenium and quercetin.

(+ add link to Johan Giesecke, professor “Why Lockdown are the wrong policy.”
(+ add link to Knut Wittkowski, Rockefeller University, retired professor of biostatistics, epidemiology and research design

Social distancing and lockdown is the absolutely worst way to deal with an airborne respiratory virus.

(+ Bruce Lipton: Do not fear the bogeyman. -----Fear itself is deadly?
(+ Dan Erickson and Artin Massihi: Clear evidence after 5 weeks. Lockdowns are a very bad mistake.
(+ Simon Thornley, M.D., U Auckland, epidemiology and biostatistics.
(+ philosopher Stefan Molyneux and Shiva Ayyadurai conversation.
Censorship and Censuring

One way you can know that this is a crisis of unprecedented scope is the degree to which big-Internet companies are engaging in mass censorship at the behests of public-health doctrine. Why would they risk their reputations unless they knew that they would never have to pay the price of their violation of consumer trust. This means that Google, Facebook and the rest likely know that freedom of speech is dead or dying.

Is there enough political will left in the USA to reverse censorship? I have my doubts.

**Plandemic.** Banned from YouTube, May 2020.

★★★★ https://citizenmedianews.com/2020/05/06/plandemic-documentary-part-i-dr-judy-mikovits/

The biggest conspiracy video about foreknowledge of a pandemic, issues of gain-of-function research and acknowledged public-health vaccine policies. Arguably, the first censorship target of 2020.

★★★ https://www.youtube.com/watch?v=Oqe-9mOLj3o

Steve Blake. “Beyond the mask: How to survive Covid-19.” SVHI video interview, August 20th, 2020. Banned by YouTube. Reposted on my YouTube channel in raw (unedited) format (so there is stuff at the beginning and end that you may want to skip over).

How the Taiwanese Track Hidden Chinese Policy

The Taiwanese have figured out how to track Chinese public policy. They know that the Chinese government engages in massive propaganda campaigns and regularly lies about events taking place in China. The way the Taiwanese get around that problem is to track the Chinese social media platforms and observe what Chinese censors are actually censoring. That tells them exactly what the Chinese government is up to and does not want anybody to know.

Maybe US citizens should pay equal attention to US censorship for the same reasons?

A Few Censorship Quotes

“No government ought to be without censors, and where the press is free, no one ever will.”

——Thomas Jefferson (1743 - 1826), letter to George Washington, September 9, 1792.

“I can imagine no greater disservice to the county than to establish a system of censorship that would deny to the people of a free republic like our own their indisputable right to criticize their own public officials. While exercising the great powers of office I hold, I would regret in a crisis like the one through which we are now passing to lose the benefit of patriotic and intelligent criticism.”


“All censorships exist to prevent anyone from challenging current conceptions and existing institutions. All progress is initiated by challenging current conceptions, and executed by supplanting existing institutions. Consequently, the first condition of progress is the removal of censorships.”

——George Bernard Shaw (1856 - 1950)
Appendix G: Great Barrington Declaration

The Great Barrington Declaration was written by three eminent scientists with expertise in public health, epidemiology and statistics. It is written as a simple declaration of suggested policy (quarantine only those that need protection) instead of as a statement of science. This makes it very easy to understand.

This policy suggestion is not only diametrically opposed to current policy, it endorses the traditional public-health policy that has been implemented in all past epidemics to date. Quarantine those who are at risk.

So what is the science behind the declaration?

The first thing is that the dangers of SARS-CoV-2 have been exaggerated. The original official estimates for Covid-19 mortality were about 3%, when the actual mortality is 0.3% (conservatively), and by best argument 0.2%.

How could a ten-fold exaggeration be justified?

The answer is that public-health bureaucrats and in-the-trench physicians totally misjudged the number of infected individuals. What they saw was the sickest of the sick and they generalized that everybody with Covid-19 would be apparently sick. There was no evidence for this, but public-health bureaucrats made the pronouncement and used the 3% figure to justify draconian measures at utter odds to the traditional quarantine protocols that had been used for centuries.

Here’s what the picture looks like.
Without decent testing methods, there was no way to screen people. When testing methods were developed, they were not used beyond the acute clinical environment. The plans to produce large quantities of testing kits were derailed by the NIH, who insisted on developing and validating the testing kits themselves and botched the process, sending medical providers back to square one by maintaining their refusal to purchase testing kits from other countries. As a result, the public-health analysis continued to be based on guesswork.

Wrong guesses. And in 20:20 hindsight, disastrously wrong guesses.

Ultimately, when testing became a massive political priority, the testing revealed that a huge percentage of the population had already been infected. These were people who had (a) asymptomatic Covid-19, (b) near-asymptomatic Covid-19, (c) mild to moderate Covid-19 not severe enough to warrant a hospital visit, or (d) moderate to severe Covid-19 warranting clinical support but too afraid to go to a hospital, or not able to be admitted because of over-crowding. At this time, Covid-19 was still being treated as a pneumonia-like condition and those placed on mechanical ventilation had an 80-90% mortality rate.

But when this data started pouring in, it was deliberately misinterpreted in defense of the draconian policy. These former cases were labeled as “new cases” and alarming media reports resulted. But the massive increases of “new cases” was not matched by correspondingly increasing deaths.

As of 14 January 2021, roughly one year after SARS-CoV-2 entered the United States, the death rate is 0.197% (Statista.com), 0.167% (CDC.gov), 0.165% (covidtracking.com), with the only two states with percentages above 0.2% being New Jersey (0.226%) and New York (0.206%). There are 20 states with numbers less than 0.1%, including Wisconsin, Nebraska, West Virginia, Colorado, Wyoming, Idaho, Ohio, California, North Carolina, Oklahoma, Kentucky, Virginia, New Hampshire, Washington, Utah, Oregon, Maine, Alaska, Vermont and Hawaii (0.022%). However, I personally do not believe that reporting is particularly honest or uniform among the states, so this data should be taken with a pound of salt.

I projected this seasonal attenuation for SARS-CoV-2 back in March, 2020, publicly and privately, based only on the known seasonality of all the typical lipid-enveloped viruses, of which SARS-CoV-2 was one. Plus, this seasonal pattern had been previously observed in 2003 with SARS-CoV-1, which bloomed in the winter of 2002 and attenuated at the start of the 2003 summer. In the above illustration, this is represented by a pale, pinkish underlay, which is a generalized display of how colds and flus affect people. This is shown as a 1:2 proportion between mild years and severe years, but the reality is that some years are even milder than shown (2010) and others are substantially worse (2017).

The curve follows both temperature trends and vitamin D trends. However, the bias in scientific circles is that it is a marker for seasonal vitamin D levels, and in the public-health community, the bias is that it is more a temperature phenomenon. The evidence is now becoming clear that the temperature hypothesis is wishful thinking on the public-health community’s part (see page 33), likely due to their extreme reluctance to admit that such illnesses can be prevented and that they are choosing to do nothing about it. But, sadly, that is the case. They are doing nothing about a situation that can be easily prevented—and treated. It is now painfully obvious (no pun intended) that vitamin D supplementation totally reverses this seasonality and even improves clinical situations far better than anything seen during the anti-flu summer season.

When dealing with statistics, it is important to know that data is being politically manipulated. During the 2020 flu season, the CDC suddenly stopped collecting flu statistics. Why? The flu numbers were crashing.

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352 Protein-capsid (non-lipid-enveloped) viruses also display this identical seasonal pattern.
Their explanation for this policy change is that flu deaths no longer mattered. But the conspiracy theories are that flu deaths were being reclassified as Covid-19 deaths and the dropping influenza death numbers were the smoking-gun evidence.

This is not just a problem with reclassification of flu deaths. It is happening for heart-disease deaths, diabetes deaths, pneumonia deaths, stroke deaths, COPD deaths, old-age deaths, and possibly most chronic and degenerative causes of death (excluding mostly accidents). This may not make sense to anybody with a logical mind, but the US government pays hospitals $13,000 for every Covid-19 death and zero for other causes of death. This might be easily described as institutional corruption in hospital administrations if it were not for the fact that federal governmental institutions have told physicians and administrators, in writing, to classify any death as Covid-19 if it is possible that it is related to SARS-CoV-2 in any way. In any way at all.

These ways are not limited to positive Covid-19 test results. It would certainly be misleading to classify somebody as a Covid-19 death when they actually died of end-stage heart or kidney failure. But the official instructions were to use any sign of Covid-19 in the absence of a test result, which included such common symptoms as a chronic cough.

With financial incentives and official permission, Covid-19 statistics have become massively inflated. 

*Caveat emptor.* “Let the buyer beware” is now “let the perceiver of CDC statistics beware.”

**Cause-of-Death Statistics**

How much would cause-of-death statistics need to be manipulated to make SARS-CoV-2 a true threat to humanity and Covid-19 ten times more deadly than the typical flus and colds that have plagued us for millenia?

Here are the “top ten” CDC statistics from before the SARS-CoV-2 pandemic for a single year:

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>Count</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart disease</td>
<td>632,636</td>
<td>34.075%</td>
</tr>
<tr>
<td>Cancer</td>
<td>559,888</td>
<td>30.156%</td>
</tr>
<tr>
<td>Stroke</td>
<td>137,119</td>
<td>7.385%</td>
</tr>
<tr>
<td>Chronic obstructive respiratory distress accidents</td>
<td>124,583</td>
<td>6.710%</td>
</tr>
<tr>
<td>Diabetes</td>
<td>72,449</td>
<td>3.902%</td>
</tr>
<tr>
<td>Alzheimer’s disease and senility</td>
<td>72,432</td>
<td>3.901%</td>
</tr>
<tr>
<td>Influenzas and pneumonias</td>
<td>56,326</td>
<td>3.034%</td>
</tr>
<tr>
<td>Nephritis (kidney failure)</td>
<td>45,344</td>
<td>2.442%</td>
</tr>
<tr>
<td>Septicemia (blood infection, sepsis)</td>
<td>34,234</td>
<td>1.844%</td>
</tr>
<tr>
<td>Total</td>
<td>1,856,610</td>
<td>99.999%</td>
</tr>
</tbody>
</table>

If you will notice, influenzas and pneumonias are a tiny fraction of the total deaths. And while it might seem that chronic obstructive respiratory disease (COPD) might be related to acute respiratory distress syndrome (ARDS), they are actually not related at all. COPD is a chronic degenerative disease that may have opportunistic infection involved, but ARDS is not a degenerative disease at all. So we have 3% if total deaths being caused by flu-like infections that include coronavirus conditions.
If we round up the annual US death rate to 2 million and round up the US Covid-19 deaths in 2020 to 100,000, then the Covid-19 mortality is now 5-8% of total deaths instead of 3%. I provide this range because there is a potential overlap of flu-like deaths and Covid-19-like deaths.

What is the overlap?

If all of the flu deaths have been reclassified as Covid-19 deaths, the 100,000 Covid-19 deaths in 2020 is roughly double the expected 50,000 annual cold-and-flu deaths. If none of the Covid-19 deaths were reclassified, then the viral death rate is triple its normal annual amount. But since we do not now to what extent flu deaths are being counted as Covid-19 deaths, there is uncertainty in the numbers.

The fact that the CDC stopped tracking flu deaths and that they were plummeting before they stopped counting is some evidence that flu deaths were reclassified. But the smoking-gun evidence is directives from the CDC to hospitals instructing them to declare deaths to be Covid-19 deaths if there was any evidence that SARS-CoV-2 was involved. This evidence was overtly stated to not require a positive test result. This made re-classification of deaths a matter of official public policy.

So the question has become, how many flu, cold and pneumonia deaths were classified as Covid-19 deaths? And since this official policy is not condition specific, how many diabetes deaths and cardiovascular deaths were reclassified as Covid-19 deaths? How many cancer deaths were reclassified as Covid-19 deaths? Let’s pick something closer to home symptomwise; how many COPD deaths and stroke deaths were called Covid-19 deaths? COPD deaths can look superficially like Covid-19 deaths and Covid-19 side effects include blood coagulation pathology, so why not? Not only is the government telling you, a practicing physician, to call everything possible a Covid-19 death, the same government is paying your bosses (and their accountants) $13,000 for every Covid-19 death, and $39,000 for every Covid-19 death on a respirator. So even if you are an ethical physician wanting to stick to your guns and call a cardiovascular death a cardiovascular death, your employers will quickly tell you the errors of your ways. Physicians have been fired for this.

What possible evidence can sort this out? Actually, there is one. Total mortality. You might be able to lie about why somebody died, but you cannot lie about that somebody died. So total mortality figures give us a different perspective. One stated statistic is 99.92% survivability went to 99.91% survivability.

Everybody is justifying official US public health policy because of how bad the US statistics are, but they are officially corrupted by those people citing them as evidence of danger. Is that not a fox-guarding-the-henhouse situation?

And for whatever their motives are, we agree to sacrifice the education of our children, our family values, our freedoms, our economic livelihoods, and the economic livelihoods of our neighbors.
And produce sustained psychological stress of an unprecedented magnitude.

These disease-death numbers do not reflect total mortality; they are merely what is reported. For example, flu deaths in children must be reported, but not adult flu deaths. There are many published papers presenting evidence that there is significant under-reporting of influenzas and pneumonias. There is also no reporting of iatrogenic deaths as a separate category even though it is admitted by multiple authorities to be third on the list of causes of death—and likely first on the list if the truth were told.

**Santa Clara County Tells the True Story**

Dr. Jay Bhattacharya, a Stanford scientist, professor, statistician, epidemiologist and physician, conducted one of the earliest studies of antibody levels for the SARS-CoV-2 virus. Antibodies tell us who was infected, not who is infected. In April, 2020, the case-associated death rate was publicly stated to be 3% by the CDC and WHO. This was the justification for a host of radical public-health policies, including quarantining healthy people, shutting down “non-essential” businesses (including doctor’s offices), and mandating mask wearing in public.

Yet when Dr. Bhattacharya and colleagues tested people in Santa Clara, they found that 50,000 people had been infected with SARS-CoV-2, 50 times the roughly 1000 cases that had been identified by the county up to that time. Of course, the public-health experts did not believe it and dismissed it as a flawed study, but this was not the case. This result has now been replicated more than 50 times all over the world with the same results. We now know, beyond a shadow of a doubt, that the public-health officials were wrong and the case fatality rate for Covid-19 was 0.2% rather than 3%. But their 3%-justified draconian public-health policies remain in place.

While such draconian public policies may have been warranted in March and April when they did not know better, there was a problem with Covid-19 disease treatment. The doctors and hospitals did not know what they were dealing with and used the wrong treatments. Covid-19 looked like a viral pneumonia in some ways, and was treated that way, with disastrous results. The hospitals were packed with people in respiratory distress, who tended to linger on ventilators, dying in high numbers. This was the basis for the flattening-the-Covid-19-curve justification for the draconian policies.

**The CDC Drops the Ball**

But there was a problem. The CDC and other public-health officials were not doing their jobs. They did not study the validity of different testing systems. Epidemiologists know that data sets from different testing modalities cannot be meaningfully correlated without knowing how accurate and precise they are. This is done by head-to-head testing using both systems on the same test population. This tells them not only validity, accuracy and precision, but also false-positive and false-negative rates. This well-known and historically traditional job for epidemiologists was completely ignored.

Who changed this policy at the CDC? And why? Nobody outside of the CDC knows.

It’s much, much worse. The CDC also had the job of examining the differences between the seriously affected and the minimally affected. This is also, historically, Job One for public health officials. Had they done their jobs, the course of Covid-19 would have been drastically different. Had the vitamin D risk factor been discovered in the USA, it couldn’t be ignored or dismissed by appeal to prejudice. But it was found in Singapore and Malaysia, where it has been dismissed and ignored. Because of the CDC not doing their job, some of our hospitals are over-crowded and ICUs overloaded. If the hospitals had adopted vitamin D and lysosomotropic drugs (zinc plus hydroxychloroquine, and ivermectin) in a timely manner,
we would have emptied out the hospitals and ICUs, allowing the end of the flattening strategy and transitioned to quarantine of only the susceptible. The Covid-19 crisis would be over.

But the CDC failed to do their job. A century of precedent, decades of technical sophistication, all abandoned for reasons unknown outside of the CDC. And because of this bureaucratic malfeasance, the Santa Clara study was ignored, the most effective treatment (vitamin D) was ignored (see page 26), and a whole class of highly efficacious drugs (lysosomotropic agents) was dismissed without cause (see pages 194-197).

Many Democratic doctors, nurses and academics believe this was because of Trump, but it was actually an internal discretionary decision beyond Trump’s influence. The trust and faith invested in the CDC is misplaced.

**The Thousand-Fold Covid-19 Risk Profile**

One of the other public-health mistakes was (1) recognizing the thousand-fold difference between the risk to the elderly compared to children, (2) withholding that realization from the public, media and other branches of government, and (3) not changing public policy from lockdowns of everybody to mere quarantine of those at risk. In other words, quarantining children, teens and adults to protect the elderly was backwards. Historically, quarantine policy has always been the exact opposite; quarantine those at risk and not everybody else.

When Covid-19 risks to children are put in perspective, roughly three times as many children die of flu every year than died from Covid-19 in 2020. But for seniors, Covid-19 is much more deadly than the flu. Some of the reasons for that are outlined in this book. Just one, the higher prevalence of vitamin D deficiency in elders, if corrected, could easily bring Covid-19 mortality down into the ranges commonly seen with low-severity flus. And it is my assertion, that Covid-deaths overall can be equivalently decreased in every segment of the population where vitamin D is a risk factor. This involves more than half of the world’s population.

So what justification remains for lockdowns?

There are only two: (1) pandering to the egos of public-health officials, and (2) maintaining social control for Machiavellian motives. Neither of these are legitimate.

**Catastrophic Economic and Social Consequences**

Although vehemently denied by official spokespersons, the economic and social costs of lockdowns are quite severe. The damage to small businesses has been catastrophic, and the cost to major businesses has not yet been fully realized. The United Nations may not be particularly honest or fair minded, but they have estimated that the lockdowns will directly and indirectly result in 130 million more people starving to death this year. If we accept that figure, and the inflated figure that 2.2 million people have died from Covid-19 to date, that’s 60 people’s lives sacrificed to starvation for every life lost to Covid-19.

So the argument that any life is worth saving at any economic cost takes on a new meaning when Covid-19 lives are paid for with other human lives.

And it gets worse when we realize that the Covid-19 lives lost and those we are trying so hard to save (allegedly) are those with serious pre-existing conditions and elders. On the other hand, the lives lost to starvation are young and poor.
Ethically, it’s indefensible.

What other costs of lockdowns are not being considered?

**Mental Health**

Human are social beings. The quarantines and lockdowns prevent many aspects of social interactions that support human welfare and happiness. Dr. Bhattacharya cites a CDC survey in June 2020 that found that 25% of young adults between the ages of 18 and 24 had seriously considered suicide. But, apparently, the CDC’s own surveys are not worth consideration. And the ethics of forcing young people to pay this price when they are not as risk is a reversal of the fairness principles that underly most societies today.

**Education**

The shutting down of schools and forcing education to be solitary has never happened before. It may take a decade or a generation to assess the true cost that is being paid for this policy. Certainly, the public-health officials have no idea what magnitude this cost will be.

There is another cost, that parents have to resume their educational roles for their children. The delegation of educational responsibility to schools is one way parents can generate a two-income household. But with children at home 24/7, parenting responsibilities must be resumed for education. This is not only an educational burden when parents are not prepared, but it is a mental health issue for both the children and parents as well.

**Reversing the Quarantine**

How can this be accomplished? First, shelter those at risk. Have groceries and supplies delivered. In nursing homes, have staff, residents and visitors tested frequently. Hire staff with acquired immunity. Minimize staff rotations. Set up exterior patios for family visits. Take advantage of airflow patterns. Hire a medical director who knows about functional medicine. Provide vitamin D supplements as routine care.

For businesses, establish a stay-at-home policy for symptomatic employees. Pay for frequent PCR and infrequent antibody testing, assuming that this is not officially subsidized. Divert employees with acquired immunity to cover essential networking requirements. Make sure that medical insurance coverage includes functional treatment for Covid-19 and IV vitamin C. If you internalize medical costs, hire a medical director to educate your entire medical team about functional Covid-19 treatment. And provide free supplements to all employees who want vitamin D, vitamin A, vitamin C, selenium, zinc, potassium and magnesium.

For hospitals, give 50,000 IU of vitamin D to every inpatient on day one, before you even know if they test positive or not. Then employ the full spectrum of innate-immune nutritionals, with ivermectin and/or a zinc-hydroxychloroquine protocol. Collect data and share it with other hospital teams. Use intravenous vitamin C and heparin in all ICU cases. Provide free nutritional supplements for all hospital employees.

For the government, give away vitamin D to low-income and unemployed people who ask for it. Fire the top bureaucrats of the CDC, NIH and FDA, replacing them with people who will do their jobs without industry bias and with the highest ethical standards. And lastly, preferentially hire people who fired for speaking up against fraud and malfeasance.
Science and Civility

The propagandizement of the lockdown policy by public health officials and media moguls has resulted in intolerance towards civilized discourse. Scientists, educators, scholars, and citizens in the street have been treated inhumanely when they have called in to question the assumptions and mistakes that have been made by those enforcing lockdown policies. Medical doctors and scientists have been fired from their jobs. People are accused of “not caring” and “putting people’s lives at risk,” when the facts are the other way around. Citizens engage in public shaming of those who disagree, on both sides of these issues. Civility is a lost virtue.

When vaccines become mandatory, divisiveness will escalate another order of magnitude.

Those pushing lockdown policy have backed themselves into a corner; they cannot back down. They have their egos, careers, reputations and livelihoods at stake. There is no way that they can back down gracefully. There is no face-saving path for them to make amends.

And while most people in the USA are relieved by the passing of Donald Trump into history, their nearly unanimous belief that things will rapidly get better is setting them up for disappointment. Now that public-health officials have an entrenched defender and ally in the White House, US policy will swing towards the public-health agenda of masks and lockdowns, not away from it.

So US politics is likely to become even more divisive.

★★★★★ https://www.americasfrontlinedoctors.com/summit2/

A collection of more than a dozen videos by a wide variety of medical doctors and academics who are fundamentally opposed to involuntary treatments with experimental pharmaceuticals and oppose current public-health policies.

★★★★★ https://www.youtube.com/watch?v=cwPqmLoZA4s&t=15s

John Ioannidis, M.D., D.Sc. has long held views of reason in the face of malfeasance in science. Here he speaks about Covid-19 and SARS-CoV-2. Unlike his previous video, this video has not yet been censored.

★★★★ https://www.youtube.com/watch?v=PC3nptwY50I

John Ioannidis, M.D., D.Sc. again. The case that Covid-19 mortality rates are comparable to the flu.

★★★★★ https://www.youtube.com/watch?v=T-saAuXaPok

John Ioannidis, M.D., D.Sc. an explanation of the Santa Clara antibody study and comparative risks assessments with other human activities, hot spots, hospital loading risks, Sweden, suicides, unemployment, and more. This was in April, 2020, yet his observations fared better with time than all of the news stories and official announcenets alsest any others.

★★★★★ https://www.youtube.com/watch?v=QUvWaxuurzQ

John Ioannidis, M.D., D.Sc. with an excellent analysis of the Covid-19 data collected from the Diamond Princess cruise liner and the hot spot in northern Italy.
Appendix Q for Quora

Quora: What does it mean to have a good metabolism? 353

Original Question: What does it mean to have a good metabolism? How does it impact our body?

A good metabolism refers to the rate at which you burn food for energy. And the good part is that the rate is sufficient to meet your metabolic needs. The “energy” that is implied in “basal metabolic rate” (BMR) comes in two forms, ATP (adenosine triphosphate) and NADH (nicotinamide adenine dinucleotide hydride). The first is the driver of the enzymatic reactions that we term “metabolism” and the second is the active reducing (anti-oxidizing) molecule. ATP also serves signaling functions and is converted into other “energy” molecules like GTP and creatine phosphate. And NADH converts oxidized glutathione into reduced glutathione, and oxidized ascorbate (vitamin C) into reduced ascorbate. Glutathione is the primary redox-buffering molecule of the cell and vitamin C is the primary redox-buffering molecule of the extracellular matrix.

Redox is the reverse contraction of OXidation and REDuction. The redox-buffering system is a necessary and essential aspect of the antioxidant defense system. While most antioxidants protect us from energetic and dangerous free radicals like umbrellas protect us from the heat of the sun, the redox-buffering system actually cools us off from the sun-driven ambient temperature. The redox-buffering system is the foundation of the antioxidant defense system, taking the oxidized glutathione and ascorbate and “recycling it” (cooling it off) back to reduced glutathione and ascorbate. So it “buffers” (defends against) oxidative stress by cooling off “hot spots.” All this is driven by the NADH production of your “good metabolism.”

Now let me challenge the censorship ethic of Quora to see if they are following the same regrettable censorship practices of Facebook and YouTube by tying this answer into coronavirus infections as an example of a hotspot. Not only does the immune system relate the heat of the hotspot to the base temperature of the redox-buffering system, but the “depth” of the redox-buffering “pool” determines how much oxidative stress you can handle from the free radicals that your immune system produces to fight the coronavirus. If your metabolism is good, the pool of “reducing equivalents” is deep and able to prevent the oxidation insult from the infection from overwhelming your body’s redox-buffering capacity and causing a cytokine storm. If your metabolism is less than good, you have a “pre-existing condition” that makes you more susceptible to the virus. That’s not a trivial “impact” to our bodies. It’s a life-and-death issue.

But it’s a life and death issue only to the extent of the degree of oxidative stress that is being buffered. That oxidative stress varies from mild, like a scraped knee or a traditional cold, to serious, like coronavirus infections, radiation poisoning and second- and third-degree burns. So coronavirus infections challenge our redox-buffering systems to a degree beyond what we experience in “normal” life, whatever that might mean. So the coronavirus is “uncovering” the hidden “pre-existing conditions” that most of have and do not know. Insulin resistance is the hidden risk factor that becomes visible when we get a diabetes diagnosis. Vascular inflammation from MMP (matrix metalloproteinases) activity is a hidden risk factor that becomes visible when we develop cardiovascular disease.

It’s like the proverbial iceberg, where the tip is visible (what you know about your pre-existing conditions) and the greatest mass is underwater (what you do not know). The good news is that mild pre-existing

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conditions are everywhere and most people with them survive coronavirus infections just fine. But there is potential danger in not knowing what pre-existing conditions you may have, or how mild or severe they are. Some of them, like metabolic rate, are statistically related to aging itself. We tend to lose one percent of our metabolic rate for every three years of aging. I’m 68 years old, and I have had significant insulin resistance for 30 years. But I know about it, and “lean into it” by monitoring my hypometabolic symptoms (low body temperature, cold hands and feet, slow pulse rate, tendency towards constipation, tendency towards depression, edema (facial puffiness) and cognitive difficulties, and take active steps to mitigate my constitutional weaknesses, by, for example, taking thyroid hormone intermittently, fasting for a day once every two weeks, doing carb-restriction half of the time, and taking dietary supplements of selenium and vitamins C, A and D₃ (to mention the ones I think are most related to coronaviral risks). Because I have this systems-level understanding of the redox-buffering system and know how to feed it (deepen the pool) during an oxidative challenge, I’m not worried about coronavirus virulence despite my age and pre-existing conditions.

But in terms of your question, having a good metabolic rate is at the top of my list of things to cultivate for a long and active lifespan.

I really appreciated your question.

**Quora: What is happening to children?**

Original Question: *What is happening to children who have been diagnosed with COVID-19?*

The same things are happening to children as are happening to adults and the elderly. The covid-19 infection manifests as an oxidative stress on the body, which activates the immune systems (cellular and humoral), causing massive oxidative stress and upregulation of humoral immune mechanisms (as expressed by cytokine cell-signaling mechanisms) and upregulation of, for example, cathelicidin for cellular immunity.

When there are pre-existing conditions that interfere with these mechanisms, things go wrong and people can die. For example, vitamin D level affects the cellular expression of the antiviral protein cathelicidin. Its is not just the elderly that have vitamin D deficiencies, because of declining kidney function for activation of vitamin D₃. Some children and elderly are kept indoors during the noon hours in the mistaken belief that it will lower their risk of skin cancer or because it’s just too expensive to hire the staff to supervise sunning.

The brunt of the oxidative stress of the humoral immune system, which uses vitamin C in high amounts to generate hydroxyl free radicals to “kill” invading microorganisms, is buffered by the antioxidant defense system. That system depends on glutathione, ascorbate anion (vitamin C) and cellular energy production (the Krebs cycle), which makes NADH, which converts to NADPH, which recycles the oxidized glutathione and oxidized ascorbate back into their reduced (un-oxidized, anti-oxidized) forms. This requires high levels of vitamin C and tiny amounts of the trace element selenium. Not only are children not supplemented with C during infection, some have deep metabolic syndromes that minimize their energy-productive capacity. One sign of this is obesity; carbs cannot get burned so they convert into body fat. We have an epidemic of childhood obesity, the causes of which are being either ignored, or treated with platitudes that have no basis in the actual mechanisms involved. Energy production capacity declines on an

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average of one percent every three years in aging humans, so this is a problem that is more common in the elderly and infirm than adults and children. But the markers of energy pathology in our children are escalating to unprecedented degrees, autism, learning disabilities, chronic health issues, allergies, upper respiratory infections, and more. So when you hear on the news that Covid-19 is an unprecedented viral risk, consider if it’s the virus, or is it that we are now unprecedentedly susceptible.

I am writing about this kind of stuff (1) because I believe that humans are rapidly losing their God-given healing powers as we have moved towards civilized lifestyles and away from Mother Nature, and (2) because this is a reversible phenomenon that public-health officials cannot shut down at the point of a gun.

We have the power to cultivate the sun and take vitamin D supplements. We have the power to alter our diets to reduce refined foods and carbohydrate foods, eat organ meats, and practice intermittent, partial and selective fasting. We have the power to turn off the news and cultivate our spiritual natures. We have the power to grow vegetables and have produce (meat and veggies) delivered to our doors by nearby organic farms. We can buy selenium, vitamin A, zinc lozenges, transdermal copper cream, vitamin D, kilos of vitamin C, nattokinase, vitamin K2, non-homogenized milk, fermented foods of many kinds, and wearable tech that gives us ongoing feedback regarding our energy pathways, our sleep quality, our autonomic balance, and our cognitive functionality.

— Or not.

The coronavirus is giving us a message. Shape up or ship out. The media want you to believe that it’s all about the virus and that our only prayer is a vaccine. That is not true in the least. There is massive research to show that there are many powerful natural anti-viral self defenses within reach. And those “renegade” (enlightened) doctors who talk about all this are being censored by Facebook and Google, and some of those who are practicing it have been raided by the FBI.

The land of the free and the home of the brave?

**Quora: Reset your Immune System?**

Original Question: *Can you reset your immune system?*

Yes. In many ways.

One that is likely most unappreciated is to restore a reducing environment through the IV administration of a very large amount of vitamin C. Vitamin C is the primary extracellular reducing agent. Intracellularly, it’s glutathione.

The humoral immune system (antibodies, white blood cells, cytokine cell-signaling agents, antigen presentation mechanisms, etc.) operates in the extracellular environment. Antibodies react to oxidative stressors and relax when the body is highly reduced (the opposite of oxidized). Cytokine storms are triggered by out-of-control oxidative stress.

The high concept is redox-buffering for immune modulation. Redox is a contraction of REDuction and OXidation. Reduction is the opposite of oxidation, which makes it hard for people to easily understand. What’s the opposite of a burning fire? What’s the opposite of rusting iron? What’s the opposite of a browning apple slice? Well, the part we do not see is the reduction of atmospheric oxygen by the wood in the fire, the reduction of oxygen in the air by the iron that is rusting, and the reduction of oxygen in the air...

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by the vitamin C in the exposed surface of the cut apple. So every oxidation is accompanied by an equal but opposite reduction. That’s why biologists, chemists and physicists call it redox. Calling it oxidation or reduction just depends on what you are looking at. If you look at the apple, vitamin C is being oxidized. If you are looking at hydrogen peroxide in a mouthwash, it’s being reduced by the microbes in your mouth.

Unlike pH (acidity and alkalinity), where we live in the middle, close to “neutral,” we live in a highly reduced state. In this state, our immune system idles. It’s waiting for something to happen. That “something” is oxidation. That something is a skinned knee from falling off a bicycle. That something is a cut from a bougainvillea or rose thorn. That something is an infection from a bacteria, fungus or virus. When an inner tissue is exposed to the air, oxygen causes oxidation. This triggers immune cells. When an antigen from a thorn or poison oak triggers an antigen-presenting cell, cytokines are released, which tell other cells how to release reactive oxygen species (ROS) to fight the infection. The cytokines also trigger vasodilation and vasoconstriction to help get blood to the site of an injury or infection, or shut it down to prevent blood loss. It’s a beautiful and adaptive system where oxidation turns on immune reactivity and reduction turns it off. Since we are reduced when healthy, it works like a charm: temporary oxidative stress when we get sick, and back to reduced when we recover.

Mitochondria produce 90% of the body’s total energy. People with normal, robust basal metabolic rate divert NADH (the primary reducing chemical in the body) from the Krebs cycle in the mitochondria to recycle antioxidants like glutathione and vitamin C (ascorbate anion) to keep the body reduced. Think of it as a refrigerator (reduction) to keep the body cool from the “heat” of oxidation from oxygen and infection. The NADH that is not diverted to reduce the body goes on to generate ATP (chemical energy or anti-entropy) that activates enzyme activity and drives “metabolism.” When that energy-production system falters, our immune system remains in a slightly activated state, and we are at risk of autoimmune diseases, degenerative diseases, shallow sleep, and impaired healing. In 2020 language, these hypometabolic conditions are called “pre-existing conditions.”

Vitamin C carries reducing power. When the body is flooded with vitamin C, reducing power is “topped up.” But vitamin C does not shut down oxidative stress. Vitamin C has a catalytic role in producing ROS. It’s called Fenton chemistry, and you can look it up. Basically, most of the reducing and oxidizing chemicals in the body have paired electrons that keep them from easily reacting with each other. Vitamin C, when combined with either iron or copper, unpair those paired electrons to make them react easily. This is the basis for the immune system’s ability to produce superoxide, hydrogen peroxide and hydroxyl radicals. It’s also how we make other cell-signaling molecules. So when we flood the body with vitamin C, we are promoting both oxidation and reduction at the same time. The oxidation part of it makes the immune system work better, and the reducing part of it also makes the immune system work better. The immune “on” and “off” switches become integrated, better coordinated and resistant to the “runaway” of the cytokine storm.

This might be called a “reset” of the immune system.

Fabulous question. I hope my explanation does the subject justice.

**Quora: What’s a disease that has been recently cured?**

The reversal of Alzheimer’s disease is likely the most surprising. Up until recently, Alzheimer’s disease was universally considered a terminal diagnosis. Drugs and cholinergic nutrients were merely

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356 9 July 2020. [https://www.quora.com/Whats-a-disease-that-has-been-recently-cured/answer/Steven-Fowkes](https://www.quora.com/Whats-a-disease-that-has-been-recently-cured/answer/Steven-Fowkes)
ameliorative, and only briefly so. But this has changed in the last ten years with many dozens of cases of Alzheimer’s disease being reversed and many hundreds of cases in mid-process. The mechanisms are known and protocols are in place.\textsuperscript{357} So it’s a scaling-up challenge. It’s not that the triage and protocols are simple. Hardly. They are devilishly complicated in clinical practice.\textsuperscript{358} But at this time, cases are being reversed with regularity. Even longstanding and severe cases.

I can also mention Covid-19 infection as a disease that is also being “cured” by redox therapy. Cure is in quotes because it’s a value-dependent and context-dependent meaning that has even more layers of connotation. Viral diseases are endemic and seasonal, so even if you got cured from the 2017 flu, you are still susceptible to both of the 2020 flus. Same with coronaviruses in general, and Covid-19 in particular. The “cure,” if you can even call it that, is merely temporary (months or years) and you can “catch it again” just like we catch colds and flus year after year.

The media and public-health officials may still be telling you that Alzheimer’s disease is uncurable and that there is no reliable or successful treatment for Covid-19. They are wrong. Most of those people truly believe what they are saying, but they are still wrong. Such beliefs may self-justify the censorship of those individuals who are using these protocols successfully, but they are still wrong.

What this means in reality is that the government has the power, right now, to drop the death rate from Covid-19 to as low as they want politically.\textsuperscript{359} There are doctors doing this, in hospitals and private practice, in the USA and in China, who are more than happy to share their protocols to make this happen on a national and worldwide basis in less than 30 days. But it will not happen because of the belief that it cannot be done.

Belief is a powerful thing that can be either a curse or a blessing.

What do you believe? And what beliefs would you be willing to give up to either reverse Alzheimer’s disease or survive Covid-19 infection without masks, social distancing, fear or high expense?

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\textsuperscript{357} Read Dale Bredesen’s book, \textit{The End of Alzheimer’s} (Avery 2017) for a top-down view of the treatment triage process. He lists 3.5 different “types” of Alzheimer’s disease, the 0.5 being an issue of overlap. This is not unrelated to this book’s topic; the underlying factors in Alzheimer’s disease are the same as the pre-existing conditions for Covid morbidity and mortality. Alzheimer’s disease is a steady-state failure of the redox-buffering system, which is almost the same thing as an acute failure of the redox-buffering system for acute viral infections.

For a mechanistic, bottom-up view of the pathology of Alzheimer’s disease, watch my nine-part YouTube series on Reversing Alzheimer’s Disease (https://www.youtube.com/watch?v=j1FmK4582mA&list=PL620DC3CA557284EB) and my second Tech-Talk presentation to Google employees: https://www.youtube.com/watch?v=PBTr9iQQ4&t=310s.

\textsuperscript{358} Surviving Covid-19 and reversing Alzheimer’s disease are comparable difficult. And simple. Both involve 90% of the same pre-existing conditions. That’s complicated. And they both have the same underlying redox failure. That’s simple.

\textsuperscript{359} This statement was first made in a presentation (https://www.youtube.com/watch?v=216F71qwURg) to the Silicon Valley Health Institute on 21 May 2020 shortly after I decided that the governmental stonewalling against life-saving redox-buffering treatments should be actively obstructed by all conscientious citizens. Two months earlier, a similar talk, What the CDC is Not Telling You, was presented to the Foresight Institute: https://www.youtube.com/watch?v=4iga-Jfuye4&t=49s.
Why is it difficult to reduce or prevent the spread of Covid-19? 360

Many will take the lazy way out and blame it on the President or the idiotic public-health policy. Even though those issues played a role, the intrinsic properties of the SARS-CoV-2 virus is independent of such political or ideological foibles.

Covid-19 spreads so easily because the SARS-CoV-2 virus is optimized for infecting humans. It is optimized at the receptor level (ACE-2). It is optimized at the enzyme level (furin activation). So it infects humans with an unprecedented efficiency. If it were not for its comparably mild lethality, when considered in the context of, for example, hemorrhagic viruses, it would be an efficient bioweapon. It’s only a couple of times more lethal than the bad flu strains that hit us regularly.

Another aspect of the spread of Covid-19 that has quite a bit to do with politics and public-health policy is the susceptibility of the world’s population to viral infections. If you look at the Covid-19 epidemic from a public-health perspective, you see that it is those with pre-existing conditions who are dying. Many believe that “healthy” people are also dying, but that is simply not true. Pre-existing conditions are like an iceberg, there are the ones above the waterline that you can easily see, like age, diabetes, cardiovascular disease, age, kidney disease, age, being male, being black and age, but there are vastly more under the waterline that are much harder to see, like insulin resistance, vitamin D deficiency, leptin resistance, vitamin C insufficiency, poor selenium and zinc status, type-II sequestration of iron, copper and zinc, mitochondrial senescence, and a host of metabolic disorders from impaired ability to conduct beta-oxidation, enter ketosis, or engage autophagy to increased toxic burdens of heavy metals, arsenic, fluoride, halogenated biphenyls, phthalates, glyphosate, and more. You might think that all these would be considered public-health issue to be dealt with, but you would be wrong. At least as the dealing-with is concerned.

The good news is that every single one of these pre-existing conditions are treatable. Anybody who has them can mitigate them if they listen to the right people. But those people are not in the FDA, CDC, NIH or WHO. And they are not in the censorship departments of Facebook and Google.

Let me give you two examples for your consideration:

First, Facebook and Google consider any mention of vitamin C and Covid-19 to be false information to be censored. Yet vitamin C has an 80-year history of treating the worst viral infections on the planet, from colds and flus, to super flus and polio. In a recent study conducted in three hospital ICUs in Wuhan, China, intravenous vitamin C cut the death rate for the most seriously infected Covid-19 patients in half. This was a double-blind, pilot, placebo-controlled study of just over 50 people. Nevertheless, cutting the death rate in half is an unprecedented finding compared to the other drugs being currently favored. So Facebook and Google are actually “doing evil” because of their abject inability to tell truth from fiction.

Second, look at the data on vitamin D. Although many doctors are correctly realizing that vitamin D deficiency is massively widespread and that doses of 5,000 IU per day is the average supplement to get to healthy levels, public health officials still think that 1,000 is the top end and that 2,000 is sticking your neck out. Then along comes a Spanish hospital study where 6,000 IU per day is the treatment for the first week and 3,000 IU per day is sustained for the duration of hospitalization.

The results:

Of 50 people on vitamin D, one was admitted to the ICU and none died.

Of 26 people without vitamin D (it was a 2:1 randomization), 13 were admitted to the ICU and two died.
2% ICU versus 50% ICU.
Zero deaths versus two deaths in half the number of patients.

In my book, I describe several limitations of both of these studies. However, the writing is on the wall. When you look at the entire body of published literature, the evidence is damning. Vitamin D and vitamin C (and selenium, zinc, vitamin A) are potentially the best of the best treatments possible, yet you are not allowed to take either (or any) of them with you into 99% of US hospitals. They are confiscated. Public-health officials not only are failing to deal with the epidemic of pre-existing conditions, they are in denial of their very existence. And they have persecuted medical doctors who have spoken out (1) for these options and (2) against these atrocities. The American Way? Being fired for telling the truth? Has it really gotten that bad?

In 20:20 hindsight, I think we will find that lockdown and social coercion were entirely the wrong policy. Maybe if it had been temporary for the spring and suspended for the summer (the anti-flu season), with summer-school as an educational compensation policy. But no, once committed, we cannot admit to being wrong. Human history with lipid-enveloped viruses like influenza and coronavirus is that they are endemic. Every year, we have a flu season when lipid-enveloped viruses infect the vast majority of humans on the planet. Year after year. Wave after wave. Decade after decade. This is the norm.

Now, suddenly, everybody thinks that this particular lipid-enveloped virus is different, that we can stop it from spreading, despite the fact that it is uniquely structured to infect humans better than any previous virus. This has never been done before with lesser viruses, nor is it likely to work now with this super-spreading virus. Lipid-enveloped viruses are something that we have had to live with for all of known history. And those who escape SARS-CoV-2 exposure now will face exposure every coming flu season. Current evidence from Spain, France and the UK suggests that the next flu season has already started.

So my suggestion is to consider an alternative or adjunctive strategy to avoidance. Identify and deal with your pre-existing conditions. Don’t ignore them when your doctors says, “you are fine.” Take a pro-active stance towards reducing your viral susceptibility, so that you can not only survive SARS-CoV-2 when you eventually get it, but so you can live a longer, more productive, happier life without the fear and isolation that so many people want you to embrace.

Learn what the WHO, CDC, NIH and FDA do not want you to know. Learn what your pre-existing conditions are. Then, research how to mitigate and reverse them. Finally, do something to actually reverse them.

It’s far from easy. But your life and the lives of your loved ones are worth it.

(Quoran response) Interesting. What are your thoughts on wearing masks in public to minimize spread of infection? It seems that some Asian countries have kept their infection and fatality numbers low, while also going about day to day business relatively normally, due to almost everyone wearing masks. It didn't really work here in the US because of noncompliance, it seems.

The Asian mask myth is alive and well, but mask-wearing is not particularly effective. In the absence of good data, the justifying reason is “it’s better to be safe than sorry.” But when you look at the numbers, mask wearing is not very effective. I’ve looked at the studies, and the ones most cited are very badly done and their “findings” egregiously exaggerated. Most masks do not fit that well, and even ones that can fit well are not fitted properly nine times out of ten, plus some of the best masks for protecting the wearer have exhaust valves that spray the exhale on those people around them. There is just too much blow-by for both
inhale and exhale. The masks actually concentrate viruses on their interior and exterior surfaces and provide the moisture that keeps viruses from being destroyed in hot, dry air. They may be way better than nothing in crowded urban settings, but in open air in summer in full sunlight, they may actually be quite counterproductive. Plus, if you understand that SARS-CoV-2 is here to stay, the public-health goal should be NOT to prevent infection, but should be to delay infection during the flu season and encourage infection during the anti-flu season (the summer). There are two compelling reasons for this. First, the virus attenuates in the summer and people are less susceptible during the summer. Death rates from lipid-enveloped viruses bottom out in June, July, August and September (in the northern hemisphere). In other words, the summer months are the time when Covid-19 is mildest. Second, the winter flu season is when hospitals are most crowded with viral-disease patients, so summer infections offset the seasonal peak demand for medical services. However, public policy is not aligned with the facts. The stated goal IS to prevent people from ever getting infected with SARS-CoV-2. Frankly, I do not believe this is possible. It was and is possible to delay infection, a season for sure, and maybe a year at best, but the valid reason to delay is not to prevent infection indefinitely, it was to give time to the medical profession to figure out the best way to treat Covid-19 disease, and of course, how NOT to treat it. I think that most people feel that this has been accomplished, but I am certain that they are wrong. The best ways to treat Covid disease requires, to mention most of the top ten, inclusion of vitamins D3, A and C, zinc, selenium and some kind of anticoagulant (heparin or nattokinase). At this time, only a few of these are accepted as standard therapy, and as I mentioned, the hospital staff strip you of those supplements even if you have a prescription for them. (Administrative law strips you of all your Constitutional rights, including medical rights.) The only remaining reason for delaying infection is waiting for a vaccine. That, also, is likely wishful thinking. Although the effort devoted to vaccine development is ten times more now than following the 2002 SARS outbreak and the 2012 MERS outbreak, all of those previous efforts, although extensive, failed. Some merely failed functionally. Others failed catastrophically. Pharmaceutical companies are drooling about the potential profit of selling SARS vaccines, but if natural immunity to SARS-CoV-2 lasts only for 2–10 months, why would vaccines do any better? And that assumes that (1) the vaccines actually work, and (2) that there are zero viral-interference effects. There is good reason to doubt both. — I wish it were easier to answer some of the simple questions simply, but when science and politics are mixed, things get complicated, especially when the malefeasance of scientists and scientific institutions rises to match that of politicians and governmental institutions. — I hope this answers your mask question with sufficient context. If you want more information, feel free to read my book. The fourth release is available as a free PDF download.

(Quoran further response) I appreciate the reply. It’s definitely food for thought. The part about supplementing with certain vitamins and minerals makes sense to me, and I take steps regarding that. I tend to disagree about wearing masks, but I’m just an ordinary person with no background in this kind of thing. I also would rather avoid catching it for as long as possible, so I guess I am hoping for a relatively effective vaccine, plus as you mentioned, hopefully we’ll continue to learn more about how it affects people and how to treat it better. I’m more concerned about acquiring a long term health issue from it, not necessarily dying from it. I’d just rather not add some new ailments to what I already am dealing with now (or worsen them, who knows). Thanks again for the reply. I find your content here to be interesting and educational.

Avoiding catching it is, I think, the ideal strategy only in situations where the pre-existing conditions in an individual are intrinsically difficult to treat effectively, or on a timely manner. The extreme elderly with diabetes and approaching kidney failure, for example, would require tens of thousands of dollars of cutting-edge treatment with stem cells or exosomes and very closely supervised dietary modifications that would
be economically out of the question for the vast majority of such people. Certainly, the government would never pay for that. So quarantine is the ideal strategy. But if a person is young and relatively fit, is capable of entering ketosis in three days without distress, is willing to take supplements to bring their vitamin D₃ above 50 ng/ml (125 nmol/L), their selenium into the top quartile, has zinc and vitamin A in their medicine chest, it’s much better to plan a Covid-19 infection in July or August. Assuming, of course, that you could do such a thing. And then begin active treatment on exposure (the earliest possible time) and ramp treatment up in proportion to the symptoms. — Those long-term consequences that people are experiencing are not intrinsic to Covid-19, but rather to failure to treat it properly. Micro-clots do not form if your redox-buffering system is robust and you prevent coagulopathy. That can be accomplished by (1) taking high-dose vitamin C, (2) taking low-dose NAC, (3) having a broad spectrum of bioflavonoids on board, (4) not having a type-I or type-II copper deficiency (or using transdermal copper with either kind of copper deficiency), and (5) having a robust basal metabolic rate (or being in ketosis). Fibrosis does not take place if your collagen maintenance stays strong and your coagulation is dialed in. Collagen maintenance depends on tissue vitamin C, tissue copper, tissue silicon, and zinc-mediated protein synthesis in the cell, which takes moderate quantities of three amino acids, proline, glycine and lysine. Coagulation in a hospital is prevented by heparin administration, which you could ask for if not automatically given. But at home, you can use nattokinase, lumbrokinase, serrapeptase, and digestive and systemic enzyme therapy. These aspects of Covid-19 therapy are known right now. If you anticipate being in a hospital with Covid, there is good reason to want to postpone your experience. Hospitals are way behind the curve in adopting these biological therapies due to their myopic focus on proprietary pharmaceuticals and their entrenched political aversion to vitamin C. But if you are going to ride out a Covid infection at home, all of this is totally within your control. — But please do not use this minimalist summation as a guide. There are aspects to facilitating innate immunity in the earliest stages of Covid-19 disease that I did not even mention. And knowing the right way to use zinc and hydroxychloroquine is likely important. The proper treatment of Covid-19 is not only depending on universal things like coagulation, redox buffering and vitamin C, which likely apply to 99% of the population, but also to things that are not so universal and which may or may not be an issue in a particular person. If it’s a nutritional issue, it is usually OK to treat it whether or not it exists as long as it is short term. But what about mercury or thallium toxicity, glyphosate load, glucose-6-phosphate deficiency, the presence of thiaminase bacteria in the gut microbiome, leaky gut syndrome, or trans-fat and pesticide residues in fat storage that get released when ketosis starts and before autophagy engages? Humans are intrinsically complicated.

If you believe that masks are effective, then they are. It is an intrinsic part of human nature that belief has biological manifestations. It is the basis of the placebo response, and might be the basis for many spontaneous “miracle” cures. With a belief, the facts do not matter. This is because the facts are only important in one’s thinking mind and, truth be told, the subconscious and emotional minds rule. This is why we are not just what we eat, drink and breathe, we are what we think, believe and value. So mask wearing becomes a personal value which can range from protecting yourself (a Western value) to protecting others (an Asian value), and pretty much anything else that it may come to mean. This is the basis of mask-shaming behaviors on both sides when tolerance and civility are bypassed due to personal and religious beliefs that are assumed to be universally true (for the other guy). Some people believe that drugs are evil and would not take hydroxychloroquine for Covid-19. I do not generalize and actually take two pharmaceuticals along with my nutrition supplements. And I have BHT and DMSO in my medicine chest. But, however your believe, there is a likely adverse consequence to operating in opposition to your beliefs. This is, I think, part of wisdom. And realizing that operating in harmony with your own beliefs is personal and utterly unrelated to what others do is a sign of enlightenment. Thanks for the conversation!
(Quoran final response) *Thanks for the interesting viewpoints! I'm always open to broadening my knowledge base, especially if it influences my health.*