The BHT Book: A practical guide to resolving viral disease

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This book is an evolving work.
Organizational and editing suggestions are welcome.
Personal stories are invited.
About-the-Title Preface

This book offers a biologically sustainable solution to chronic viral disease.

That solution involves shifting your metabolism (i.e., correcting a metabolic imbalance that makes you vulnerable to viruses) by

1) taking a drug, BHT (butylated hydroxytoluene), an FDA-approved food preservative, and/or
2) using a “natural” combination of foods, supplements, hormones, and/or lifestyle factors.

The metabolic shift is experientially subtle. Many people cannot tell that there is a change, other than through a cessation of symptoms. But some people will see other changes, like improvements in skin quality, reduced asthma symptoms, improved wellbeing, decreased sensitivity to cold weather, better sleep, faster recovery from colds and flus, faster wound healing, greater stamina, reduced PMS symptoms, and/or a decrease in severity and frequency of migraine headaches.

The metabolic shift is sustainable. In other words, it is fully in accordance with “natural” processes of biology, which minimizes side effects that might otherwise be associated with therapies directed only at symptoms. When done properly,¹ this metabolic shift does not induce compensation by body homeostatic mechanisms that might tend to try to restore “balance.”

The program is easy in that is can be as simple as taking one to four 250-mg capsules of BHT every day (90% effectiveness). Or it can be a bit more complicated, like combining the BHT with a few extra dietary supplements (99% effectiveness). Or, it can be even more complicated, by not taking BHT and relying entirely upon nutrition, exercise, supplements and hormone replacement therapies, probably with comparable results. Because human metabolism is complex, the program that you choose to implement can also be complex. But it can be simple and easy, if you need it to be simple and easy.

Bottom line, it doesn’t have to be any more complicated than what actually works for you.

You can accept the results by the mere cessation of symptoms, or you can verify the results by before-and-after PCR testing.²

The program is broadly applicable to many viral conditions. It has been successfully applied to recurring herpes, shingles, herpes encephalitis, influenza, raging intestinal CMV, and hepatitis C.³

The program is inexpensive in that a year’s supply of BHT typically costs about $10 (in bulk) or $50 (in capsules). The supplements and hormones could cost about $100 to maybe $1000 per year. The dietary changes might add a few hundred dollars, but could save a few hundred dollars. Such costs are inexpensive compared to the cost of medical insurance (assuming that you’d have coverage for non-drug therapies for viral disease), out-of-pocket medical expenses for orthodox viral therapies, and the cost of medical expenses associated with the underlying metabolic imbalance that is almost always associated with chronic viral disease. This would also include asthma, migraine headaches, autoimmune disease, hiccups, panic

¹ If a metabolic shift is produced by taking high doses of one agent while a deficiency of another similar agent is present, there is likely to be some form of compensation that will make the shift nonsustainable. This is explained in the section on metabolic balance. For example, if very high doses of selenium (an antiviral influence) are taken when hypothyroidism (a pro-viral condition) is also present, the selenium will have an attenuated or unstable effect on viral virulence. In other words, the “deficiency” of thyroid hormone is sustainably corrected only by actual thyroid hormone, not selenium over-compensation.

² PCR stands for polymerase chain reaction. Unlike antibody testing, PCR testing identifies viruses by their DNA. As a result, the PCR test measures current (at the time of the blood draw) viral replication (shedding into the blood stream). Antibodies linger long after an infection has cleared, so a positive antibody test does not distinguish between an active infection and a past infection that has cleared.

³ It has the potential to be applied to swine flu, bird flu, SARS, ebola virus, and other viral hemorrhagic fevers. I just have not yet received any communications about these other viruses and viral diseases. For a full list of lipid viruses and lipid-viral diseases, go to page 49.
disorders, chronic fatigue conditions, fibromyalgia, cardiovascular disease and known risk factors for the majority of cancers.

It will take the rest of this book to impart the details of how, what, and why.

Please keep reading.

2012 Note

As of this edition of the book, footnoting is now used to separate the more technical information from the main text of the book. This change is an attempt to make the reading of the book easier for those readers who are non-technical (i.e., most of the people of the world). If you are not technically inclined, simply ignore the footnotes, unless your curiosity sneaks up on you and twists your arm behind your back. If you are technically inclined, you can enjoy the added technical information in the many footnotes included in this edition and future editions.

There is also a more technical section on metabolic balancing on page 54.

2014 Ebola Addendum

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, 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.

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 (see page 52). Swine flu is a lipid-enveloped viral disease, and in Alan’s case, it progressed to coma, a respirator 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 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.

4 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.

5 This is what happened to Allan Smith in New Zealand after contracting swine flu and falling into a coma. See further information and footnotes on pages 43 and 44.
In hours, Alan Smith woke up.
In a day, his lungs cleared.

Despite his recovery, the hospital administrators stopped the vitamin C. They “reasoned” that his recovery could not have been from the vitamin C and it was no longer ethically permissible to give a non-dying man a worthless therapy. Alan lapsed back into a coma. The family had to smuggle vitamin C into the hospital to save Alan’s life.

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.

For more details on ebola and selenium-treatment options, see the Ebola Update on page 52.

2015 Measles Note

In writing this book, I have featured herpes as the prototypical viral disease to illustrate the anti-viral information and techniques of this book. In some ways, this has been unfortunate.

First, the “herpes” message is off-putting to some people who would otherwise have a strong interest in antiviral therapeutics. By being viewed as a sexually transmitted disease, it has an associated stigma by people who disapprove of other’s sexual expressions and choices.

Second, the BHT message is off-putting to other people because it is a preservative and not a natural substance. Regardless of the fact that much of this book is metabolically and nutritionally oriented, the title is about BHT.

To provide a counterexample to these two liabilities, I offer the following special message regarding measles infections in children. This message deals with the known beneficial effects of vitamin A—one of the antiviral nutrients discussed in this book—on the clinical course of severe, life-threatening measles infections. When very high doses (approximately 300 times the RDA) of vitamin A were administered in routine clinical practice, the hospital stay was decreased by 20%, the need for intensive care was reduced by 60% and deaths were cut by 68% (Hussey and Klein, 1993). No adverse effects were observed. For more information, see page 53.

2016 Alzheimer’s Disease Note

The appreciation-process note from me on the following page is in no way limited to viral diseases. If you know somebody dealing with mild dementia or early-stage Alzheimer’s disease, or even somebody dealing with a relative with full-blown Alzheimer’s disease, check out the nine-part video series on “Prevention and Reversal of Alzheimer’s Disease” on YouTube (www.youtube.com/user/swfowkes/videos). It takes 80 minutes to watch all nine parts (each is less than ten minutes long). To watch them all in order, select the 1-9 playlist, or use the following link to that playlist:
https://www.youtube.com/watch?v=j1FmK4582mA&list=PL620DC3CA557284EB

Contrary to public belief and the pronouncements of pretty much every mainstream medical organization on the planet, Alzheimer’s disease is not only reversible, it is relatively easily reversible. This may be shocking but have a little faith (and some considerable skepticism) to suspend your disbelief for long enough to read my summary on page 58.

You can also read all about it in Dale Bredesen’s book, The End of Alzheimer’s (Avery 2017).

Many of Dr. Bredesen’s public and medical talks are online for your edification.
2017 Note on Medical Testing without a Prescription

The availability of medical testing has gone through a revolutionary change over the time course of this book and many testing-service brokers now handle the medical-prescription requirement for you in a transparent way. So all you have to do is go online and search out these companies (there are a dozen) and click on the tests you want to order.

There are still certain specialty-testing labs that are not yet covered by these services. But pretty much any test offered by Quest Diagnostics or LabCorp are now essentially available over the counter.

Once you have purchased the test, you will be directed to an office geographically close to you where your blood will be drawn, or your urine sample collected.

2020 Note on 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 may be, 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 47 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.

Please read on.

Learn the ins and outs of viral diseases so you can best defend yourself.
What if they gave a cure and nobody came?

(an appreciation-process message from Steve)

This is the page of the book where I ask you for money.

This book contains much of the content from an earlier book, Wipe Out Herpes with BHT, written back in 1983 by John A. Mann and myself. Back then, we sold it for $5. This book also contains 99% of the content from a supplementary pamphlet, the BHT Toxicology Report, which was updated every few years since 1984, and which also cost $5.

This new, rewritten book incorporates considerable additional information and perspectives gleaned from 40 years of scientific inquiry and personal health investigation, and countless conversations with clients, readers of earlier books, and random people who heard about BHT, tried it for themselves, and tracked me down. What is all that worth? What is that worth to you?

Experts say that this book is worth what you think it is worth. Right now, it’s already worth the time and trouble that you have invested in tracking it down. For most readers, this is not trivial. But, more importantly, if you invest in reading it, it will become worth much, much more. How much is your time worth? What would you rather be doing? And when you implement some of the information contained within this book, your investment will grow higher. It takes considerable effort to develop new habits, to break old routines, to actually have to think about things that used to be automatic. Change? That’s a high investment indeed.

Traditionally, in reading a book, you have to invest your hard cash before you get to read a book.

The “danger” in this freeware-book approach, according to academic psychologists (who are supposed to know), is that you may not value this information as much as you would if you had to pay for it in advance. In addition, they opine you would value it more if you paid $69.95 than if you paid $6.95. Maybe the average person would value it more if a one-minute summary of the information were related by a 500-per-hour, middle-aged man (sexism intended) in a white lab coat with a stethoscope draped on his shoulders. This is the wisdom behind the adage, “it’s worth what you pay for it.” So I have three suggestions.

First, be generous with investing your time and effort in understanding the options presented in this book, and in applying your chosen approach to your personal issues with viruses. This is an investment on your part that is as real as the cost of any cash you might have paid for this book or for BHT, or dietary supplements, or the effort you invest to change your lifestyle. Appreciate your investment. Thank yourself for making it.

Second, be true to your values. If you need to check out what you read here, do so. If you need to ask me a question, do so. If you need time to get comfortable with this approach, take it.

Third, when you get results, send some money in appreciation of the benefits you see. Just send a small portion of the value you receive, whatever you think it is, within the practical limits of what you can afford. If you don’t have discretionary money, consider non-monetary appreciations, like 1) telling your story (anonymously, or not), and/or 2) giving the book away to other people who might end up appreciating it like you do.

And don’t forget to thank the person who told you about this book.

—Steven Wm. Fowkes, February 2020

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Introduction
This book is written for several audiences.

First, it is written to inform herpes sufferers and health practitioners of the therapeutic use of BHT (butylated hydroxytoluene) against herpes and other viruses, as well as other, non-viral conditions (like heart disease, skin aging and free-radical pathology).

But it is also 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**: vitamins A, D₃, B₆ and B₁₂,
- **amino acids**: cysteine, N-acetylcysteine (NAC) and glutathione (a cysteine-containing peptide),
- **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), and
- **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. 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).

Of course, there is no reason that the BHT and non-BHT approaches cannot easily be combined into an integrated anti-viral program. In fact, that is the reason why this book is being written this way.

The remainder of this chapter deals with “setting the stage” for the following nuts-and-bolts information.

In a hurry?
If you are impatient to get on with the practical aspects, skip to the section How Well Does BHT Work? (on page 12) or How to Use BHT (on page 25). Or if you want to avoid BHT and use only nutrients, skip to page 31, the beginning of the section on The Metabolic Hypothesis.

If you want a list of lipid-enveloped viruses and the diseases they cause, skip to page 59. If you are already taking BHT and want to fine-tune your program, go to page 26. If you want to dissolve BHT in coconut oil for transdermal delivery, go to page 27. The table of contents above can guide you to other sections not mentioned here.

World View, Politics and Ideology
What could how we think have anything to do with BHT or viruses?

Cognitive dissonance (thought conflicts, or value conflicts) can disrupt body systems through the mind-body connection (the neuroendocrine regulatory systems). So in a very real sense, you are what you eat, you are what you think, you are what you feel, and you are what you believe. Some readers may have a negative attitude towards BHT. After all, BHT is a food preservative, and many people think or feel that preservatives are bad. BHT is a xenobiotic (a drug-like substance foreign to life, both plant life and animal life), and the very thought of using a drug may be repugnant.⁶ This is very relevant to the topic at hand.

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⁶ BHT has now been found in multiple plant species, so it might now be considered natural.
Some readers may have read that BHT is a carcinogen (it isn’t), and that it causes birth defects (it doesn’t). The key point is not whether these views are true (as some say), or not (as I say), but whether you believe they are true—or merely harbor doubts about either “truth.” The point that I’d like you to understand is that we all are biologically compelled to be “true and faithful” to our thoughts and beliefs, whatever they may be, or may become in the future. Some very officious organizations are saying some wrong, prejudicial and inaccurate things about BHT. If you hold those organizations in esteem, your beliefs may undermine the effectiveness of BHT.

Some people reading this book may carry the common belief among long-standing health-food consumers that the FDA is hopelessly and irredeemably corrupt. I believe that myself. However, I do not believe that all FDA-banned dietary supplements are likely effective, and that all FDA-approved drugs (and preservatives) must be bad. I personally take two xenobiotic substances (deprenyl and piracetam) on a regular basis for health-enhancement purposes, and one just happens to be FDA approved. (I only use BHT intermittently.) In other words, even a thoroughly arbitrary and capricious organization like the FDA cannot be trusted to be wrong 100% of the time. And even reputable and politically correct organizations cannot be trusted to be right all the time.

It is my hope that some readers may overcome significant prejudices against BHT, fostered by careless (or biased) organizations that have inadvertently (or purposefully) distorted the scientific/medical record on BHT.

It is also my hope that many readers will gain hope against the pervasive pessimism in modern Western medicine that there is nothing to do against viral infection but 1) take one or more of several toxic-and-almost-ineffectual pharmaceutical drugs, or 2) learn to “just live with it.” There is a better way.

Using BHT is a better way (see How to Use BHT on page 25 and Optimizing Results on page 26).

Not using BHT is a better way (see the Metabolic Hypothesis on pages 31 through 46).

These two approaches add to each other’s effectiveness.

The Logic and Emotion behind Changing One’s Mind

If you believed that BHT was a carcinogen before I told you otherwise, what would it take for you to change your mind? For some, it takes only a straightforward explanation: for example, that an erroneous scientific experiment that couldn’t be replicated was later contradicted by follow-up experiments. If you are in this group, you have not necessarily formed strong emotional attachment to this particular belief. For others, emotional attachment to beliefs may be substantial, or even intense. If you are in this group, it may take reading the scientific papers themselves, and tracking the evidence from start to finish before you will change your mind. Or it may just take time—time for emotional attachments to reform to new information. However it works for you, take responsibility for the process for yourself (whatever you need). This may also mean allowing other people to do it their way. Husbands and wives may have divergent cognitive orientations (“feelers” versus “thinkers”). This may also involve consensus-group decisions (i.e., parents making a decision for a child, or a family making decisions for an incapacitated family member).

Even after reading the new information in this book and re-forming your opinion about BHT and viral diseases, you may decide not to use BHT, and instead, to use a more “natural” or “biological” approach (see pages 32-36). Even if you’ve taken BHT before, and feel quite positive about the literature on BHT, you might decide to forgo BHT in favor of dietary supplements, dietary changes, or non-mainstream medical treatments that produce metabolic shifts that are closely similar to or parallel to those produced by BHT. After all, why resort to a drug when it is not necessary?

When John Mann and I wrote Wipe Out Herpes with BHT in 1982 and 1983, we both believed that BHT worked by a direct chemical action against the physical structure of herpes viruses and that BHT was a
unique antiviral substance in this mechanism.\(^7\) I no longer believe this. I believe that BHT’s efficacy against viruses is mediated by a concerted metabolic shift that can also be caused by countless other substances, like hypericin and pseudohypericin extracted from the herb St. John’s wort, and like vitamins A, D\(_3\), B\(_6\) and B\(_12\), and minerals selenium, sulfur, magnesium, calcium, strontium, vanadium, manganese and copper. I now believe that regular exposure to full-spectrum sunlight (red-enriched light at dawn and dusk, and blue-rich light at noon) and the cultivation of aerobic exercise also produce similar metabolic shifts to those produced by BHT. Indeed, there are a plethora of options for treating viral infection or viral susceptibility without the need to resort to BHT, acyclovir (Zovirax), famciclovir (Famvir) or valacyclovir (Valtrex). If mother nature is your lady, follow her.

### Information Sources

What you read in this book will be based upon either of two sources. Much of this information is founded on careful literature review of scientific and medical research that has been published over seven decades.\(^8\) Many aspects of this research still remain to be investigated, for reasons that will be discussed later.

A significant remainder of this information is based upon four decades of personal observations and anecdotal reports from many hundreds of herpes sufferers and a dozen physicians and scientists.

It has been a half century since the first reports of BHT’s use in treating herpes were published, and many tens of thousands of people have already used BHT to treat their herpes (and other viral diseases) with varying degrees of success. The most memorable “miracle” cases include 1) three cases of reversal of herpes encephalitis coma, 2) multiple cases of complete elimination of chronic (painful) shingles, 3) successful treatment of intractable diarrhea from gastrointestinal CMV infection in an immune suppressed organ-transplant recipient (with restoration of normal bowel movements), and 4) multiple conversions of hepatitis C infections (seronegative by both antibody and PCR testing).

In most of these cases, the beliefs of the “patients” that BHT was responsible were discounted by their attending physicians, who preferred to provide alternative explanations, like “the lab must have made a mistake” or “it was a spontaneous remission.” However, in one case of hepatitis C infection, there were a half dozen viral workups over two years involving positive PCR and antibody tests, and repetition of negative antibody and PCR tests after BHT use. Yet, still, BHT could not have been responsible.

### The Good, the Bad, and the Internet

There is a lot of bad information out there.

For example, during 2011 at least a dozen sites have repeated the error that “BHT contains toluene.” It may seem entirely obvious that since BHT is called butylated hydroxytoluene that it must contain toluene. But it does not. None at all. Zip. Nada. BHT can also be correctly called butylated para-cresol, but it does not contain any cresol. You could spend a lifetime trying to extract toluene or cresol from tons and tons of BHT and not get even a microgram of toluene or cresol. Vitamin E is also a derivative of toluene and cresol, but, similarly, you cannot obtain any toluene or cresol from vitamin E without a fully equipped organic chemistry laboratory and drastic reaction conditions that would destroy the vitamin E and convert it into toluene or cresol.

Why would there be hundreds of such pieces of bad information? Because there is one. Bad information is repeated just as easily as good information is. This may be especially true when the bad information is

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\(^7\) This viral-disruptive effect of BHT had been elegantly demonstrated in test-tube experiments in the late 70s. However, the technique involved conditions—the formation of microcrystals from solvent-water interactions—which are impossible to replicate in living organisms. This will be discussed in more depth later in the book.

\(^8\) My apologies for the lack of direct citations in many parts of the book. The original *Wipe Out Herpes* book was written without footnotes, and the *BHT Toxicology Report* was written with footnotes and references, so this re-written book is a blend of both. Some remedial footnoting has been done in the initial chapters. However, more remains to be done.
politically correct or self-serving on the part of the purveyor. The bottom line is that many purveyors of
information are not well qualified to judge the quality of the information they pass on. It is equally true
that many purveyors of product cannot manage their conflict of interest regarding the information they
provide. Caveat emptor. Let the buyer beware!

Even scientists repeat bad information. There are thousands of sites that pronounce that BHT is a known
carcinogen, or suspected carcinogen. Many of these sites are supposed to be reviewed by scientific,
medical and technical experts, yet they get it wrong. Why? Because the original source of the information
was wrong, and the subsequent correction did not get equal time. If a lie is repeated sufficiently, it
becomes the TRUTH (i.e., a “big lie”). So please cultivate at least a modicum of disbelief about what you
read, everywhere—including this book.

There is also a lot of good information out there that does not get the respect it deserves. Let’s face it,
scientists and doctors tend to be snobs. For example, if it isn’t validated by double-blind, placebo-
controlled studies, it is not real. Really? Any empiricist will tell you that an effect is either real or not
real, regardless of what kind of studies have or haven’t been done. US doctors tend to be snobbier than
foreign doctors. Lots of good information is dismissed because “it wasn’t invented here.”

I have spent much time investigating (and finding) fraud in modern American science. You don’t have to
believe me, but I have to tell you that it is not a rare occurrence. And it is getting worse in recent years.

Scientific and medical prejudices against Internet-derived information—good or bad—are severe.

In 2010, while preparing a talk for the Smart Life Forum (smartlifeforum.org) on “Prevention and Reversal
of Dementias and Alzheimer’s Disease” I found numerous stories in the popular press that “Herpes causes

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9 Stanford epidemiologist (and statistician) Dr. John Ioannidis (E-O-nee-dee) is showing that an alarming number of such
studies are invalidated within ten years of publishing, and that this is a generalizable phenomena across multiple disciplinary
boundaries. Somewhere between 50% and 90% of positive double-blind studies are found to be false. Interestingly, double-
blind studies also show an attenuation effect whereby the statistical significance decreases with each subsequent replication of
the initial results. Ioannidis uses the same statistical methods in his analysis that are used in mainstream science, so there is
something going on that is not yet understood. Ioannidis’ analysis has so far withstood peer review from vehement critics.
His results have also been replicated by independent researchers.

10 It is almost as if the skeptics are stating that reality somehow changes when studies are done, or that the efficacies that we
accept today were not efficacious back in the “bad old days” prior to their discovery. Obviously, they do not actually believe
this. But they talk and write as if they do, by stating with positive assertion that “unproven” therapies do not work, despite
their not having any actual evidence that they do not work. Many aspects of reality are reciprocal. But knowledge is not.
One can look back with 20:20 hindsight and state that the critics of 1) handwashing before assisting childbirth, 2)
homocysteine as a predictor of cardiovascular mortality and morbidity, and 3) sunlight exposure causing skin cancer were
wrong. That’s easy. But it is not easy at all to judge the efficacy of therapies in advance, prior to the completion of research
and the widespread clinical application of the therapy.

11 Doctor Professor Vladimir Dilmun presented clear evidence that insulin resistance increased risks of cancer back in the 1950s.
But because he was a Russian and published his findings in a Russian medical journal, his finding was ignored until it was
replicated decades later by US researchers, who repeatedly failed to cite his work or credit him for the insight.

12 Here’s a recent link (http://blog.pubpeer.com/?p=164) to a web site that was set up to expose minor problems in scientific
research, offering scientists a platform to point out “the finer points experimental design and interpretation.” But what they
have uncovered was far more serious, involving “questionable research practices” and “obvious misconduct.” Much of this
came to light only after they implemented anonymous posting on their website (only the website administrators knew the
actual identities of the posters), which assured fears of retaliation by criticized scientists, who have political and academic
connections, and who review government grant applications. With anonymity, they started getting widespread reports of
intentional fraud and outright manipulation of data from all levels of modern science. What this means is that modern science
has been corrupted by numerous pressures far beyond what scientists and regulators believed. It is no longer trustworthy.
Perhaps this aspect of science is what John Ioannides was statistically measuring (see footnote 9).
Alzheimer’s disease. The truth is actually the other way around. Alzheimer’s disease is associated with opportunistic virulence of herpes and other viruses, as will be amply detailed in this book.\textsuperscript{13}

I also found Pediatrician Mary Newport’s report on her husband’s case of Alzheimer’s disease on the Internet. This is an excellent report of the beneficial effects of coconut oil in not only stopping but actually partially reversing a case of Alzheimer’s disease, supported by clinical assessments and cited scientific papers documenting the possible (probable?) mechanisms. It was an information gem,\textsuperscript{14} and not available through any medical journal. And does it get any respect? No.

Please do not automatically believe scientific, medical and media authorities and automatically disbelieve independent sources and anecdotal reports. Both provide a mixture of good and bad information.

The trick is telling them apart, and even with a lifetime of training and effort, it is not easy.

Now, on to the meat of the book.

\textbf{How Well Does BHT Work?}

Some fortunate people respond phenomenally to even moderate doses of BHT. This might take the form of a week or two of BHT use, followed by months or years without flare-ups. For them, it looks like a miracle cure.

Most people merely get good results. This may take the form of 1) near-complete suppression of flare-ups with continuous BHT use, or 2) satisfactory control with intermittent use of BHT (like during times of “stress” or excessive sunlight exposure that would typically trigger an outbreak). Such viral-activating stresses might also include an on-the-job deadline, academic final exams, premenstrual hormone swings in women, chemical exposure to perfumes, paint solvents or adhesive fumes, or way-above-average sexual activity. Such stressors are largely anticipatable. So it is not too difficult to start taking BHT a few days before the stress is supposed to happen or a few hours after the stress actually happens.

Other stresses may not be anticipatable, like automobile accidents, emotional grief from an unexpected death, or receiving a tax-audit notice in the mail. However, BHT’s effect on metabolism is so rapid as to be fairly effective even when taken shortly after a triggering event.

Then there are stresses that are basically “invisible,” such as transitory mercury toxicity from eating fish, formaldehyde poisoning from walking into a “sick” building, or constitutional states of metabolism, like being estrogen dominant or hypothyroid/hypometabolic. Sometimes a viral flare-up is the first obvious sign of the problem.

For an unfortunate minority of people (approximately one in ten or twenty), even continuous high-dose BHT does not produce effective control of outbreaks.

What accounts for such differences?

\textsuperscript{13} Alzheimer’s disease is also called type-III diabetes. Although there is strong evidence that pre-diabetic changes (insulin resistance) are involved in Alzheimer’s disease, diabetic process has a strong effect on the body and a weak effect on the brain. This is because body tissues predominantly use the GLUT-4 glucose transporters, which are compromised by insulin resistance, and the brain predominantly uses GLUT-1 glucose transporters, which are not affected by insulin. But there are indirect ways in which insulin resistance and diabetes adversely affect brain metabolism, via loss of control of body antioxidant defense systems, which expose the brain to increased inflammatory consequences. Inflammation is a direct mechanism for Alzheimer’s disease. Inflammation is the mechanism by which dietary fish strongly reduces Alzheimer’s disease risk despite the fact that it is often the largest single dietary source for mercury exposure.

\textsuperscript{14} Mary T. Newport’s new book \textit{Alzheimer’s Disease: What If There Was a Cure?} was released late in 2011.
I believe that there are several biological factors that influence viral susceptibility (viral disease “caused” by the weakness of the host) and viral virulence (viral disease “caused” by the strength of the virus). \(^{15}\) (For more information about this difference, see page 31.) These may involve:

1) **metabolic imbalances,**
   a) pre-existing anaerobic/aerobic (anabolic/catabolic, alkaline/acid) imbalances (see page 32),
   b) measuring pH imbalances (cellular, tissue or blood) (see page 45),
   c) autonomic imbalances (sympathetic or parasympathetic),
   d) oxidative imbalances (fast or slow oxidizers),

2) **nutritional deficiencies or excesses,**
   a) iron overload (clinical or subclinical hemachromatosis, see page 37),
   b) zinc and/or selenium deficiency (see page 46),
   c) secondary copper deficiency (see page 38),
   d) deficiencies of magnesium, vitamins A, D\(_3\) and B\(_{12}\) (see page 37),
   e) calcium toxicity (high calcium in the presence of low magnesium and low vitamin D),

3) **hormone swings or imbalances,**
   a) estrogen dominance (see page 41),
   b) hypothyroidism (low thyroid production), or hypometabolism (low thyroid effect) (see page 38),
   c) adrenal exhaustion (see page 41),
   d) adrenal hyperactivity (hypercortisolemia) (see page 41),

4) **chronic inflammation,**
   a) an unrecognized or unresolved chronic infection (see page 43),
   b) chronic allergy (immediate hypersensitivities, IgE)
   c) delayed hypersensitivities (IgA, IgM, IgG) to foods (see page 43),

5) **toxics exposures,**
   a) heavy metal poisoning (see page 44),
   b) oxidant exposure (bleach, chlorine, ozone, sulfite),
   c) cross-linker exposure (formaldehyde, acetaldehyde, alcohol),
   d) exposure to high levels of natural phytotoxins (herbs, vegetables) or mycotoxins (molds),
   e) exposure to high levels of pesticides, herbicides or xenobiotic chemicals, or

6) **unbalanced diets,**
   a) vegetarian or vegan diet (simple avoidance of any animal-flesh food),
   b) allergy to grain-based foods like wheat, oats, rye and corn), or goitrogenic millet,
   c) eating excessive cruciferous vegetables (cabbage, broccoli, kale, Brussels sprouts, cauliflower, etc.),
   d) eating flesh foods from animals fed moldy grain (toxicity to mycotoxins accumulated in the meat),
   e) eating flesh foods raised with hormones and antibiotics and fattened with grain (pre-diabetic meat), and
   f) eating the SAD (standard American diet) rich in processed and refined foods.

7) **any combination of the above.**

So if you don’t get phenomenal results with BHT, don’t despair. There are a plethora of adjunctive approaches that could change your results for the better. It just takes some time and trouble to figure out what the problem is. Then you can do something effective about it.

Because many of the above factors and conditions are routinely ignored or misdiagnosed by mainstream medical professionals, it falls to you to identify whether these overtly medical conditions might be causing

\(^{15}\) This topic can be traced back to the 19th-century dialog between Louis Pasteur and Antoine Béchamp. Pasteur argued that disease was primarily caused by simple exposure to microorganisms, and Béchamp argued that disease was primarily caused by the biological “terrain” of the individual. Although French and British science hold Pasteur as the clear victor in the debate, Pasteur conceded the argument to Béchamp on his death bed. That concession had no influence on Pasteur’s position or Béchamp’s reputation, or the course of modern scientific theory.
you some kind of health problem. If you employ self-care diagnostic techniques, quantified-self monitoring methods and/or a bio-hacking strategy, and uncover a likely medical problem, there are medical tests that you can ask for to confirm any medical diagnosis that was missed in your past. Furthermore, even if you experience disbelief, verbal abuse and/or an uncooperative attitude on the part of your physician, much medical testing is now being made available on an over-the-counter basis (see page 5), and this trend to patient-access medicine is one of the most rapidly developing sectors of the health-care industry.

This book is very much different from Wipe Out Herpes with BHT due to the inclusion of this kind of information.

It is also different in being frequently updated.

The good news is that, once you figure it out, the chances are excellent that you will not need to continue to take BHT to maintain control of viral outbreaks.

**Theory vs Practice. How does BHT Work?**

Just because BHT chemically disrupts the physical structure of lipid-enveloped viruses (fat-containing viruses) in a test tube does not necessarily mean that it does so in an animal or a human being. This observation provides a concrete example of the difference between theory and practice. From a functional or empirical point of view, it really does not matter how BHT works against viral disease, but only that it works.

Does it work, or not? That is the practical question.

Knowledge or speculation about how something works is not particularly useful in determining whether something works. It either works or it doesn’t. This is an essence of simplicity; it works or it doesn’t. However, knowing how something works or believing how something works can influence how well something works. There are two reasons for this. First, as mentioned previously, the power of belief enables neuroendocrine mechanisms of body regulation. Belief is the necessary and essential component of the placebo response. Belief is also the foundation of hopefulness (as opposed to helplessness, which undermines efficacy). Second, understanding mechanisms of action enables the optimization of therapy, by modifying timing, dosing and adjunctive therapies to synergize with the mechanism of action. In other words, we can use theory to predict how to improve the therapeutic outcome in situations with which we have no direct experience.

I have spent the last 40 years learning about mechanisms of health and their influence on disease. This has allowed me to form new hypotheses about methods of controlling viral outbreaks, which, combined with user feedback from people using these methods, has grown into a global understanding of viral/host dynamics. This understanding now allows me to explain why…

1. BHT does not tend to work as well in vegetarians and vegans.
2. Viral outbreaks are more likely to plague younger women and older men.
3. Women tend to have flare-ups in synch with their menstrual periods.
4. Herpes sufferers tend to have the typical symptoms of hypothyroidism.

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16 In drug studies, placebos often produce efficacy rates of 30% in the control group, which usually requires the drug company to establish efficacy rates of 35% or higher to achieve approval. What this means is that belief is comparable to drug efficacy in its ability to ameliorate disease processes and alleviate symptoms. The power of belief is worth cultivating, and should not be dismissed as fringe science. Acting in concert with ones beliefs, knowledge and values can be a positive means to enabling the natural healing processes of the body and mind.

17 These hypothyroid symptoms include (1) low body temperature, (2) low heart rate, (3) cold hands and feet, (4) difficulty in warming up after getting chilled, (5) mild-to-moderate depression, (6) lowered strength and stamina, (7) sleeping difficulties, (8) tingling or burning sensations, especially in the feet and hands, (9) edema (water retention in the ankles, puffiness of the face and under-eye region, (10) fatigue and lethargy, (11) apathy, (12) constipation, (13) memory problems, (14) brain fog,
5. High-dose vitamin E supplementation may significantly worsen herpes flare-ups.
6. Vitamin B₁₂ is particularly effective in enhancing antiviral response in BHT-nonresponsive cases.
7. Viral susceptibility is associated with a sedentary lifestyle.
8. Some of the most virulent viral pathogens (Hong Kong flu, Asian flu, SARS, coronavirus) come from China.
9. Most “deadly” viruses stop killing Americans within months of spreading into the US.
10. There is a multi-fold variation in ebola mortality in geographically adjacent areas of Africa.

There is a clear thread that ties these observations together—the metabolic hypothesis (see page 31). Those rational-dominant readers who need to know the why of this approach can get their “mechanistic” fix in those pages.

Practitioners who run into problems with certain clients can straightforwardly figure out what the glitch is.

The connection between vegetarianism/veganism and herpes could be from vitamin B₁₂, which is not found in vegetable foods. B₁₂ has important antiviral activities. Vegetarian diets can also contain excessive alkaline ash if not “balanced” by 1) fat-containing grains with acid ash, 2) fat from meat, 3) a high basal metabolic rate (BMR), which balances food-born alkaline ash with metabolically generated carbon dioxide (carbonic acid), and/or 4) lots of aerobic exercise. In other words, you can get the “why” of what happens and have an understanding of “what” to do about it. People suffering from undiagnosed hypothyroidism (or to be more precise, hypometabolism, or low basal-metabolic rate) are more likely to have a serious herpes problem. Metabolic rate is primarily determined by mitochondrial metabolism, which is the single largest acid-generating metabolic mechanism of the body. Therefore, this book describes the symptoms of hypothyroidism, and the best tests for its accurate diagnosis (which 90-99% of doctors will not tell you about, or will dismiss this mechanism based on a “normal” blood test which does not actually correlate with metabolic rate).

There are also ways to obtain thyroid glandulars and thyroid hormones without a prescription, if your doctor will not practice medicine the way you want.

But there is another potential value to be had, for readers who do not really want to take BHT, but are presently desperate. Their reasons may be:

1) because it is a preservative (heavy pejorative overtones),
2) because it is non-natural (i.e., it is a xenobiotic drug),
3) because it thins blood for two days at low doses, and for the duration of therapy at high doses,
4) because it causes cancer (it actually doesn’t),

(15) dry or coarse skin, (16) hair loss, and (17) potassium over-utilization (lower serum potassium and elevated cellular potassium).

See page 29 for more information about highly alkalinizing diets, including an explanation of why cellular acidity is an essential aspect of health while tissue acidity is often a sign of disease.

The correlation of thyroid tests with metabolic rate depends on the test. The TSH test has the worst correlation, yet it is considered the gold-standard for assessing thyroid status by 80-90% of clinicians. The best correlation is the T₃-to-rT₃ ratio, both tests of which are routinely denied by Kaiser, for example. “That’s not what we do in Western medicine,” is a common explanation for such illogie. This institutionalized resistance is one reason why herpes is so troublesome in our society, and why many other diseases flourish. Be prepared to experience resistance, even if you are willing to pay out of pocket for the full thyroid hormone panel. And do not forget to include the two thyroid autoantibody tests.

There is a recent trend in the USA to make medical tests available without a prescription. This ranges from over-the-counter salivary testing methods and blood-spot testing to the Life Extension Foundation’s in-house prescription service that is bundled with the tests. See www.LEF.org. So if you are willing to pay for testing out of pocket, a non-cooperative physician is no longer a bottleneck to your health-discovery process. 2016 update: There are now multiple online services where pretty much any blood test can be ordered using the services medical director instead of your own physician.
5) because it can make you dizzy from lowered blood pressure after you take it,
6) because it is a promoter and antipromoter of carcinogens (slightly bad and very good, respectively), or
7) because the FDA approved it (and they have pretty much screwed up every other decision they have ever made regarding industry conflicts of interest).

Whatever the reason, these adjunctive therapies can easily be used as primary therapies. There is no requirement to use BHT at all!

Or, alternatively, readers could decide to limit their BHT use to topical applications (i.e., for surface lesions or shingles treatment). Dissolving 10-15% BHT into refined coconut oil is easy, takes only an hour or two, and is stable for months at room temperature and years in a refrigerator.

While the number of people using BHT is still increasing, it remains difficult to collect the information from which a systematic and quantitative evaluation of BHT’s efficacy might be made. What factors enhance BHT’s success? What factors impair its efficacy? The simple reality is that most people who choose to use BHT do so in the privacy of their own lives. There is no “reporting system” to collect their data, and there are significant privacy issues surrounding “sexually associated” diseases.

Some people have chosen to share their experiences with me, and this has strengthened my knowledge of practical issues dealing with BHT use. These “tips” are and will be included in this book. However, there is a selection process involved in who chooses to talk to me and who does not. Does treatment success or failure increase or decrease the likelihood of reporting? How does personal embarrassment affect the decision? What about being male or female? Young or old? Rich or poor? Famous or anonymous?

The economics of BHT are just as difficult. BHT is a generic food preservative that costs about $10 per pound in the wholesale chemical market. A pound of BHT (approximately a half a kilogram) can be expected to last a person from a minimum of 6 months to as long as 4 years! No drug company is going to be interested in such poor profit potential, no matter how many millions of people might be customers. Furthermore, there is much to be lost. Drug companies might suffer financially were BHT to successfully compete with their current antiviral drugs that can cost dollars per day instead of pennies. Without a profit incentive, and with profit disincentives, it’s no wonder that drug companies haven’t exploited BHT.

Even if a drug company was willing to spend 20 million dollars (a very optimistic figure) to get FDA approval for BHT as an antiviral drug, and try to sell a high-markup FDA-approved brand of BHT, they could not prevent competing companies from selling BHT at low cost, or prevent disclosure of public information (like this book) about using generic BHT to treat herpes at a huge savings. The chemical patents on BHT expired half a century ago. Even the “use patents” on antiviral applications of BHT expired more than two decades ago. At the present time, it’s a wide-open market, and BHT is as thoroughly generic as it is possible to be.

This same “generic” liability also applies to dietary supplements. For example, the antiviral substance hypericin, which is extracted from the antidepressant herb St. John’s wort, has not been adequately investigated for its antiviral potential. Yet it has produced some spectacular results. Another example, the essential amino acid lysine is also well known to suppress herpes flare-ups. Yet it is rarely prescribed by medical professionals. Why? Hypericin and lysine are natural substances. As natural substances, they cannot be patented as new chemical entities. Without patent control of the chemical, price competition between cheap generic products and high-markup pharmaceutical products cannot be prevented. Without high profit potential, drug companies are not interested in investing the research-and-development costs required for the FDA’s drug-approval process. Drug-approval costs now average a significant fraction of a billion dollars per drug. Drug companies are also not interested in promoting generic therapies that
compete with their proprietary substances. It may be sad, but drug companies are currently the single biggest sources of medical information to doctors.²¹

Drug companies run on profits. Foundations and governments do not. Why haven’t any non-profit corporations or government grants been applied to researching the antiviral properties of BHT or hypericin? The answer to this question is more difficult to understand. It is easy to assume that a governmental agency like the Food and Drug Administration (FDA) would be interested in a treatment for an otherwise incurable viral disease. After all, these viruses cause immense human suffering and death. However, the FDA doesn’t work that way. The FDA is not empowered to independently develop therapeutic agents. They are legislatively empowered only to supervise the investigations of drugs brought to them by outside institutions, like drug companies. Generally, these companies only investigate therapeutic agents with large profit potential.

What about foundations? Well, this is even more complicated to explain and understand. It may seem cynical, but I have observed that foundations with a stated purpose to cure a disease spend lots of money on basic “research” and patient management (symptom-oriented “therapy”), but little on functional therapy (dealing with the cause(s) and mechanisms of their chosen disease). If one didn’t know better, one might assume that the cure for their disease is the last thing they want.

Many also spend considerable money on “educational” advertising and promotional activities that attack alternative approaches (alternative only in the sense of being different to theirs) as “quackery.” It is thinly disguised, politically motivated self preservation.

When you think about it, that actually makes twisted sense. If the cure is found, there is no more need for the foundation. Whatever the original good intentions of the founders, the survival of the foundation—and the people who earn their livelihoods from the foundation—becomes more important than the founder’s “goal” to cure the disease. Survival instincts win out over the long run.

It is unfortunate that BHT’s use against herpes has fallen through the cracks of institutional medicine and government bureaucracy. Regardless of how dysfunctional our institutions may be, they do not yet control the personal decisions of individuals in our society. Whatever their choices, you have yours. If you want to use BHT, hypericin, lysine or some other generic substance for a use of which the government disapproves, that is your decision. As long as the purchase is legal, the decision about how to use something is primarily yours.

I hope that this book will provide the necessary information such that you can better make a more fully informed decision about viral disease-treatment options.

²¹ This not only includes medical information provided to medical students in medical school, and medical doctors in residency, but also to licensing-mandated continuing-medical-education information at medical conferences. Then there are the openly commercial pharmaceutical sales representatives who directly solicit physicians in their offices. Although awareness of conflicts of interest is increasing in some medical and scientific circles, it is not in others. In the last few years, there have been media exposés of corruption of peer-review process and overt pharmaceutical-industry influence in medical journals. Despite exposure of these egregious examples, more subtle influences persist unchanged. These influences have transformed American medicine into a disease-management industry where solutions to the underlying causes of disease and death are ignored and superficial aspects of disease are treated symptomatically. Disease management economically serves physicians and pharmaceutical companies alike. Only their clients and the taxpayers suffer. The US has the most expensive medical system in the world, but is ranked between 35th and 40th in the world regarding outcomes. This is a damning statistic. As a cause of death, iatrogenesis (medically caused deaths) is now the leading cause of death in the US (780,000, compared to 700,000 for heart disease and 550,000 for cancer). If underreporting is as problematic as I think it is in the areas of adverse drug reactions, medical error, infection and malnutrition, iatrogenic deaths could currently be higher than cancer and heart disease combined—only we don’t know about it yet.
Medical Supervision

The issue of medical supervision is especially troublesome for BHT and generic therapies. Few physicians know anything about BHT, and most medical education programs—and continuing education courses—are financially underwritten by pharmaceutical companies with vested interests in proprietary drug technologies.\(^{21}\)

Unless a physician is personally motivated by an interest in nutrition, biochemistry, herbology, chelation therapy, and/or environmental, orthomolecular, functional or traditional medicine, they are unlikely to discover the unorthodox educational opportunities that are made available by a variety of US and international medical societies.

With higher-dose BHT, physician supervision is a good idea. Beyond 1 gram per day, BHT can cause liver enzyme elevations. It can also change the way other drugs are metabolized (see page 66). Hypericin and B\(_2\) can cause photosensitivity (light toxicity) at higher doses (see page 34). Knowledgeable medical supervision can reduce these risks.\(^{22}\)

But where can anybody find a doctor who knows anything about these issues?

Maybe you can. Maybe you can’t. If you are lucky enough to have an open-minded physician with the time and the willingness to listen to you, maybe your suggestion that they read the technical sections at the back of this book will give them the necessary knowledge—or a personal curiosity.

If not, then you may be forced to resort to self care. Although I am a strong supporter of self-care options, I do feel that a lot of people can have difficulties telling where the line should be drawn between self care and supervised care. Since such lines are invariably influenced by the therapeutic circumstances and each individual’s personal values, there is no universal advice I can give. However, I will endeavor to provide context whenever I can.

Personal Responsibility

I believe in the fundamental value of human choice. I think the right to make decisions which will affect our lives is an essential human right and a necessary aspect of human nature. The information in this book is provided to support this basic right. The decision to use BHT against herpes must be yours.

This book is for informational purposes only. It is not intended as a substitute for medical advice. It is not intended as a substitute for carefully considered judgment. It is intended simply as an information resource.

You will have to judge for yourself whether BHT is an appropriate option for your personal circumstance.

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\(^{22}\) Vitamin D\(_3\) can be taken safely in doses exceeding 50,000 IU daily when taken without any calcium supplementation (see Prendergast), or when taken with low-dose calcium (<100 mg supplementally). But mainstream medical recommendations for calcium supplementation of 500 mg, 750 mg, 1000 mg, 1500 mg, and in the last 15 years 2000 mg and 2500 mg for treatment of osteoporosis are exceedingly dangerous when taken with high-dose D\(_3\) (50,000 IU) and moderately dangerous when taken with physiologically normal doses of D\(_3\) (5,000 IU). Interestingly, the step-wise escalation of calcium-dosage recommendations over the years due to ongoing lack of efficacy for osteoporosis never reached a level sufficient for efficacy in osteoporosis. Never. Osteoporosis has virtually nothing to do with dietary calcium intake (or milk consumption), being much more related to deficiencies of magnesium and vitamins D and K, and the dominance of estrogenic influences. I find it even more bizarre that, during this same time, estrogen-dominant hormone replacement therapies (Premarin and Provera) were actively prescribed for osteoporosis. Although this non-biosidental hormone replacement did preserve bone density, it blocked bone rebuilding, so that bone strength and resiliency against fractures actually got worse. So, clearly, physicians must be knowledgeable for their supervision of your health to actually lower risks and be a good idea. *Caveat emptor.* Let the patient/client beware.
Herpes Basics

Herpes simplex virus infection has long been a major epidemic problem throughout the world. Up to 10% of the US population has genital herpes and more than a half million new cases are reported each year. The number of people that have oral herpes is vastly higher. More than 99% of adult humans have dormant herpes viruses in their cells and nerve ganglia. This is also true of people who think they have never had herpes.

The herpes virus is almost always transmitted through skin to skin contact (sexual or nonsexual) and results in periodic flare-ups of painful or itching blisters and sores around the mouth, face and genital regions. These are sometimes accompanied by fever and other symptoms of infection, particularly during the initial exposure. Most physicians and scientists say that herpes is incurable because they have not yet found a vaccine or other treatment that effectively controls or destroys the virus. The best that they can offer has been complicated, difficult-to-follow diets that help keep the virus in its latent (inactive) state, ointments that merely ease some of the symptoms, and a new generation of toxic, marginally effective acyclovir-like drugs that interfere with DNA transcription (both viral and human).

This book will present a safe, simple and inexpensive treatment that can reduce the severity of symptoms, reduce the frequency of flare-ups, speed healing, reduce infection and re-infection, and in some people, stop herpes flare-ups completely. BHT is not a cure for herpes. Once infected, the herpes virus can go dormant and “hang out” for an indefinite period of time. Also, the herpes virus DNA can insert itself into our DNA and become, essentially, a part of our genes. BHT does not change that. No known technology can yet change that. However, BHT does have antiviral activity against the “active” viruses that cause symptoms and infect new cells. BHT may even be able to block herpes infection in the first place—and reinfection in people already infected—if used prophylactically.

Although I will describe prophylactic uses of BHT in this book, these are unproven. Nobody knows exactly how effective BHT might be in preventing skin-to-skin transmission of herpes virus. Consequently, it is not wise to rely upon BHT for this purpose. However, if intimate contact is going to happen regardless of BHT use, it may be wise to use BHT to further reduce what are considered to be acceptable risks.

This book describes the means by which tens of thousands of herpes victims have rid themselves of troublesome herpes symptoms by taking one or more small capsules per day of BHT. It will explain a variety of ways it can be used (oral, topical, suppository, vapor), how to increase its effectiveness (empty stomach vs predissolving it in fat), how safe it is, what medical tests can be used to monitor its dangers, how to prepare it for use, and how and where to obtain it inexpensively and without prescription.

This book will also discuss the most recent findings about BHT’s effectiveness against shingles, hepatitis, cytomegalovirus, influenza and other viral diseases (see page 52). Mention will also be made of other beneficial effects of BHT.

Although many scientists and physicians will find this book fascinating and informative, it is primarily intended for lay people. Those wishing to pursue the technical details will find that the latter chapters are extensively referenced.

What is BHT?

BHT (butylated hydroxytoluene) is a synthetic food preservative that is widely used in the US to prevent rancidity in fat-containing foods, such as breakfast cereals, baked goods, potato chips, pork sausage, peanut butter, instant potatoes, and other commercially prepared foods. Even foods labeled “no preservatives” or

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23 Dormant virus can exist in an intact virus form, or it can be the naked viral DNA crossed-over into the human DNA. Many estimates for the average herpes viral DNA load in humans at death exceed ten thousand billion copies.
“no preservatives added” may and often do contain BHT, which was present in the ingredients used in making the food. Such pre-existing additives and preservatives do not need to be disclosed on labels. You can thank the FDA for that.

Since 1947, the US Food and Drug Administration (FDA) has approved BHT’s use in amounts up to 0.02% by weight of the food product (in some instances even higher). BHT is generally not approved in other countries. The typical daily intake in the USA is estimated to be about 2 mg. Although it is considered a completely synthetic substance, its unusually low toxicity makes it a much safer compound than many natural substances in food. Furthermore, it has been found to have many outstanding vitamin-like effects on humans and animals, which will be discussed later.

The only apparent long-term effect from the small amount of BHT (2 mg or so) that most Americans get is a statistical reduction in the incidence of gastrointestinal cancer since this preservative first came into commercial use in 1947. In experiments with animals, larger doses have reduced the incidence of many kinds of cancer by much greater amounts. On the basis of animal experiments and other evidence, a few scientists have speculated that a daily intake of 50-250 mg of BHT could reduce the rate of human cancer to less than half of what it is today. BHT’s powerful antioxidant and free-radical-scavenging properties could similarly lower the occurrence of heart attack and stroke. Animals that are given large daily doses of BHT live up to 50% longer than normal and maintain youthful characteristics throughout most of their lives. This will be discussed at greater length later.

Although BHT is a synthetic compound, it bears a chemical similarity to tocopherols (the vitamin E family) and to some naturally occurring nutrients (phenolic antioxidants and flavones). BHT seems to have vitamin-like activities in the body, possibly because it “preserves” the fat-soluble vitamins E and 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.

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. It is possible that some of these toxic reactions to BHT are caused by impurities in commercial-grade BHT that could be minimized by further purification (see page 27). If you try this, please relate your experiences.

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. I suspect that BHT helps the skin partly by direct enhancement of antioxidant defenses and partly by indirect preservation of vitamins A and E, both of which play a role in skin health and vitality.

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, nonmutagenic (doesn’t cause mutations), and nonteratogenic (doesn’t cause birth defects). For a more detailed discussion of this issue, turn to page 65 in the Toxicology of BHT chapter.

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24 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.
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 foods. While this may be true to a significant 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.  

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, 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 and tobacco) contain solanine, which can cause skin sensitivity and severe arthritis symptoms in susceptible people. (Solanine alkaloids are the basis for the “eggplant skin-cancer cure.”) 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 (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.

There are no figures available about acute toxicity of BHT in humans from extremely large doses. One study with rats showed the LD$_{50}$ (the amount that kills 50% of the animals) to be 1,600-3,200 milligrams per kilogram of body weight. This would scale to 1/4 pound to 1/2 pound of BHT for an average 154 pound (70 kg) person. There are likewise no figures available about the minimum toxic dose in humans. Some people take up to 2,000 mg daily without side effects, although other people develop liver enzyme elevations in doses between 1000 mg and 2000 mg. A few people have taken as much as 3,000-7,000 mg, although side effects become quite common at those dosages. One study of 36 dogs reported daily 10,000 mg dosages throughout life with no obvious toxic effects and some outstanding benefits (see page 61). The above figures might indicate that BHT probably has a therapeutic index (the ratio of the toxic to therapeutic doses) of 10 to 1, which is comparable to Tylenol and alcohol. This makes BHT acceptably safe by pharmaceutical standards.

BHT seems to have no specific long-term toxicity, even when taken in medicinal doses. The short-term toxic effects involve 1) a transitory reduction in blood clotting, which lasts only a day or two, 2) a transitory lightheadedness, which is related to postural hypotension (an autonomic condition), and 3) induction of liver enzymes which metabolize BHT, which are apparently harmless and fully reversible on discontinuation of BHT. This latter effect results in a slight enlargement of liver cells due to increased metabolic activity of this organ. This same enlargement is seen with the consumption of cruciferous (cress-siff-er-us) vegetables (broccoli, Brussels sprouts, cabbage, cauliflower, bok choy, kale, etc.), which contain natural chemicals that the liver must metabolize similarly to BHT. This phenomenon of liver enlargement is discussed in greater depth later in the section The Toxicology of BHT (pages 63-68).

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25 In the last two decades, BHT has been found in a variety of plants. So it may turn out to be natural after all.
Although BHT seems to be healthful rather than harmful to a normal liver, anyone with a history of liver pathology (hepatitis, jaundice, cirrhosis, etc.) should use BHT only under medical supervision with regular liver-function tests (see page 66). When John A. Mann and I wrote *Wipe Out Herpes with BHT* back in 1983, we specifically cautioned against BHT use in cases of hepatitis, specifically because of the compromised liver function associated with active hepatitis infections. However, over the intervening years, I have heard of a dozen cases where BHT has spectacularly resolved symptoms of chronic hepatitis (A, B and C, even though hepatitis A is supposed to be a non-lipid-enveloped virus according to some sources, and hepatitis B is non-lipid according to other sources) and none where BHT has aggravated them. (Additional details were provided on page 10.)

Although animal studies suggest that BHT does not cause birth defects, the potential risks of BHT to human pregnancy have not been directly assessed. In my opinion, only a minimal standard of safety has been met by pregnancy studies in rodents and monkeys. Stated negatively, the available data do not rule out the possibility of the clinical use of BHT in pregnancy or infancy. The prevalence of herpes virus and its special risks to pregnancy may tempt many physicians to use BHT in treating pregnant women or newborn infants with life-threatening herpes infections. Data are badly needed. I ask that anybody using BHT during pregnancy, childbirth, lactation or early infancy to please share their experiences with me. Chronic prenatal exposure to BHT may perhaps prove to be benign, however, infant exposure through breast milk must be considered as a realistic concern. BHT is significantly excreted in breast milk, although there are no data that specify exactly how much is secreted.

The strongest warning about BHT is not to take it while drinking, as it temporarily interferes with alcohol metabolism and may get you much more intoxicated than usual. If you take BHT while drinking alcohol, or you take BHT shortly after drinking even small amounts of alcohol, don’t drive! Also, refrain from trying to operate machinery, dice vegetables or juggle chainsaws. This warning against alcohol and BHT applies to near-concurrent use. BHT with breakfast and alcohol with dinner, or alcohol in the afternoon and BHT at bedtime, will not interact in this way.

BHT can also change the rate at which other drugs are metabolized, and some adjustment of dosage may be necessary. If you are taking medications like phenytoin (Dilantin) or phenobarbital, you would be wise to consult your doctor or pharmacist before taking BHT.

There have been a few reports of allergy to BHT involving very mild skin rash. The problem is extremely rare and is less likely to occur when the person is getting a good, nutritionally rich diet with adequate amounts of zinc, vitamins A, B₆, and C, and small amounts of polyunsaturated fatty acids (GLA, EPA and DHA).

**The Viral Infection Process**

When any kind of herpes-like virus is transmitted from one person to another, the virus particles penetrate the skin, bind to cells near the surface, and inject their DNA or RNA into the cell. This DNA or RNA “hijacks” the DNA-copying and protein-making machinery of the cells.

DNA (deoxyribonucleic acid) is the molecule of human inheritance that encodes all the “instructions” and “machinery” necessary to create the structure and coordinate the function of the human body. DNA is like a computer code that translates into words in an encyclopedia. The DNA “sentences” are called *genes*, each of which produces a specific protein or enzyme when it is translated. The DNA “volumes” of the encyclopedia are called *chromosomes*. The human encyclopedia, called the genome, consists of 23 volumes (chromosomes). Other animals have different numbers of chromosomes.

The DNA in chromosomes is extremely long. A typical human chromosome contains 50 to 300 million nucleic acids strung together in a chain. It takes three nucleotides (“letters”) to code for each “word,” and a dozen-to-thousands of letters to code for each “sentence.” That’s a lot of letters, words and sentences.
This extremely long strand of DNA strand is wound around protein cores called histones, very much like the way thread is wound around a spool.

Viral DNA is much smaller. Viral genomes are only 5-50 thousand nucleotides long. They are extremely small because much of the biochemical machinery that they need in order to replicate is provided by the host cell. They only need to carry the “extra” stuff not present in their host.

When virus DNA inserts itself into the cell, it is copied (transcribed, translated) repeatedly into RNA, like a Zerox machine making multiple photocopies. These RNA “copies” are then repeatedly translated (transcribed) into proteins. RNA viruses carry an extra protein called reverse transcriptase, which repeatedly copies the RNA into DNA (the reverse of the normal cellular process). This DNA is then repeatedly copied back into RNA the same way that DNA viruses are copied.

These DNA-to-RNA and RNA-to-protein processes are the exact same steps by which human DNA is translated into human proteins. Only with viral DNA, you get viral proteins. A virus “assembly line” is created that manufactures new viruses. Some viruses “bud” out through the cell membrane as they are produced, taking a coating of the membrane with them as a lipid envelope. Other viruses simply accumulate in the infected cell to the point where the cell eventually ruptures and is destroyed, spewing forth thousands to millions of new viruses into the blood stream where they travel to infect new cells.

If this viral life cycle continues unchecked, the virus will multiply until it either causes serious organ pathology or it kills enough cells to kill its host. Fortunately, the immune system counteracts this process by detecting the virus proteins and destroying infected cells and free viruses.

**Lipid-Enveloped Viruses**

In the case of herpes, the immune system has a difficult time getting at the virus because of a lipid (fatty) coating that camouflages most of its proteins. Scientists call herpes a lipid-enveloped virus because of the fat (lipid) found in the outer shell or coat. To the immune system, lipid-enveloped viruses look more like tiny fat droplets than an infectious organism.

Not all viruses are lipid enveloped. For example, poliomyelitis (polio) virus, hepatitis A and the common cold virus (rhinovirus) have no lipid covering their outer protein shell. There is not yet any clear evidence yet that suggests that BHT has an effect on non-lipid viruses. However, there has been medical reporting over many decades that antibiotic use seems to result in beneficial effects in viral disease, even though there is no known (!) mechanism. So it is possible that BHT will have a beneficial effect on non-lipid viruses like antibiotics do.

Standard vaccination approaches for lipid-viral diseases become difficult-to-impossible because they are based on the immune system’s response to proteins. Lipid viruses have much less exposed proteins. Lipid-enveloped viral diseases are among the most difficult diseases to treat. Current drugs have only marginal benefits.

Currently identified lipid viruses include all herpes strains, Epstein-Barr virus, human immunodeficiency virus (HIV, all strains), cytomegalovirus (CMV), hepatitis virus (B and C), rubella virus (German measles), varicella virus (chicken pox), Newcastle disease virus, swine fever virus, coronavirus, SARS virus (a coronavirus), West Nile virus, ebola virus (and other hemorrhagic viruses), and influenza virus (all strains, including swine flu and bird flu viruses).

**Herpes Associates with Nerve Ganglia**

Herpes viruses have a special affinity for the human nervous system.

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26 The “metabolic hypothesis” is a viable mechanism to explain how antibiotics exert an antiviral effect.
Virus that successfully evades the immune system retreats through nerve fibers to nerve clusters (ganglia) near the brain or spinal cord, where they go into a latent state. Sometimes, the virus will remain in this state for life, causing no apparent harm.\textsuperscript{27} In many cases however, it is awakened periodically by changes in body chemistry due to stress, diet, illness, weakened immune system, menstruation, overexposure to sunlight, or other causes. Even sexual activity can trigger the dormant virus to become active. The virus then travels from the ganglia, through the nerve fibers, back to the same area that it first infected and the victim has another episode of sores and blisters. These eventually subside as the virus retreats once more to its hiding place in the ganglia, where it remains until it is triggered again into its active state. We will discuss more about metabolic “activators” of herpes later (see page 31).

**Controversy about Proposed Mechanisms of BHT’s Action**

In test-tube experiments, scientists have identified two specific ways in which BHT inactivates lipid-containing viruses. First, it disrupts the relationship between the virus’s lipid envelope and its protein shell, stripping the virus and making it vulnerable to attack by the immune system. Second, it removes binding proteins that viruses need to bind to and penetrate cell membranes. Without these binding proteins, viruses are non-infective.

Whether or not these mechanisms are applicable to living animals has not been determined. In fact, it may be near to impossible to make that determination. In test-tube experiments, scientists can utilize viral preparations that are 100% whole-virus (i.e., they have been purified to remove all viral fragments). They can then study whether whole viruses disintegrate in response to an antiviral agent. This is not the case with lab animals suffering from a viral infection. The viral replication process (the assembly line) is not particularly efficient—at all. There are many more viral parts produced that there are fully assembled viruses. Massive numbers of viral fragments are released when an infected cell ruptures. Scientists cannot easily distinguish between viral fragments that are a natural product of inefficient viral replication and viral fragments which are produced by BHT disruption of intact viruses. So in real-life, scientists haven’t yet figured out how BHT works.

I believe that there is enough evidence about the mechanism by which BHT disrupts viruses in a test tube to conclude that this is not the mechanism of action in living animals. BHT precipitate has been positively correlated with its virucidal activity in test tubes, but BHT precipitates are highly unlikely to form in living animals.\textsuperscript{28} I think that it is much more likely that BHT works by a general metabolic mechanism discovered by Emanuel Revici in the 1930s (see The Metabolic Hypothesis on page 31).

Despite these unanswered questions, researchers have found that BHT does interfere in the course of lipid-enveloped viral diseases.\textsuperscript{29} Animals given BHT resolve their lipid-enveloped viral diseases much faster than otherwise (see BHT Antiviral Findings on page 60). And the vast majority of people using BHT do experience relief from their herpes infections. Empirically, it works.

Should someone discontinue use of BHT, the symptoms may or may not return, depending upon the many factors that influence herpes. Some people report going for very long periods of time symptom free, even when their previous episodes were chronic, severe and protracted. Other people report flare-ups within a

\textsuperscript{27} With newer, more sophisticated viral testing methods, detection of viral infections of the brain at autopsy are becoming more common, which opens the door to the questions of 1) whether unrecognized low-level viral infections are a common aspect of aging and/or neurodegenerative diseases, and 2) whether viral flare-ups may be an unrecognized contributory cause of death. The prevalence of high herpes viral titers in brain tissue of people who’ve died with Alzheimer’s disease has prompted some researchers to propose herpes as a possible cause of Alzheimer’s disease. I suspect that the Alzheimer brain is merely more susceptible to opportunistic infections, and that more viral infections will be found when tested. Perhaps this is only the tip of the iceberg.

\textsuperscript{28} The BHT concentration cannot be high enough, or the temperature change sudden enough.

\textsuperscript{29} For examples, Newcastle disease in birds, swine fever in pigs and influenza in humans. See later pages for more details.
month of stopping BHT. If dosage is maintained, however, the antiviral effect tends to maintain reasonably stable. However, some people have reported that the antiviral potency of BHT diminishes slowly over time. This is consistent with metabolic adaptation to BHT use. But it may be the result of homeostatic mechanisms that tend to counteract any therapeutic influence that is “judged by the body” as destabilizing in nature.

How to Use BHT

BHT comes in capsules, bulk powder or coarse granules. The most popular capsule size is 250 mg, although any size will do. The dosage can be as little as one 250 mg capsule each day, or it might be more. When BHT is taken is not crucial, but it may matter whether you take it with fatty food or on an empty stomach.

When Durk Pearson and Sandy Shaw wrote about BHT and herpes in their bestselling book Life Extension: A Practical Scientific Approach (Warner Books, 1982), they suggested that BHT was best taken on an empty stomach before bed. This method may significantly minimize liver metabolism of BHT (if there is no food being processed at the time) and it avoids dizziness symptoms (lightheadedness) in people who experience short-term drops in blood pressure when they take BHT on an empty stomach. Blood-pressure regulation is not so important to your consciousness when you’re lying down and horizontal.

When John Mann and I were collecting anecdotes in 1983, we were getting complaints from people who were not getting good results taking BHT on an empty stomach but did when they took BHT with fat (either predissolving it in fat or oil, or taking capsules with fat-containing food). So in the first edition of Wipe Out Herpes with BHT, we recommended that BHT be taken with fat, instead of on an empty stomach.

Since that time, we have had opposite complaints: that taking it with fat did not work as well as taking it on an empty stomach. So, it appears that each method works better in a subset of people. If you get poor results one way, try the other.

BHT can be taken orally (by capsule, powder or oil solution), rectally (by suppository or lipid insufflation) or topically (by dissolving BHT in fat or oil and applying it to the skin). Most people prefer capsules for the convenience they offer. However, oral use maximizes liver metabolism of ingested BHT and minimizes the amount that reaches the deep tissues of the body. Presumably, only the BHT that survives liver metabolism has any significant antiviral effect.

If you have herpes sores when you begin taking BHT, they should begin to disappear within a few days. If the sores haven’t begun to go away after two days, the dosage can be doubled to 500 mg. If the infection still persists after two more days, the dosage can be doubled again to 1,000 mg. Most people respond to less than 1000 mg doses, but some people may require more.

Medical monitoring of liver function is warranted, especially with doses beyond 1000 mg/day. I have yet to hear of a single case of liver enzyme abnormalities with dosages of 1000 mg or less, but it does happen in the 1000-2000 mg dose range. And doses beyond 2000 mg should not be undertaken without regular medical monitoring of liver enzyme function.

It has been recommended that the dosage be continued for two weeks after the herpes lesions have healed to ensure that the viral infection is completely cleared, and that the treatment be commenced again immediately whenever symptoms recur. For some people, this approach makes the most sense. Other people may prefer to take a maintenance dose on a continuous basis so that viral replication is inhibited continuously. Some experimentation may be necessary to discover the approach that produces the best results in each person.

If you wait until symptoms are noticed before taking the BHT, it will take 4 to 7 days for the sores to heal completely. Physicians advise that there be no sex contact for at least three days after the sores are gone, because the patient may still be “shedding” infectious virus. Because shedding can happen after visible
lesions have healed, a sexual quarantine of up to ten days is needed for any reasonable assurance of safety. A person may also be infectious for several days before the sores are noticeable. If the sores occur deep within the mouth, vagina, or anus, they may easily go completely unnoticed, yet still be a source of contagion. Continuous use of BHT may not only minimize infectious risk, it may offer other beneficial effects, like destroying other viruses, or helping reduce the risks of cancer, heart disease and stroke.

BHT can also be taken daily by people who don’t have herpes, not only for these beneficial effects, but also to reduce the chances of contracting herpes. Such uses appear to be effective based on anecdotal reports, but anecdotal reports are no substitute for a controlled scientific study. I suggest that such prophylactic use of BHT is unproven and therefore appropriate only for protection against inadvertent exposure, not for deliberate contact with herpes-active persons.

Optimizing Results

The exact reasons why some people require larger dosages than others is not well understood. It may be because some individuals don’t absorb fats as well as others. BHT is a fat soluble substance. That is one reason why some people do better taking BHT with a high-fat meal. In the stomach and intestine BHT gradually dissolves into the dietary fats or oils and is absorbed with them.

Other people may metabolize BHT too quickly. The blood supply from the intestine goes through the liver before it goes to the rest of the body, so BHT which is metabolized by the liver on the “first pass” never makes it to the body where the herpes infection is taking place. This is why some people suggest taking BHT at bedtime on an empty stomach. Without food, the liver is quiescent (temporarily semi-dormant) and BHT metabolism may be at low ebb. Less BHT metabolism in the liver means more BHT to fight viruses.

In some sensitive people, BHT on an empty stomach may cause a transitory drop in blood pressure leading to lightheadedness. This is another reason to take BHT immediately before bed. A drop in blood pressure is less noticeable (and less dangerous) when lying down in a horizontal position. People with postural hypotension (dizziness from sitting up or standing up too suddenly) may be more susceptible to this problem.

Administration of BHT through the bowel or skin bypasses the liver first-pass effect and maximizes BHT delivery to the body. Topical application of BHT maximizes skin concentrations of BHT which can be especially important with skin-active viral diseases like herpes and shingles. Recipes for topical BHT mixtures are described in the next section.

Viral diseases which can attack the intestinal walls, like cytomegalovirus (CMV), can be treated with coarse BHT granules, which dissolve too slowly to be fully absorbed in the stomach and upper intestine and are therefore carried deeper into the GI tract and possibly all the way through the bowel. Efficiency of absorption can be measured by examining a stool sample for undissolved (floating) BHT granules. BHT has a low specific gravity (1.06 density) and BHT granules can float in salty or mineral-laden water, or when fecal samples are mixed with an excess of saline water. If the BHT granules do not survive to the bowel, fat restriction and/or fiber supplementation may be necessary. Extremely coarse BHT can be made by dripping molten BHT into cold water, or onto cracked ice.

30 The ethical issues for avoiding infection are straightforward as they only involve the informed consent of the person using BHT. But some people use topical BHT to prevent infection of others. Topical BHT can dramatically increase BHT concentration on the skin surface far beyond what can be achieved with oral intake. However, this approach has not been studied. And it involves the consent of the partner. Topical BHT (10-15% BHT dissolved in refined coconut oil or palm oil) is reported to be very effective in treating shingles. So there is reason to believe that it might be an effective prophylactic against herpes transmission, (1) from an infected person to a non-infected person, and (2) from two infected persons to each other, and also (3) from an infected person to themselves (spreading the infection to new nerve ganglia).
Another reason people may require more BHT than others has to do with metabolic factors that can increase or decrease viral susceptibility. This concept may be difficult for many people to understand, so I will devote considerable time and space discussing it (see page 31).

**Dissolving your BHT in Fat**

Although any vegetable oil will dissolve BHT, I recommend coconut oil because of its high saturated fat content, which is more stable against rancidity (oxidation, peroxidation) than vegetable oils grown in the temperate climate of the central United States.31 This means that the BHT-coconut oil mixture has a longer shelf life.

To dissolve BHT in oil, warm the oil to about 100°F (40°C) in a double boiler, or in a glass jar sitting in hot water. If you do not have a thermometer, the temperature should be warm to touch but not too hot to hold. Add 10% or 15% BHT by weight and stir or shake until it completely dissolves.

This is pretty simple.

At 20% concentration, some of the BHT may recrystallize when the oil cools, but this does not seem to impair its absorption when taken orally. When used topically, any crystallized BHT can be initially gritty when first applied, which abates as the oil warms up on contact with the warm skin. 10% or 15% is probably a better concentration for topical application of BHT for treating shingles.

I also recommend not using virgin raw coconut oil for topical use. First, it smells strongly of coconut, which you might not appreciate as a perfume or body scent. Second, it contains significant levels of aqueous fluids and proteins which facilitate oxygen interaction with unsaturated and polyunsaturated fatty acids. In other words, its shelf life is shorter. Most people like to mix up BHT in coconut oil and store it for later use. So shelf life can be important. For a longer shelf life, use refined coconut oil, which has no smell. The BHT-oil mixture will only smell of BHT and not coconut.

**Purifying your BHT**

Since BHT only needs to be pure enough to meet food-preservative standards, where human exposure is only 2 or 5 milligrams, it may not be pure enough for drug use, where doses can be 250-1000 mg, or higher. Impurities that may be well tolerated at 5 mg may not be so at 50 mg or 500 mg.32

As far as I know, there is nobody doing this for BHT sold commercially. So if you react badly to BHT, or you merely want the best product that your money can buy, you might want to go the extra mile by:

1) inquiring of your BHT provider the source manufacturer of the BHT they are selling you, and/or

2) purifying your BHT above and beyond the commercial standards that apply in the USA.

For at-home purification, activated charcoal works well to absorb the low-molecular weight impurities in BHT. You can melt your BHT by putting it in a double boiler, or by heating a glass container with BHT in it, or with BHT dissolved in vegetable oil, coconut oil, or MCT oil in it. The charcoal can then be added to the liquid BHT or BHT in oil. The longer time you leave them together, the more impurities are absorbed. A significant amount of BHT will also be absorbed (the charcoal does not absorb just the impurities; it absorbs everything in roughly equal amounts). So you will loose a significant amount of BHT in the purification process. But, given its low cost, this is not an economic hardship. After the BHT-and-charcoal

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31 Coconut oil also avoids omega-6 (ω6) fat loading, which can aggravate a high ω6-to-ω3 ratio.

32 This impurity problem was also true of some commercial B6 manufactured and distributed in the 1960s, which caused peripheral neuropathy when taken at doses 25 to 250 times the RDA levels. Although this neuropathy was (and still is) attributed to vitamin B6 itself by misinformed authorities with anti-supplement agendas, vitamin manufacturers who knew what they were doing used higher-purity B6 that did not produce any neuropathy, even at doses up to 1000 times the RDA.
mixture has been allowed to mix and sit, it can be suction filtered to remove the charcoal.\(^{*}\) Or it can be consumed with the charcoal.\(^{*}\)

Since activated charcoal is a remedy for poisonings that can be taken orally, there is no necessary need to filter out the charcoal once it is added to the BHT or BHT-oil mixture. As long as the taste is OK with you,\(^{*}\) and the dose of charcoal is not excessive,\(^{*}\) you may be able to reduce your BHT side effects caused by impurities. I do not know how this might affect the absorbed dose. This is also true of transdermal BHT; nobody knows how much gets through the skin into general circulation and how much gets trapped in the skin layers or how much lies on top of the skin.

**Preserving Your Spices, Herbs, Coffee and Foods**

Many foods contain oils and fats that can go rancid on exposure to the oxygen in air. Nuts, seeds and grains are typical examples. The extracted oils and fats have the same problem, only they are generally less stable and age more quickly after being removed from the protective matrix of the food. This higher reactivity and shorter shelf life also apply to associated dietary supplements, like fish oils, seed oils, lecithin and phosphatides (phosphatidylcholine, phosphatidylinositol, phosphatidylethanolamine and phosphatidylserine).

Coffee, herbs and spices have aromatic oils that age similarly to vegetable oils and animal fats. Cinnamon, mint, oregano, rosemary, black pepper, bay leaf, marijuana and pretty much anything with a volatile smell and pleasant odor can oxidize with extended contact with air. In other words, they get stale. This is why chefs insist on fresh herbs for their cooking and why coffee aficionados will often go out of their way to freshly grind their coffee beans before making their chosen brew.

The aromatic oils in spices and the roasted volatile aroma in coffee can be protected by BHT. This can be accomplished by very tiny amounts of BHT added to the food or spice by vapor-phase deposition. There are two easy ways to do this.\(^{*}\)

**Method One:** Glue a piece of felt to the underside of a jar lid. After the glue is dry, pour a teaspoon of melted BHT into the felt. Then, use the jar as you normally would to store flax seeds, coffee beans, ground coffee, stalks of rosemary, whole bay leaves, or ground oregano. Since the BHT is on the lid, it does not come in direct contact with the food or spice. However, the enclosed air space allows the BHT vapor to surround the food effectively.

**Method Two:** Wrap up some BHT crystals in filter paper (a coffee filter works great), staple it to trap the BHT inside, and then wrap it again with another layer of filter paper. Toss the assembled paper package into the food, coffee or spice. The paper layers keep the BHT from direct contact with the food, and the porous paper allows the BHT to “breathe” through the paper. This does not work with oils and nut butters in which the food will penetrate into the paper and dissolve the BHT.

\[^{33}\] Filtering charcoal from pure liquid BHT is a serious technical challenge due to the higher temperature required to keep the BHT molten enough to go through a filter. If you want to try this, call me if you have difficulties and I can make suggestions. Filtering BHT dissolved in oil is much easier. MCT oil is the easiest to filter due to its extreme fluidity. For the chem-lab challenged, you might want to hire a chemist to purify your BHT for you. If you choose this option, make sure that the process is either (1) charcoal filtration, (2) fractionation (a kind of distillation that separates compounds by their boiling points), or (3) solvent purification with a solvent that is easily removed and safe in residual amounts (like spectrograde ethanol, and not hexane, methanol or methylene chloride).

\[^{34}\] Charcoal imparts a charcoal-briquet taste/smell that is less than pleasant and often quite objectionable.

\[^{35}\] Charcoal is also constipating.

\[^{36}\] This applies to room temperature applications only. It will not work well at refrigeration and freezer temperatures. BHT’s vapor pressure is strongly temperature dependent.
This is one way to keep dry dog and cat food fresher in a container with lots of air (boxes, bags, and bins). There is fat and oil in animal feed, and it goes rancid with extended storage, just like human foods. Many commercial animal feeds add BHT and other preservatives as part of their formula. But some may not.

This approach may also be useful for preserving medical marijuana. Not much is known about all of the specific compounds in marijuana that are associated with a variety of medical benefits, but it is possible that some of the most therapeutic are oxygen and radical sensitive. Tetrahydrocanabinol is definitely sensitive to free radicals, undergoing isomerization with age. But there may be other, as-yet-unidentified substances contributing to the medical benefits of marijuana. From the aroma and smell perspective, BHT definitely prevents marijuana from getting stale.

**Strategies for Dealing with Physicians**

People who write about new disease treatments that have not yet been approved by the FDA usually suggest that the treatment be conducted under supervision of a physician. Unfortunately, most physicians are unaware of BHT therapy or Revici’s metabolic approach to health. Even if they may have “heard about it,” they are likely to be prejudiced against it. *Physicians tend to be very down on things that they are not up on.* You will probably have to do a lot of shopping around before you find one who is willing to consider using BHT against herpes, or willing to provide medical supervision for your use of BHT. If you do find one, be sure that he or she gets to read this book before commencing the treatment.

You may get lucky and find a physician who already knows about treating herpes with BHT and has used it on some of his or her patients. I know of several doctors who have used it successfully on hundreds of patients, and even pioneered nutritional adjunctive therapies. But I cannot disclose their names. They are risking 1) liability by using a therapy that has not been approved for this purpose or in such dosages, and 2) loss of their medical license by practicing medicine that is not agreed to be “the standard of care” by other doctors (and state medical boards). There is no “loophole” in the medical regulations if a non-standard therapy actually works. There is no exemption if it is safer for you than accepted therapies. There is no burden of proof that is applied to regulators to establish any level of danger from a non-standard therapy. None at all. Because charges of medical misconduct relating to licensing are a matter of administrative law, the doctor can be assumed to be guilty until proven innocent, and the evidence of their service to their clients (patient welfare) can be dismissed as inadmissible. *The land of the free, indeed!*

The way you can work this with non-standard therapies is to give your doctor a get-out-of-jail card.

1. Assist in the process of creating a clear medical rationale for the therapy.
2. Do not ask for a prescription for the drug(s) or nutrient(s) in question unless it is a controlled substance. Testosterone (a Schedule-III steroid) and Xyrem (Schedule-III GHB) are the only two of the things in this category. This sets the stage for the following item.
3. Document in your medical record your decision to use the non-standard therapy against the doctor’s specific recommendation to the contrary. This way, the doctor’s decision to provide medical supervision for you (including prescribed tests and any prescriptions) is a clear matter of “protecting you from yourself.” If you need to go the extra mile, see a psychiatrist or psychologist and get a “second opinion” that your belief in the therapy can only be dislodged by trying it and failing.³⁷

³⁷ With the expansion of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) to define most psychological foibles as diagnosable mental diseases, anybody (and everybody) is at risk of getting a mental diagnosis inserted into their medical record, which can serve to erase human rights of self determination. Please ascertain in advance that this will not happen regarding your informed decision to favor a non-sanctioned therapy. The DSM criteria for mental disease are not scientific, nor are they objective. You could be diagnosed with “abuse of dietary supplements” or “obsessive ideation relating to [fill in the blank]” that would provide legal precedent to bypass your rights to consent to medical treatments under the US Constitution, Helsinki Accords and UN Universal Declaration of Human Rights. Even a barred lawyer can lose the right to
4) Provide additional documentation of your superior responsibility (asking for advice regarding everything you should pay attention to, like the symptoms of hyperthyroidism if you take thyroid hormones) and meticulous data collection abilities (disciplined measurements of body temperature, pulse rate, or blood pressure, or conscientious tracking of other, subjective, mental or emotional symptoms), and insist that the collected data be added to your medical record.

5) Then, after therapy commences, document your benefits! And insist that they be placed in your medical record.

6) If you can do it and are willing to suffer the consequences, consider doing an A-B-A-B protocol, where you stop the therapy to provide data that your symptoms return, before going on the therapy again. The A-B-A-B protocol is the scientific standard for experiments involving a single subject. You were not taking BHT, then you were, then you stopped to see what would happen, then you resumed therapy. If you symptoms go away, come back, and then go away again, you have generated scientific evidence that the therapy works. Again, put it in your medical record.

In other words, the goal is to create a paper trail that the doctor can use to document that he or she is behaving responsibly (as measured by the bureaucratic standard-of-care yardstick).

In talking with people over the last 25 years, I can say that most people who use BHT to stop herpes do so without medical supervision. They simply buy BHT capsules or bulk BHT from a mail-order or on-line dietary supplement company. 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 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.

If you think that you have herpes and intend to treat yourself with BHT, it may be a good idea to at least see a physician for diagnosis. Some herpes-like symptoms that didn’t respond to BHT were eventually found to be conditions other than herpes. Delaying proper treatment because of incorrect diagnosis can allow a serious problem to progress unchecked. On the other hand, you may have a viral condition that does not manifest in the usual manner. I had a shingles outbreak at age 40 that manifested with the typical trigeminal-nerve pattern on the forehead, except that I had absolutely no pain. None. So my initial thought was that the redness was contact dermatitis from exposure to urethane adhesive, which I had just handled, and to which I am highly allergic. But it did turn out to be shingles, and it responded to BHT-plus-B₁₂ therapy. Within 12 hours, the redness had reduced markedly. And within 48 hours, the redness was gone and the healing was advanced.

There are simple blood tests for viruses. They fall into two categories, 1) antibody tests which are relatively inexpensive, but do not measure the active virus, and 2) PCR tests which are relatively

represent himself or herself in a legal proceeding in the USA once a psychiatric diagnosis is made, or even if the individual is placed in a psychiatric facility for an arbitrary matter of police convenience. [This actually happened in California.] No demur or plea is sufficient to overrule a psychiatric diagnosis and restore your rights. In other words, you may not want to associate in any way with the psychiatric profession.
expensive and actually measure the viral load on the day of the test. PCR (polymerase chain reaction) measures the DNA and RNA of the viruses, so it tests the actual level of virus in your system when the blood is drawn. Sometimes it is a good idea to have both done, first the antibody testing and then the PCR testing.

The Metabolic Hypothesis

Why is it that only 5-10% of people experience herpes flare-ups when more than 99% of adults are infected with herpes viruses? The answer to this question goes to the heart of how to most effectively treat viral disease.

Most Western scientists and physicians think that infectious diseases result from aggressive invading organisms. This view is, to a very large part, the direct result of the ascendant influence of Louis Pasteur, whose demonstration of the role of bacteria in infectious disease was so compelling to scientists of 19th century France (and England) that they abandoned any consideration of the role of host resistance and susceptibility, or the role of microbial virulence and attenuation. In the shadow of Pasteur’s flamboyance, Antoine Béchamp had carefully identified “pleomorphic” changes in microbes that were associated with infection and pathology, and Claude Bernard had identified the influence of biological “terrain” in causing pleomorphic transformations. Although Pasteur ultimately acknowledged the primary importance of these observations on his death bed, this had no effect on the momentum of scientific opinion of his peers, and Béchamp and Bernard were ignored with the arrogance of the righteous. This political prejudice survives to this day.

What does this mean to you? It means two things:
1) people come in virus-susceptible and virus-resistant forms, and
2) viruses have variable virulence (viruses will become more virulent in people and animals that are virus susceptible and less virulent in people and animals that are virus resistant).

As an example of these observations, consider that most of the most virulent viruses in recent history come out of Asia. Remember the Hong Kong flu, the Asian flu, and SARS? Remember the bird flu? Swine flu? They all developed in highly populated rural regions of China, where there is a widespread deficiency of selenium in the soil. Selenium is a vital trace element, essential for many critical enzymes.

This lack of selenium in this region of China causes a heart disease, called Keshan disease (endemic dilated cardiomyopathy), after the county in China, but it also breeds abnormally virulent viruses, in both humans and animals (Tan et al. 2002; Li et al. 2014). These viruses were virulent enough to kill people. When they entered the United States, they also killed people. But within weeks to months, the death rates began to drop, and eventually, after many months, the virulence was decreased to the point that people rarely died from these viruses.

Selenium, as I will explain more on the next few pages (and page 47 and following), is a key nutrient for viral resistance. And viruses are known to become more virulent in selenium-deficient people [Beck et al. 2003; Ren et al. 2004]. So selenium deficiency is one of a few dozen pro-viral risk factors that can be

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38 The patents on PCR testing have recently expired, resulting in significant drops in cost.
39 There are regions of Africa with low selenium levels, which are not so thoroughly studied as China for the correlation between selenium levels and viral virulence of ebola, which roughly correlates with mortality rate (how many infected people die versus survive). There are also low selenium levels in other areas of the world, like the southern island of New Zealand, and in Croatia. In North America, the Pacific northwest, Michigan, northeast states, and Florida are low, and the eastern Rocky Mountains and plains west of the Mississippi river (nearest South Dakota and Iowa) are highest.
corrected by simple dietary changes (eating liver, kidney and Brazil\textsuperscript{40} nuts) or by taking selenium supplements.\textsuperscript{41}

Although the specific mechanisms of immune function were not understood back in Pasteur’s time, modern scientists have accepted that the strength and potency of the immune system is a very real and important aspect of host resistance or susceptibility. This is only one side of Béchamp’s and Bernard’s views. The other side, which is still ignored today, is that the metabolic state of host directly and critically influences the invading organism. The invading organism reacts to the state of the host, becoming more virulent or less virulent as the situation warrants (from the microbe’s survival perspective, of course). The degree to which this host-influence contributes to herpes flare-ups is neither widely nor fully appreciated. I believe that it is far, far from trivial.

The good news is that many common vitamins, minerals, nutrients and foods have specific effects on metabolic systems which can result in increased and decreased susceptibility to viral disease (or increased or decreased viral virulence). Through selective use of dietary supplements and foods, viral disease can be significantly decreased.

**Metabolic Balance**

One of the pioneers in the study of the relationship between metabolism and disease was the late Dr. Emanuel Revici (pronounced reh-vee-see). He measured tissue pH (acidity and alkalinity) and found that pH disturbances were correlated with metabolic shifts in a well-organized manner. He described a linear metabolic continuum as “anabolic” in one direction and “catabolic” in the other.

Revici’s definitions of “anabolic” (healing, repair and growth-oriented metabolism) and “catabolic” (digestion and energy-production metabolism) are quite different from the modern English meanings of these terms. Since anabolic metabolic systems are also anaerobic (not oxygen based) and produce alkalinity and the catabolic systems are aerobic (oxygen based) and produced acidity, I prefer to describe Revici’s metabolic continuum as anabolic-anaerobic-alkaline versus catabolic-aerobic-acidic. This tends to break the connotations we automatically invoke when reading words for which we have well-established understandings.

According to Revici’s views, health is defined by a dynamic balance between anabolic-anaerobic-alkaline systems and catabolic-aerobic-acidic systems, the dominance of which swing back and forth on a 24-hour

\textsuperscript{40} The selenium content of Brazil nuts is accidental, not essential. Plants do not need selenium. So the selenium content of Brazil nuts is severely variable depending on the selenium content of the soil and its pH. The USDA lists the selenium content of a single Brazil nut at 96 mcg. But when analyzed by region, the selenium content per nut varied from 10 mcg to 325 mcg in one study, and from less than 1 mcg to more than 2000 mcg in another report. The high selenium Brazil nuts were from the Amazonas and Amapa regions (300 and 230 mcg/nut, respectively) and the low selenium nuts were from the Roraima, Acre and Mato Grosso regions (50, 15 and 10 mcg/nut, respectively). Where are your Brazil nuts grown?

\textsuperscript{41} I supplement 100-200 mcg of sodium selenite solution per day. One drop of 100 mcg in the AM and an optional 100 mcg in the PM. Sodium selenate (notice the “a” instead of an “i” in the name) is an equally effective selenium supplement. Selenite and selenate are both hypoallergenic, unlike the selenomethionine and selenocysteine that are heavily promoted in health food stores. Selenite and selenate are not stored, so their toxicity is acute. It takes about 2000 mcg in a single dose to produce a hypoglycemiac reaction in an average person and 600 mcg in the most susceptible people. Since it is not stored, I can take it once, twice or four times per day to achieve selenium repletion without any concern of selenium toxicity. Selenomethionine and selenocysteine are not acutely toxic but are cumulatively toxic. They build up in the body and subtly poison methionine-containing and cysteine-containing enzymes in a cumulative-dose manner. Because selenomethionine and selenocysteine are soluble in blood and hang out for extended periods, they are promoted as having superior bioavailability. This is absolutely not true for selenomethionine, which must be broken down to release the selenium. But they are, in essence, a timed-release form of selenium. But because they are often made from yeast, they can have allergic and inflammatory effects. Selenite and selenate are hypoallergenic, and 100 times cheaper. That’s why they are my preferred form of selenium.
basis. Before I go any further with Revici’s insights into the virulence of infections, I must first point out that metabolic acidity and alkalinity is distinctly different from the acidity or alkalinity of the food itself.

Acidic foods like lemons and oranges are quite tart to the tongue, but they produce a strongly alkaline influence when metabolized. This is why citrus fruits are said to have “alkaline ash.” Likewise, fats, meats and grains do not taste tart at all, but they have acidic ash (they produce acidity when metabolized). It is the final metabolic consequences of foods and nutrients that determine their acidic or alkaline classification.

Secondly, I must clarify that pH is not simply a one-dimensional issue of acidity and alkalinity. There are numerous metabolic compartments in the human body, and pH effects in one do not necessarily correlate with pH effects in another. In fact, it is often the case that they may be directly opposite. In other words, a particular dietary substance might alkalinize the cellular environment and acidify the blood at the same time.

So in a very real sense, speaking of foods as being inherently “acidifying” or “alkalinizing” is potentially misleading and possibly inaccurate unless one identifies the particular metabolic compartment which is being considered. To make matters even more complicated, your personal metabolism and immune system also influence the effects of foods on your system. If you are allergic to a food, it will acidify higher levels of your body’s fluids even if the food would be alkalinizing in another person who is not allergic to that food.

So I must caution you not to confuse Revici’s “acidifying” and “alkalinizing” influences with those of Macrobiotics, Metabolic Typing, or other pH systems. Revici measured wound pH, which is an interstitial fluid which oozes from the spaces between cells. His discussions of the pH characteristics of metabolic systems refer to cellular (and subcellular) pHs, which he was never able to directly measure. And these are quite different from blood pH effects, which are commonly discussed by other pH models.

Revici discovered that there was a daily (circadian) metabolic rhythm in healthy people that was determined by the ebb and flow of these two, opposite metabolic systems. In humans, the anabolic (anaerobic, alkaline) metabolic systems dominate at night and the catabolic (aerobic, acidic) systems dominate during the day. This metabolic “excess” of acid and alkali can sometimes be measured in urine with special pH papers. I’ll discuss this in more detail later.

In people with chronic degenerative diseases, especially cancer, Revici discovered specific disturbances in the circadian metabolic rhythm. Almost all patients exhibited strong suppression of the strength (amplitude) of the metabolic rhythm (i.e., the pH swings were damped out). Healthy people would tend to swing 2 full pH units per day, while cancer patients tended to shift less than 1 pH unit, and sometimes less than half a pH unit. Furthermore, the average pH (the baseline) shifted dramatically away from normal (approximately 6.0 to 6.2) towards either acidic or alkaline values. Often the magnitude of the shift was a half-pH unit. The direction of the shift correlated with 1) the type of cancer, and 2) its response to treatment. Anabolic/catabolic balance also affects the course of infectious diseases. Revici discovered that anabolic influences increased the virulence of bacteria! When animals were fed extra cholesterol (an anabolic metabolic influence), they became more susceptible to anthrax (a bacterium). When animals were fed extra fatty acids (a catabolic metabolic influence), they became less susceptible to anthrax. It works the same way with viruses (and oppositely with most fungi).

The anthrax bacteria also reacted to the change in metabolic state of the animals. Anthrax bacteria extracted from anabolic animals were much more virulent than normal anthrax bacteria. It would kill normal animals much more rapidly (“normal” means animals that were not exposed to either excess cholesterol or excess fatty acids). Similarly, anthrax from catabolic animals became less virulent. After several generations in catabolic animals, even anthrax became non-lethal to normal animals.
The implications of these observations to humans are profound. I have noted that adults who catch colds and flus from children seem to have far more debilitating symptoms than other adults who catch colds or flus from other adults. That could be because children are much more anabolic than adults. School teachers and parents frequently attest to the special virulence of kid-vectoried bugs.

I have observed that individuals with anabolic dominance (or catabolic deficit) are especially prone to viral problems, including herpes. They have more severe symptoms and they have more frequent outbreaks. Likewise, individuals with catabolic dominance (or anabolic deficit) seem to only rarely have problems with viruses. So catabolic agents should be helpful and anabolic agents counterproductive. That is exactly what I have seen.

How does BHT influence metabolism? It’s catabolic.

Is this a coincidence? Maybe, but I don’t think so. This pattern is too consistent. Vitamin E is anabolic and supplementation in high amounts (200-800 IUs) seems to be associated with an increase in the frequency of herpes outbreaks. Vitamin A is catabolic, and it appears to significantly help BHT work better.

Vitamin D is also catabolic and antiviral. Although it has received little popular or scientific recognition until recently, the emerging vitamin D literature is suggesting a powerful role for vitamin D in not only decreasing influenza mortality but in explaining the “seasonal factor” that makes influenzas more deadly in the depth of winter and so strongly latitude dependent. In my opinion, pretreatment with high-dose vitamin D$_3$ (50,000 IU per day for 10 days in the fall, or 5,000 IU per day for the duration of the winter) will be found to be more effective in preventing flu (even bird flu and swine flu) than any and all flu vaccinations, with or without antiviral drugs.

Vitamin D deficiencies have also deepened in recent decades due to pronouncements by dermatologists that sun exposure causes skin cancer. Actually, this claim is not true. In fact, regular sun exposure decreases risks for the vast majority of skin cancers, and especially those that are the most difficult to treat. It is true that sun over-exposure (sunburn, erythema, reddening of the skin) is a significant skin-cancer risk. But this does not apply to regular sun exposures and absolutely does not justify avoidance of the sun entirely. The best advice is to maintain constancy of sun exposure. But this message has been absent. By creating abject fear of the sun, dermatologists have put everybody at risk, fortified their business (increased the number of their skin-cancer clients), and increased overall viral susceptibility in the general population. Vitamin D levels are conservatively less than half of what they should be, and may be a fourth of the levels that promote optimum health. If true, this would make vitamin D deficiency roughly as common as magnesium deficiency.

Hypericin (a substance found in St. John’s wort) is very strongly catabolic. It is an oxygen catalyst which directly potentiates aerobic metabolic processes. And it has strong antiviral influence in many viral diseases.

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42 Many individuals have vitamin D levels of less than 20 ng/ml, when 40 ng/ml is the bottom end of the recommended range. I suggest that 50-55 is a good minimum. Some experts feel that 60-80 ng/ml is the ideal target range. (Lifeguards, for example, have D levels above this range.) Some testing companies report the results in nanomoles (nm) instead of nanograms (ng), which makes the numbers 250% higher (50 nm/ml, 100 nm/ml, 125-140 nm/ml and 150-200 nm/ml, respectively).

43 Normal oxygen in the air is triplet oxygen, which has two unpaired electrons. Triplet oxygen is not only a free radical form of oxygen, it is a double free radical. With the addition of a small amount of energy, triplet oxygen can convert to singlet oxygen, where the two free radical electrons pair up. Singlet oxygen is not a free radical. This is important because singlet oxygen is not as reactive towards single-electron targets and has a longer duration of activity. With ozone, the energy difference between triplet and singlet states is reversed; the singlet ozone being the lowest energy state and the triplet ozone being higher energy. This makes ozone a very selective oxidant in its ground state, and accounts for its unique therapeutic properties.
**Figure 1: Metabolic character of foods, nutrients, drugs and chemicals**

<table>
<thead>
<tr>
<th>Categories of Revici’s metabolic influences</th>
<th>aerobic-acidifying-catabolic</th>
<th>anaerobic-alkalinizing-anabolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>vitamins</td>
<td>vitamins A and D</td>
<td>vitamins E and K</td>
</tr>
<tr>
<td></td>
<td>vitamins B₆ and B₁₂</td>
<td>vitamins B₁, B₂, B₃ and B₅</td>
</tr>
<tr>
<td></td>
<td>choline, inositol, folic acid</td>
<td></td>
</tr>
<tr>
<td>amino acids</td>
<td>glutamate and aspartate</td>
<td>arginine and lysine</td>
</tr>
<tr>
<td></td>
<td>methionine and cysteine</td>
<td>tryptophan and histidine</td>
</tr>
<tr>
<td></td>
<td>carnitine and acetyl-L-carnitine</td>
<td></td>
</tr>
<tr>
<td>elements and minerals</td>
<td>magnesium, calcium, strontium</td>
<td>sodium, potassium and lithium</td>
</tr>
<tr>
<td></td>
<td>oxygen, ozone, hydrogen peroxide</td>
<td>chloride, bromide, iodide, fluoride</td>
</tr>
<tr>
<td></td>
<td>superoxide, hyperbaric oxygen</td>
<td>chromium and iron (reduced)</td>
</tr>
<tr>
<td></td>
<td>selenium and sulfur (reduced)</td>
<td>zinc</td>
</tr>
<tr>
<td></td>
<td>manganese, vanadium and copper</td>
<td></td>
</tr>
<tr>
<td>heavy metals</td>
<td>lead and tin</td>
<td>cadmium and mercury</td>
</tr>
<tr>
<td></td>
<td>arsenic, antimony and bismuth</td>
<td></td>
</tr>
<tr>
<td>lipids</td>
<td>fatty acids</td>
<td>fatty alcohols</td>
</tr>
<tr>
<td></td>
<td>polyunsaturated fatty acids</td>
<td>cholesterol and other sterols</td>
</tr>
<tr>
<td></td>
<td>testosterone and progesterone</td>
<td>estrogen and cortisol</td>
</tr>
<tr>
<td>chemicals</td>
<td>phosphoric acid</td>
<td>glycerol, ethanol, sugars</td>
</tr>
<tr>
<td></td>
<td>vinegar</td>
<td>sodium bicarbonate (baking soda)</td>
</tr>
<tr>
<td></td>
<td>magnesium thiosulfate</td>
<td>salt (sodium chloride and sea salt)</td>
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<tr>
<td></td>
<td>BHT</td>
<td>alcohol (distilled spirits)</td>
</tr>
<tr>
<td>drugs</td>
<td>antibiotics</td>
<td>pain killers, aspirin (NSAIDs)</td>
</tr>
<tr>
<td></td>
<td>sulfonamides</td>
<td>narcotics (opiates)</td>
</tr>
<tr>
<td></td>
<td>chloroform</td>
<td>benzodiazepines and antidepressants</td>
</tr>
<tr>
<td>foods, spices and herbs</td>
<td>meat, nuts and hard cheeses (aged)</td>
<td>dairy (and soft cheeses, yogurts)</td>
</tr>
<tr>
<td></td>
<td>preserved meats</td>
<td>green leafy veggies</td>
</tr>
<tr>
<td></td>
<td>whole grains</td>
<td>refined grains, wine and beer</td>
</tr>
<tr>
<td></td>
<td>fried and hard-boiled eggs (hard yolks)</td>
<td>soft-boiled and raw eggs (liquid yolks)</td>
</tr>
<tr>
<td></td>
<td>mayonnaise, butter and oils</td>
<td>soy sauce and salt</td>
</tr>
<tr>
<td></td>
<td>hypericin from St. John’s wort</td>
<td>chocolate, coffee</td>
</tr>
<tr>
<td></td>
<td>ginger</td>
<td>rutin, pollen, alfalfa, kelp</td>
</tr>
<tr>
<td></td>
<td>tomatoes</td>
<td>most herbs (and tobacco)</td>
</tr>
<tr>
<td></td>
<td>cranberries, cherries, pomegranates</td>
<td>vegetables (almost all)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>fruits (almost all)</td>
</tr>
</tbody>
</table>

Similar things can be said of negative ions, which is another term for superoxide radicals. Negatively charged superoxide ions are produced by electrons combining with oxygen molecules. So $O_{2}$ becomes $O_{2}^-$.  

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Negative ions are found at the beach, where waves mix seawater with air, near waterfalls, where air and water are mixed, and during storms, when rain falls through the air. Negative ions make people happy. They also make people antiviral. Like oxygen, superoxide is an essential component of aerobic metabolism. About 15% of our oxygen metabolism is superoxide dependent, including the pathway for the synthesis of serotonin, the “happiness neurotransmitter.” Positive ions from dry desert winds blowing across shifting sands deplete negative ions and cause irritability, depression, increases in traffic accidents, hostility, fights and homicides. The Santa Ana winds of Los Angeles, the Sirocco winds of northern Africa, and the Foehn winds of Germany are well known examples of positive ion-rich winds.

In addition to oxygen and superoxide, hydrogen peroxide, hyperbaric oxygen and ozone are catabolic, too. Each has significant literature documenting antiviral effects.

Vitamins B6 and B12 are the only catabolic B vitamins, and I (and others) have observed that B12 is very effective at augmenting BHT efficacy in people for whom BHT works poorly by itself.

B1, B2, B3 and B5 are the anabolic B vitamins, and they do not seem to be of any particular help, at least beyond the low doses that would correct a simple deficiency of any of them. Although B1, B2 and B3 are mitochondrial nutrients and essential for energy production, which is catabolic and antiviral, they are, overall, anabolic in character and not known for their antiviral effects.

Two common causes of anabolic dominance in people are vegetarianism and subclinical hypothyroidism (hypometabolism).

Vegetarianism

Vegetarians and vegans tend to become anabolic dominant and overly alkaline (at the cellular level) because most non-meat foods are anabolic/anaerobic/alkalinizing. The reasons for this are too complicated to explain fully in a book of this size, but I will mention two factors that have a lot to do with it: 1) the low-sodium, high-potassium content of vegetable foods, and 2) the low levels of polyunsaturated fatty acids that tend to occur in vegetables.

Grains are the primary exception, and this is largely due to their high polyunsaturated fatty acid content. However, not all grains are acidifying. So vegetarians who do not emphasize sufficient quantities of catabolic grains or catabolic exercise can easily drift into anabolic territory, with resulting cellular alkalinity. Such a state might be better described as catabolic deficient, or aerobically impaired, but the important take home message is that complementary metabolic systems are out of balance with respect to each other.

Before going on, I’d like to clarify my use of the terms alkalinization and acidification with those of traditional Eastern medicine, for which alkalinity is always promoted as essential to health and acidity a cause of degenerative disease. This “traditional” Chinese-medicine precept only applies to tissue pH. Acid tissue is associated with disease and degeneration. But acid tissue can also be associated with alkaline cells. Alkaline cells are hypometabolic cells, and they produce disproportionate amounts of lactic acid, which when exported from the cell produces extremes of tissue acidity, which is bad. Cells with normal metabolism are acidic cells and they make predominantly carbon dioxide (i.e., carbonic acid). So there is little lactic acid to be exported into the surrounding tissue to accumulate and cause the traditional-Chinese-medicine “acidity” phenomenon. Carbon dioxide does not accumulate. It diffuses as a gas, and the bicarbonate anion is easily and efficiently transported to the lungs for outgassing. In other words, lactic-acid acidity accumulates locally, and carbon dioxide acidity dissipates globally. This is the difference between acid-as-disease and acid-as-health, respectively. So the traditional-Chinese-medicine maxim that

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44 Please see also take a look at Revici’s periodic table of the elements which maps out the pattern of anabolic and catabolic elements.
“body alkalinity is good” is because it facilitates healthy cellular acidity! The pH issues of health are not one dimensional. So do not be misled or concerned by this apparent discrepancy.

With vegetarianism, I have to mention B12 again. Vegetarian foods do not contain any B12. To be fully accurate, B12 may be found in small quantities in vegetarian foods due to the presence of bacteria and insect parts, which may “contaminate” the food, especially in poorer countries. However, this animal-source B12 is not an intrinsic part of the vegetarian food itself, and it tends to be absent in vegetarian foods grown and harvested in affluent countries, like the United States, where the cleaning and storage of agricultural foods tends to be more thorough. Either way, strict vegetarians run a significant risk of B12 deficiency. And B12 is an important catabolic nutrient, one with antiviral properties.

Fortunately, modern fermentation methods produce large amounts of microorganism-derived B12 at quite reasonable costs. So affordable B12-containing dietary supplements are available to offset this particular vegetarian lifestyle risk.45

**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.

**Subclinical and Clinical Hemochromatosis (iron-overload syndrome)**

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 the infection 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

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45 Raw microbial-source B12 contains B12-analogs that are anti-metabolites in humans. Anti-metabolites interfere with regular vitamin function. Commercial manufacturers of B12 remove these anti-metabolites enough that RDA-level doses are safe, but when dosages are ten, a hundred or a thousand times higher, the level of anti-metabolite may be troublesome. In addition, some medical tests do not discriminate human B12 from non-human B12, so be deliberate in your choices all around. See also the parallel story of B6 antimetabolites mentioned in footnote 32.
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 testing. It’s not expensive. Men should be 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. 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.

**Type-1 and Type-2 Copper Deficiencies**

Copper deficiency may have pro-viral consequences (Yörük 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.

**Hypothyroidism**

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

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46 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 blood letting. 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.

47 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.
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 setting 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.

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.

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48 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.
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.

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 nutrure 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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49 It is a USA tradition (and arrogance) to report temperatures in Fahrenheit. For readers in other countries, you can approximate the Fahrenheit written 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.
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.

**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

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50 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.
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 the 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.\(^{51}\) 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.\(^{52}\)

**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 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 E\(_2\)) 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

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\(^{51}\) The emeramide-mercury complex is so strong that EDTA cannot dislodge it.

\(^{52}\) Emeramide is also known as NBMI, OSR, BDTH2 and Irminix.
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.

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 dissynchrony (timing disturbance).

**Catabolic Adjuncts**

If the metabolic hypothesis is correct, then other compounds with similar effects on catabolic metabolism should exhibit synergy with BHT against viruses. Although this hypothesis has never been systematically

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53 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.
studied, several physicians and more than a dozen people have noted that vitamin B\textsubscript{12} (either oral or injected) seems to markedly aid in the control of herpes. B\textsubscript{12} and B\textsubscript{6} are the catabolic B vitamins. Vitamin A is a catabolic lipid-soluble vitamin, and it seems to help not only with herpes control, but with general immune reactions to infections of all kinds.

Vitamin E, which is an \textit{anabolic} fat-soluble vitamin, has been reported to aggravate herpes flare-ups when taken in amounts many times greater than the 30 IU recommended daily allowance, but not when only 30 IU are taken. Although this latter finding is highly questionable due to its methodologies (it was a questionnaire-based study), I felt that it should be mentioned because it fits the pattern in a mild way.

And, finally, it has been noted that antibiotics (which are catabolic) have a therapeutic effect against infections that later prove to be viral, even though there is no known mechanism for this action—other than the general catabolic property of antibiotics, of course.

A generalized list of the metabolic effects of numerous substances appears on page 35. Substances were classified based on their general effect on metabolism, not any quantified antiviral effect.

**Selenium, China, Africa and Ebola**

Selenium is a highly effective catabolic-aerobic-acidifying adjunct to antiviral therapy (see also page 57). 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. 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 presentation 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 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, the 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 57). As it turns out, poor selenium status is also strongly associated with the ebola virus and ebola lethality in Africa.

**Ebola Update**

The below-left map of ebola outbreaks from 1976 to the present (CDC) shows four different ebola strains, their distributions and the approximate number of cases. To the right of this 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 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 spectre of ebola outbreaks in non-African countries and the spread of ebola into the world population.

Unlike public-health officials, who do not know about BHT, viral virulence and host resistance, you have access to techniques to minimize your risks. This not only relates to the use of selenium, which is known to be widespread deficient in the Democratic Republic of the Congo (formerly Zaire), where ebola first appeared, but also to the use of vitamin C and collagen therapy, for minimizing hemorrhagic outcomes (internal loss of blood and body fluids). Although ebola is known for causing hemorrhagic effects, this is not always present, and may not even show up in the majority of cases. There are many reasons for this, many of which are the factors affecting viral virulence and host susceptibility described in this book. But there is a very real mechanism connection between selenium (the active coenzyme in glutathione peroxidase), glutathione status (the primary antioxidant of the human body), vitamin C (the secondary antioxidant of the human body, and scurvy (the vitamin C deficiency state characterized by widespread bleeding and clotting pathologies). Vitamin C status is highly variable in both rural Africa, where malnutrition is widespread, and in urban area, where over-consumption of refined foods and insulin resistance are widespread.

Through the lens of this mechanism, hemorrhagic fevers induce acute scurvy in days instead of weeks or months. The accomplish this by sequestering selenocysteine in useless proteins and inducing an acute selenium deficiency. This selenium deficiency sabotages antioxidant defenses and puts a heavy strain on vitamin C reserves, which causes a dramatic shift in the body’s redox potential. Normally, this potential is highly reduced by efficient recycling of both glutathione and vitamin C. However, with an acute infection, elevated inflammation and high cytokines, the body’s NADH- and NADPH-generating systems for recycling glutathione and vitamin C falter, and ascorbate levels quickly fall into the scurvy and acute scurvy levels. Without vitamin C in the extracellular spaces, collagen maturation fails. Without vitamin C in the blood, platelets aggregate, forming miniclots. Without vitamin C in the blood stream, capillaries become fragile. Membranes start to deteriorate. Red blood cells rupture, adding “loose iron” to the destruction of vitamin C and the aggravation of oxidative stress (see iron chapter on page 37). The ultimate result is a complete loss of tissue integrity, massive edema, fluid leakage, bleeding and death by bleed out. The suddenness of this unique manifestation of scurvy is unprecedented. And it is a selenium-dependent phenomenon.

The good news is that this is not so hard to fix. Selenium and vitamin C are easily made available through nutrition supplementation (for vitamin C, see page 52).

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54 Deficiencies of selenium are not quite as severe in Africa as they are in China. African countries with the lowest selenium levels are Zambia, the Democratic Republic of the Congo, Zimbabwe, Malawi, the Central African Republic, Botswana, Mozambique, Kenya and Ethiopia. None of the current west-African countries are on this list. But of the west-African countries, Liberia and Benin have the lowest levels of selenium, and Senegal, the Ivory Coast, Ghana, Burkina Faso and Nigeria have the highest levels of selenium.

55 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].
If public health officials bother to look, I predict that they will find a linear correlation between the selenium status of patients and the mortality of ebola. In other words, in the current selenium-deficient regions of ebola outbreaks, the official ebola death rate varies from 50% to 90%. I think researchers will find that the people with the highest selenium levels will have 50% mortality and those with the lowest selenium levels will have 90% mortality.

I also predict that loss of vitamin C will be universally associated with the timing of the emergence of hemorrhagic symptoms.

But because the selenium status of people with HIV infection is a sore point with the US Centers for Disease Control and National Institutes for Health, who have a long political history of under-prioritizing research grants dealing with the role of selenium deficiency in the transition from HIV infection into 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 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 Constitutional 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 (as opposed to (1) acute non-hemorrhagic viruses, and (2) chronic viral diseases) requires two therapeutic strategies, not just one. To decrease viral virulence and increase in host viral resistance with a combination of BHT, nutrition and/or metabolic therapies 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 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 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

56 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].

57 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.
“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.\(^{58}\)

If you have the foresight to stockpile vitamin C and IV bags,\(^ {59}\) do not leave such essential supplies in your doctor’s offices where they could be “confiscated in the public interest” during an “emergency” or 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 do?

In other countries, 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 (see page 52). Swine flu is a lipid-enveloped viral disease, and in Alan’s case, it progressed to coma, a respirator 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 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. In hours, Alan Smith woke up. In a day, his lungs cleared.

Despite his recovery, the hospital administrators stopped the vitamin C. They “reasoned” that his recovery could not have been from the vitamin C and it was no longer ethically permissible to give a non-dying man a worthless therapy. Alan lapsed back into a coma. The family had to smuggle vitamin C into the hospital to save Alan’s life.

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.

**Vitamin D\(_3\) and Calcium**

Calcium is another one of the four most catabolic-aerobic-acidifying elements that Dr. Emanuel Revici found (see page 57). Calcium’s efficacy is strongly connected with vitamin D, the “sunshine vitamin.” It is also influenced by magnesium and vitamin K\(_2\). Deficiencies of vitamin D\(_3\), K\(_2\) and magnesium are common in the United States and the rest of the world.

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\(^{58}\) This is what happened to Allan Smith in New Zealand after contracting swine flu and falling into a coma. See further information and footnotes on pages 43 and 44.

\(^{59}\) 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.
Vitamin D₃ is deeply associated in the literature with the seasonal variations of viral epidemics. This seasonality has been noted with influenza outbreaks dating all the way back to the Spanish flu outbreak of 1918-1919, and more recently with the coronavirus outbreak of 2002-2003.⁶⁰

Host resistance to viruses is also vitamin D dependent. Vitamin D is really a hormone that raises metabolic rate, but it’s called the sunshine vitamin because it is derived naturally from the ultraviolet-B rays of the sun. Because of this sun connection, vitamin D levels follow the seasons, peaking in the northern hemisphere about two months after the summer solstice (late June) and troughing about two months after the winter solstice (late December). That makes November through March the flu months, and May through September the anti-flu months.

See how it operates in historical accounts. The Spanish flu epidemic of 1918-1919 actually started in the late spring of 2018, but mortality paused for the duration of the summer of 1918 to re engages in October, November and December with unprecedented death rates. More recently, the SARS outbreak of 2003 started in February, was attenuating by May and was fully attenuated by July, after six months in the USA. The 2020 coronavirus outbreak began in the depth of the winter, and if history repeats itself, it will attenuate before the end of the coming summer.

**High-Dose Vitamin C Therapy**

There is another technique, pioneered by Dr. Klenner⁶¹ for polio back in the 1940s, that is highly beneficial for 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 without neurological impairments, the vast majority in only 3-5 days.

This unprecedented clinical report has been ignored by public-health officials for 70 years. Alternative and complimentary-oriented physicians who have adopted Klenner’s methods affirm that it works exactly as Klenner reported.

In New Zealand, the family members of a man stricken with swine flu found out about vitamin C treatment for viral diseases. Their father, Alan Smith, an Auckland farmer, had become infected, and had fallen into a coma. The family members asked that vitamin C be tried. The hospital refused. He stayed in a coma. Finally, the hospital wanted to “pull the plug” on his respiration machine. The family objected because vitamin C had not been tried. After several backs and forths, the hospital reluctantly agreed to administer vitamin C by IV. In hours, Alan Smith woke up. In less than a day, his lungs had cleared on x-rays.

The hospital’s staff’s reaction? Stop the vitamin C. Their reason? It was no longer ethical to give a worthless treatment to a person who was no longer dying. (Watch the 60 Minutes story⁶² on this case if you do not believe me.) The man lapsed back into a coma.

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⁶⁰ The 2019-2020 coronavirus outbreak in China is being promoted as a brand-new virus, but in point of fact, the SARS outbreak of 2003 was caused by a coronavirus. It was named, however, by its symptoms (severe acute respiratory syndrome) instead of by its virus name. Despite the hype, this new coronavirus outbreak shows little sign of being a danger of a different magnitude. It is just one more viral variant that shows greater lethality in a selenium-deficient population.

⁶¹ 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).

⁶² You can find the 60-Minutes story on YouTube (http://www.youtube.com/watch?v=VrhkoFtOMII). 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= zlKDBoiXnE). However, the best single information resource is a DVD movie / documentary about the story (Living Proof, 2-DVD set) that
The hospital refused, again, to give vitamin C.

Fortunately, the family learned more of what I am telling you. They “smuggled in” vitamin C in a new, liposomal form, and their father again recovered consciousness. They checked him out of the hospital to prevent 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 refuse to administer.

As of 2016, the man was still alive and had resumed ranching.

As of 2017, vitamin C is not even being considered as a “possible” treatment for ebola by Western public health officials. They have 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. 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.

Do you want such people to control your public health policy?

How about your personal treatment choices?

**Measles and Vitamin A**

Here is a great example of the information in this book in action. Vitamin A, one of the strong antiviral nutrients discussed in this book, has a profoundly positive effect on clinical course of the most severe measles infections.

When very high doses (400,000 IU, approximately 300 times the RDA) of vitamin A were administered in routine clinical practice, the hospital stay was decreased by 20%, the need for intensive care was reduced by 60% and deaths were cut by 68% (Hussey and Klein, 1993). No adverse effects were observed.

A previous double-blind study of 189 children hospitalized with severe measles found comparable results (Hussey and Klein, 1990). Of the twelve children who died, ten were in the placebo group and two were in the high vitamin A group.

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63 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.”

64 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.”
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 35. 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 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 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 nutrients 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-catroten 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).

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65 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.
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. There are nuances to this approach that I cannot do justice to in this book.

To illustrate the breadth of “invisible” influences, consider the case of a man taking lots of vitamins and antioxidants for his health who develops persistent impotence. He wonders what might be causing it. We talk on a regular basis about his dietary changes, his temporary discontinuance of his regular supplements, his addition of other supplements, his exercise, sleep, mood, etc. He tries everything he can think of. More than a year goes by without results.

Then he mentions that his impotence vanished for two days at the end of week-long fishing trip to Mexico. So I ask what was different from his routine in Mexico compared to the US. One item came up immediately: diet Coke. He had never considered it in all that time, but he always drank diet Coke in the US and never did in Mexico. And I never thought to ask him about diet soda consumption. Ten days later, he calls up and, presto change-o, his impotence is gone. It never came back.

His exposure to aspartame was an invisible influence in his life that was lost in his automaticity. It was like the air that we all breathe without noticing it, or the water in which fish swim which is everywhere and taken for granted by the fish. Everybody and anybody can have such an influence in their lives. It’s hard to see, except in 20:20 hindsight.
Revici’s Periodic Table

This illustration of Revici’s periodic table highlights the anabolic and catabolic nature of the elements in red and blue colors (see the green key). 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.

This is why much of this book is devoted to those elements. Oxygen ties to aerobic therapies are mentioned starting on page 34 and the chart on page 35. Sulfur ties to glutathione, cysteine, N-acetylcysteine and thiosulfate (see page 35). Selenium ties to glutathione peroxidase (the primary glutathione recycling enzyme) and viral resistance. And calcium ties to vitamin D₃ (see the metabolic Balancing chapter on page 32, and pages 47 and 54) and possibly vitamin K₂.

Not quite as strong, but measured, were magnesium, strontium and barium (in the same column as calcium), and manganese, copper, lead, cerium, thorium and uranium.⁶⁶

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⁶⁶ Revici’s interest in barium, cerium, uranium and thorium (and cesium, tungsten and praseodymium) was due to his study of the way the levels (compartments) of the body related to the levels (periods) of the periodic table. The association of the 6th period toxic elements (mercury, thallium, lead, bismuth) with the sub-nuclear environment means that detoxification of such elements is the greatest challenge. Note: these 6th period elements are also called the third-period transition metals by chemists and physicists because the transition metals start with the 4th period. In other words, first-period transition metals are 4th period elements, second-period transition metals are 5th period elements and third-period transition metals are 6th period elements.
From the pattern, Revici noticed that the anabolic-catabolic character of elements had to do with the even or odd number of electrons of each element. Odd elements were anabolic and proviral and even elements were catabolic and antiviral. From this he extended his assessments to “theoretically” anabolic and catabolic elements, which were even and odd but not experimentally measured.

The remainder of this book will focus on the primary technical literature.

2016 Note on the Prevention and Reversal of Alzheimer’s Disease

The appreciation-process note from me on page 6 is in no way limited to viral diseases. If you know somebody dealing with mild dementia or early-stage Alzheimer’s disease, or even somebody dealing with a relative with full-blown Alzheimer’s disease, check out my nine-part video series on “Prevention and Reversal of Alzheimer’s Disease” on YouTube (www.youtube.com/user/swfowkes/videos). It takes 80 minutes to watch all nine parts (each is less than ten minutes long). To watch them all in order, select the 1-9 playlist, or use the following link:

http://www.youtube.com/watch?v=oX6RG6ky0yU&list=PL620DC3CA557284EB&index=3).

Contrary to public belief and the pronouncements of pretty much every mainstream medical organization on the planet, Alzheimer’s disease is not only reversible, it is relatively easily reversible. This may be shocking, but have a little faith (and some considerable skepticism) to suspend your disbelief for long enough to watch the videos for yourself before dismissing my statement to the contrary.

The reason that this is not well known is that the researchers who first figured out the critical step in the mechanism were not Alzheimer’s researchers, but rather dental scientists at the University of Kentucky and the University of Calgary. Dental scientists get no respect in the field of Alzheimer’s disease.

The smoking gun, in one sentence, is that all of the enzymes that are known to be strongly inhibited in Alzheimer’s disease are sulfhydryl enzymes, and all the non-sulfhydryl enzymes are only slightly inhibited, or collaterally inhibited, if inhibited at all. In other words, one entire class of enzymes malfunctions. Neatly, meaning no known exceptions on either side of the class distinction. This malfunction sabotages the brain’s back-up energy supply, disassembles the brain’s transportation highways, and freezes the brain’s structural biorhythm.

Without a redox understanding of what a sulfhydryl enzyme is, specialist Alzheimer’s researchers have not yet seen the significance of this startling finding. If you read this book, you will get an understanding of

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67 Note that the even and odd rule reverses (1) at the boundary of the transition metals, where electrons have completely filled the orbitals of the s and p shells and start to fill the orbitals of the d shells, and (2) again, for the rare-earth elements which involve the orbitals of the f shell. So the Ca-to-Sc increase of one electron does not result in a change of metabolic character, and the Zn-to-Ga one-electron increase remains anabolic. I have no explanation for why anabolic-catabolic is odd-even for the elements of the second and third periods, even-odd for the transition metals, and odd-even for the rare-earth elements. But I do recognize that there is a consistent pattern for which there is no known exception.

68 The context of “relatively easily reversible” is to Parkinson’s disease, which is more difficult to reverse than Alzheimer’s disease. I believe this is because the permanent and irreversible damage that occurs with Alzheimer’s disease, if any, starts at its onset, whereas with Parkinson’s disease, it accumulates for years or decades before the symptoms become blatant enough for the diagnosis.
the redox mechanisms underlying the antioxidant defense system. If you want a short cut, watch the nine-part video series.

It might also surprise you to learn that some cases of Alzheimer’s disease have been reversed by perispinal injections of the FDA-approved prescription drug Enbrel (a TNF-alpha inhibitor). In one notable case, it took only two hours. The daughter brought her non-responsive mother into the doctor’s office in a wheelchair, and they walked out of the doctor’s office talking with each other. Unfortunately for Alzheimer’s patients and their family members, the injection has to be repeated every week or two to maintain the effect.

But I suggest that the most convincing evidence is from Dale Bredesen, a medical doctor, a Professor at UCLA, and a founding President of the prestigious Buck Institute on Aging, who has (so far) supervised more than 100 cases of Alzheimer’s disease being reversed. This is not a trivial success; many of his patients carry the ApoE4 gene for increased Alzheimer’s disease risk.

Many of Dr. Bredesen’s public and medical talks are online for your edification.

Lipid-Enveloped Viral Diseases

Although this book is written primarily about herpes, there are a host of other lipid-enveloped viruses\textsuperscript{69} that cause morbidity and mortality. These include:

1. coronavirus (several, including SARS and the Wuhan coronavirus),
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 flu),
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),
13. variola virus (smallpox virus),
14. West Nile virus,
15. yellow-fever virus, and
16. zika virus.

Animal 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), and
5. Newcastle disease (birds and horses),
6. pseudorabies (swine, crossing into rabbits, cattle, sheep, goats, cats, dogs and raccoons) [Pirtle, 1986],
7. Semliki Forest virus (also infecting humans),

\textsuperscript{69} A quote from Wolf et al. “We describe an antiviral small molecule, LJ001, effective against numerous enveloped viruses including influenza A, filoviruses, poxviruses, arenaviruses, bunyaviruses, paramyxoviruses, flaviviruses, and HIV-1.” See the full reference in the reference section for more context.
8) swine fever (pigs), and
4) swine flu (also infecting humans).

No serious effort has been made to make this list comprehensive.

The Antiviral Literature on BHT

The scientific literature on BHT prior to 1975 had developed several themes:

1) BHT’s toxicity was of a low order, especially its chronic toxicity. It is enzymatically metabolized only in the liver, with only trace quantities of changed BHT being found in the brain, lungs, heart and kidneys.

2) BHT is a powerful antioxidant. Its ability to protect fats and oils from peroxidation may be several times better than other natural fat-soluble antioxidants like vitamin E.

3) BHT extends lifespan in animal studies. These results have been well documented in many studies over many animal species, and in some animals, typical mean lifespan increases of 50% are seen. The qualitative benefits observed in these studies are consistently striking, the BHT treated animals look younger, have glossier and thicker fur coats, are leaner and more active, and have better immune responses and healing times. The mechanism of action may have something to do with the control of free radicals and reduction of oxidative stress, but it seems more likely to be the direct but unintended result of food restriction induced by the noticeable taste of BHT in the BHT-fortified animal feed.

4) BHT lowers the incidence of cancer in cancer-prone animals and in animals exposed to some carcinogens. The mechanism of BHT’s action appears to be due to two effects. First, BHT is a free-radical scavenger and intervenes in free-radical pathways of carcinogenesis. And second, BHT causes induction of certain hepatic (liver) enzymes that metabolize carcinogens before they initiate cancer.

BHT Antiviral Findings

In 1975, Wallace Snipes, Stanley Person, Alec Keith and James Cupp published the first in a series of papers concerning the antiviral properties of BHT. Their paper was entitled, “Butylated hydroxytoluene inactivates lipid-containing viruses” and was published in Science.

In their experiment, BHT was found to be a potent inhibitor of mammalian and bacterial lipid-containing viruses. Virus preparations were exposed to BHT for 30 minutes and added to host cells for assay of viral activity. The results indicate total inhibition of Phi-6 virus, substantial inhibition of HSV, and almost no inhibition of polio virus, which contains no lipids. A HSV mutant and PM2 virus, both lipid-containing viruses, were also substantially inhibited. Phi-3-1a virus, which infects the same host as Phi-6 but contains no lipids, was not affected. In the following table, the effective concentrations for 50% inactivation of the virus are compared to approximate body concentrations in the US population.

<table>
<thead>
<tr>
<th>BHT Concentration</th>
<th>Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.6 x 10⁻⁵ Molar</td>
<td>Approximate U.S. Body Concentration</td>
</tr>
<tr>
<td>1.0 x 10⁻⁵ Molar</td>
<td>50% Inactivation of Phi-6 virus</td>
</tr>
<tr>
<td>0.7 x 10⁻⁴ Molar</td>
<td>50% Inactivation of Herpes virus</td>
</tr>
</tbody>
</table>

The amount of BHT found in the body fat of the US population was slightly less but of the same order of magnitude as that necessary to inactivate Phi-6 virus. The amount needed to inactivate HSV was one order of magnitude higher. Since the average daily intake of BHT in the US is estimated to be about 2 mg, we can make a crude, order-of-magnitude estimate of the therapeutic dose for herpes treatment at 20-200 mg of BHT. This is not far from what has been found by people using BHT to treat herpes.

Later that year, a second paper from the same laboratory was published in Antimicrobial Agents and Chemotherapy entitled “Inactivation of lipid-containing bacteriophage PM2 by butylated hydroxytoluene” [Cupp et al. 1975].
In the first paper, PM2 virus was reported to be inactivated to the same degree as HSV. In this paper, evidence is presented to indicate complete destruction of PM2 by BHT treatment. Most of the killing effect occurred in the first five minutes.

The active form of BHT was postulated to be a non-soluble suspension of microcrystals. This conclusion was supported by several observations. First, surfactants that would tend to solublize BHT lessened its killing ability. Second, solvents that gave the greatest precipitate gave the greatest antiviral activity. The correlation between absorbance (turbidity of the precipitate) and virucidal activity was approximately linear. Third, the BHT precipitate of microcrystals that formed when the BHT/solvent mixture was added to the medium decreased with time, as did the killing activity. Sixty percent of the virucidal activity was lost in 40 minutes. And fourth, a freshly mixed solution of BHT and medium was filtered through a 0.22 micrometer filter removing 80% of the BHT and leaving only the finest crystals. The resulting solution retained most of its virucidal activity.

Once the microcrystal precipitate was recognized as an important aspect of BHT antiviral activity, its toxicity to the host cell was assayed. At a concentration of BHT that killed more than 99% of PM2, absolutely no effect on the growth of the host cells could be seen.

In 1976, a letter written by Robert Alan Franklyn, “Butylated Hydroxytoluene in Sarcoma-Prone Dogs,” appeared in The Lancet. The main subject of this paper is the anticancer and life extending effects of BHT. It is included here for two reasons; 1) It concerns the use of BHT on large animals, and 2) reference is made to distemper, a viral disease.

The author of the letter was a breeder of Scottish deerhounds which have a genetic weakness and usually die at age 4-6 years of large-bone sarcoma (cancer). Thirty-six animals, ages 1-1/2, 2 and 3 years, were split into a control group and a BHT group which received one tablespoon (30 grams) of BHT daily.

Control animals all died at age 4-6, mainly of sarcoma. Cause of death for BHT treated animals was distemper for the 3 year olds at age 8, and heart and kidney failure for the 1-1/2 and 2 year olds at ages 9 (6 dogs) and 10 (3 dogs).

There were qualitative differences reported between the BHT dogs and controls. The BHT dogs were sleeker, racier, thinner, trimmer, and more “typy” of the breed. There was no difference in appetite, calorie intake or diet (except the BHT). The ineffectiveness of this high dose of BHT at preventing death from distemper is puzzling. Canine distemper is reported to be a lipid-enveloped viral disease. It is also puzzling that none of the younger dogs died of distemper.

Although this paper was quite intriguing, I should caution dog owners against uncritical acceptance of the above report. Although the amount of BHT was reported to be one tablespoon or 30 grams, one tablespoon of BHT actually weighs only ten grams. So something may have gotten lost in the translation of a letter into a letter-to-the-editor. Even ten grams is a huge dose of BHT to give a dog. If the “tablespoon of BHT” was actually a tablespoon of vegetable oil, coconut oil or lard into which BHT had been dissolved, then the actual dose of BHT given to the dogs might have been an order of magnitude lower than reported.

One month later, the third paper from the Snipes-Person-Keith-Cupp research group was published in Antimicrobial Agents and Chemotherapy entitled “Inactivation of the enveloped bacteriophage Phi-6 by butylated hydroxytoluene and butylated hydroxyanisole” [Wanda et al. 1976].

BHT was found to inactivate Phi-6 virus at concentrations as low as 3 x 10^-5 Molar. BHA also inactivated Phi-6, but higher concentrations were required. The viral envelope was not stripped off by BHT treatment, in contrast to treatment with the detergent Triton X-100. The BHT treated, inactivated virus was found to be morphologically indistinguishable from the active virus. It was suggested that a missing binding protein might account for the treated virus’ inability to attach to its host cell.
Later in the following year, the first scientific study on live animals was reported in *Science* entitled “Butylated Hydroxytoluene Protects Chickens Exposed to Newcastle Disease Virus” [Brugh 1977].

Newcastle disease virus (NDV) affects a wide range of avian species including ornamental pet birds. It also infects thoroughbred race horses. Its extreme virulence was largely prevented by small amounts of BHT, 0.01% to 0.1% of diet by weight. The immunization response of chickens to a non-virulent strain of NDV was also blocked by BHT, suggesting that previously reported vaccination failures were primarily due to the use of BHT as an antioxidant in feed.

The ability of NDV LaSota to agglutinate (stick together) blood cells, a normal membrane function, was not blocked by BHT at 44 mcg/ml indicating non-disruption of the NDV virion (virus particle). The dose for NDV inactivation was one to two orders of magnitude less than that used in studies of the life extension or anti-carcinogenicity effects of BHT.

In 1978, a paper was published on BHT’s effects on cytomegalovirus (CMV), a human pathogen responsible for cytomegalic inclusion disease, intrauterine death, prematurity, congenital defects, mental retardation, post-perfusion syndromes, and interstitial pneumonia, plus viral enteritis in organ transplant patients, “Inactivation of cytomegalovirus and Semliki Forest virus by butylated hydroxytoluene,” by K. S. Kim, H. M. Moon, V. Sapienza, R. I. Carp, and R. Pullarkat (*Journal of Infectious Diseases* volume 138, number 1, pages 91-4, July 1978).

Cytomegalovirus (CMV), a member of the herpesvirus group, was inactivated by more than 99% by 40 mcg/ml of BHT. Murine (rodent) CMV, and Semliki Forest virus, were also inactivated more than 90% by the same concentration of BHT. The lipid-containing vaccina virus was inactivated only at higher concentrations of BHT. poliovirus, which contains no lipids, was not inactivated.

One and a half years later, “Studies with a hydrophobic, spin-labeled virucidal agent” was published by Neal DeLuca, Alec Keith, and Wallace Snipes in *Antimicrobial Agents and Chemotherapy* (volume 17, Number 1, pages 63-70, January 1980).

A spin-labeled (stable free-radical) virucidal agent, BPN, was synthesized and found to inactivate both Phi-6 and herpes simplex virus to the same degree as BHT. The hypothesis that BHT inactivates Phi-6 by removing a binding protein is proven to be correct. Of the five membrane proteins of Phi-6, only one is removed by both BHT and BPN. While BHT and BPN have quite different structures, they do have similar globular shapes and charge distributions, and additionally, they are quite hydrophobic. The authors present the hypothesis that it is the particular associations between the proteins and lipids in viruses that allow BHT and BPN to selectively damage the virus, and not the host cell membrane. The absence of “free lipid pools” in the viruses is the source of their weakness to BHT. This could be studied by changes in the energy level of the lone (free radical) electron in BPN, which were caused by differences in solvent (between viruses and host cell membrane). The authors suggest that each virus may require a differently shaped hydrophobic molecule to optimize the antiviral effect, depending upon the exact association of lipid to protein in the viral structure.

In March, Vern D. Winston, Joseph B. Bolen and Richard A. Consigli published a paper “Effect of butylated hydroxytoluene on Newcastle disease virus” in the *American Journal of Veterinary Research* (Volume 41, Number 3, pages 391-4, 1980) which confirmed Brugh’s work with BHT and Newcastle disease virus. NDV was found to be inactivated 92% by 50 mcg/ml of BHT. Virion adsorption to host cells was inhibited 32% and electron microscopy revealed visible disorientation and disruption of the viral envelope.

Although the mouse is not a good animal to model herpes infections, the experimenters used three different techniques in attempting to induce human-like herpes lesions. In the first experiment, mice without immunity to HSV-1 were used. These animals developed deep lesions that were not typical of human HSV lesions. In the second experiment, previously infected and recovered mice were γ-irradiated to suppress immune activity and re-infected with HSV-1. Lesions on these animals remained more localized and more typical of human HSV lesions. In the third experiment, animals were inoculated with γ-globulin 24 hours prior to infection with HSV. In this case, lesions also remained more localized. In all cases, topical BHT had significant effect on reducing clearance time of the infections. The significance of this experiment to human herpes infections is not clear.

The Toxicology of BHT

Despite BHT’s meager clinical experience and lack of approved-drug status, its effectiveness as an antioxidant and antiviral agent will provide an incentive for its clinical use. However, there are many questions about the use of BHT in human medicine that currently remain unanswered. Each one is a potential research project in itself. I hope this presentation and discussion of the available literature on BHT will prove useful in both assessing its value to a comprehensive health care system and in stimulating interest in further research. This second element is especially problematic due to the present generic status of BHT. All chemical patents have long since expired worldwide. In addition, the use patents relating to antiviral applications of BHT have also expired. Without proprietary interests, financial incentives for further research are minimal.

Animal studies are our largest source of information from which to extrapolate clinical risks in man. The inherent uncertainties involved in translating dosages between species makes it imperative to look for toxic effects in man at dosages less than those that that produce toxicity in animal experiments. Additionally, some of BHT’s toxic effects on laboratory animals have been related to vitamin deficiencies.

Since lab-animal nutrition is often better than the human average, caution is warranted in this area. Most of the studies presented here on the effect of BHT on various organ systems use dosages between 50 mg/kg and 500 mg/kg. While the higher dose is conclusively tied to adverse effects in some animals, the lower dose seems to be relatively benign.

For a 70 kg person (150 lbs), 50 mg/kg is a 3500 mg dose, which is 100 times the unconditionally acceptable daily intake of 0.50 mg/kg body weight set by the WHO [Joint FAO/WHO Expert Committee on Food Additives 1967].

Dosage/Effect Ranking

The adjacent table summarizes the published animal research on BHT and the suggested doses for herpes treatment, ranked in order of mg/kg values.

<table>
<thead>
<tr>
<th>BHT Dosage</th>
<th>Observation or Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.02-0.03 mg/kg</td>
<td>Average daily intake by US populace.</td>
</tr>
<tr>
<td>0.5 mg/kg</td>
<td>FAO/WHO maximum unconditionally acceptable daily intake.</td>
</tr>
<tr>
<td>From 1-3/4 kilos of food at 0.02% BHT (FDA maximum) for 70 kg person.</td>
<td></td>
</tr>
<tr>
<td>3.4 mg/kg</td>
<td>250 mg dose for 70 kg (154 lb) person.</td>
</tr>
<tr>
<td>20 mg/kg</td>
<td>1000 mg dose for 50 kg (110 lb) person.</td>
</tr>
<tr>
<td>15-20 mg/kg</td>
<td>(0.017% of diet) Threshold dose for temporary lowered prothrombin index in rats.</td>
</tr>
<tr>
<td>40 mg/kg</td>
<td>2000 mg dose for a 50 kg person.</td>
</tr>
<tr>
<td>100 mg/kg</td>
<td>(50% BHA, 50% BHT) All monkeys give birth to normal infants.</td>
</tr>
<tr>
<td>100 mg/kg</td>
<td>Produces no permanent liver changes in rats.</td>
</tr>
<tr>
<td>200 mg/kg</td>
<td>Threshold dose for lung damage in mice.</td>
</tr>
<tr>
<td>250 mg/kg</td>
<td>Threshold dose for sustained lowered prothrombin index in rats.</td>
</tr>
</tbody>
</table>
400 mg/kg Lung damage and cell proliferation in mice (see also 2500 mg/kg).
400 mg/kg Lowered and raised radiation sensitivity in mice.
200-500 mg/kg (0.25-0.50% of diet) Mean lifespan increases in male mice (greater at higher dose).
500 mg/kg Lowered prothrombin index in rats.
500 mg/kg Changes in electrolytes and organic transport in rat kidney slices.
500 mg/kg Lowered growth rate and delayed development of behavioral traits in rat pups
nursed by BHT-dosed rat mothers.
500 mg/kg Ultrastructural liver abnormalities in monkeys with absence of clinical
abnormalities.
500 mg/kg Increased thyroid weight & iodine uptake in rats.
500 mg/kg Non-teratogenic in rats and mice.
2500 mg/kg Absence of lung damage in mice exposed to cedar shavings or cedar terpenes.
1600-3200 mg/kg Merck Manual LD$_{50}$ in rodents.

For the treatment of herpes, doses of 250-2000 mg are suggested, which is 7-58 times the WHO
unconditionally acceptable dose for a 70 kg person. For a 50 kg person, 250 mg is 10 times the WHO dose.
Teratogenicity

Early reports on the teratogenicity of BHT found anophthalmia (missing eyes) in rodents [Brown 1959]. Later studies found no such effect [Frawley 1965, Johnson 1965] and one paper attributed the anophthalmia to a possible deficiency of Vitamin A or Vitamin E in the diet of the earlier test animals [Clegg 1965]. Subsequent testing has failed to find any birth defects in BHT gestated offspring in mice or rats at 500 mg/kg dose level [Vorhese 1981, Meyer 1980, Stokes 1974, Clegg 1965, Frawley 1965, Johnson 1965] including monkeys at the 50 mg/kg dose level. There is even a lack of evidence to support birth defects in monkeys at 500 mg/kg [Allen 1976].

Carcinogenicity

BHT has been shown to have both carcinogenic and anticarcinogenic activity in animal studies. Several experimental approaches have been investigated, varying from massive single injections of BHT to chronic, daily exposure.

In experiments with BHT alone, the incidence of cancer seems either to be unaffected or much reduced [Clegg 1965, Hirose 1981, Shirai 1982], especially in cancer-prone animals. An early study demonstrating carcinogenic activity of BHT could not be replicated by subsequent studies, the erroneous result being attributed to the presence of aflatoxin contamination of the animal feed. Aflatoxin is an extremely powerful carcinogen produced by fungal (mold) contamination of nuts and grains. It is a significant epidemiological cancer risk to humans, especially in third-world nations and in tropical climates.

In animal experiments with BHT and known carcinogens, BHT acts as both a promoter and an antipromoter of carcinogenesis, depending upon the experimental conditions. Carcinogenesis is usually decreased when the BHT is administered prior to or concurrent with the carcinogen [Ulland 1973, Goodman 1976, Clapp 1979, McCay 1980, King 1981, Williams 1983]. When BHT is administered after carcinogenic exposure, the incidence of cancer is frequently increased [Peraino 1977, Witschi 1981, Imaida 1982, Williams, 1983]. The increases are greatest with hepatically metabolized carcinogens where large single doses of BHT are administered post-exposure. Compared to phenobarbital, another hepatic enzyme inducer, BHT is a “weak enhancer” and then “only at near-toxic doses” [Maeura 1984].

Because BHT is both a promoter and an antipromoter, the type of carcinogens that people are exposed to will determine whether its net effect is to increase or decrease incidence of cancer. Because cruciferous vegetables (cabbage, cauliflower, Brussels sprouts, broccoli) are known to induce liver enzymes similarly to BHT—and also lower general cancer incidence epidemiologically—we can infer that BHT is likely to lower net cancer risks.

In the Soviet Union during the 50s and 60s, BHT (called ionol by the Soviets) was extensively studied as an anti-tumor compound [Emanuel 1963, 1973] culminating in its approval as a treatment for bladder cancer.

Psychological and Behavioral Changes

Many studies have found evidence of psychological and behavioral changes in development at 500 mg/kg doses of BHT [Vorhese 1981, Meyer 1980, Stokes 1974]. In one experiment, the decrease in weight seen in BHT raised rat pups at 500 mg/kg was found to attenuate with time, becoming insignificant by age 3 months. This effect was not seen after weaning or at 125 or 250 mg/kg dose levels.

A delayed development in a multiplicity of behavioral traits, seen only in the 500 mg/kg dosed animals, was also not evident after weaning, suggesting no “special toxicity of BHT for the central nervous system” [Vorhese 1981]. Another study demonstrating behavioral changes found that the changes were most clearly seen in the lactation phase of development. BHT is strongly excreted in breast milk. While a slight (statistically nonsignificant) reduction in growth rate was seen in BHT-gestated pups nursing non-BHT-dosed mothers at the 500 mg/kg dose level, the effect was significant and substantial for both BHT-gestated
and non-BHT-gestated rat pups nursing BHT-dosed mothers [Meyer 1980]. There was no observable effect on birth weight or gestation time.

In a test of 5 female monkeys taking 100 mg/kg of a BHA/BHT mixture (50 mg/kg BHA and 50 mg/kg BHT), no clinical abnormalities were observed during a 1 year initial exposure, nor during a second year of exposure during which they were bred and gave birth to normal infants [Allen 1976]. The subsequent two years following antioxidant exposure were also free of any clinical abnormalities. The females continued to give birth to normal, healthy, infant monkeys.

Chronic prenatal exposure to BHT may perhaps prove to be benign, however, infant exposure through breast milk must be considered as a significant concern. In my opinion, the available data do not yet rule out the possibility of the clinical use of BHT in pregnancy or infancy. More data are needed to assess this aspect of BHT’s safety to my satisfaction. However, the herpes group viruses (HSV-1, HSV-2, cytomegalovirus, etc.) pose serious health risks during pregnancy, childbirth and infancy.

**Hepatic Effects of BHT**

There are still uncertainties about the full effect of BHT on the liver of man. In rats and monkeys, the degree of liver enlargement closely parallels the proliferation of smooth endoplasmic reticulum (SER) and the increase in drug metabolizing enzymes. A concomitant depression of glucose-6-phosphatase activity appears related to the induction of drug metabolizing enzymes and is “consistent with increased metabolism of glucose via the pentose phosphate pathway in response to an increased requirement of NADPH” [Crampton 1977]. This is not felt to be indicative of any cell damage [Crampton 1977, Goldberg 1966]. Rats treated with 0.4% BHT in the diet for 80 weeks showed increased aniline-4-hydroxylase activity, decreased glucose-6-phosphatase activity, and increased liver weight, all of which had returned to normal four weeks after discontinuation of BHT.

A simultaneous increase in urinary ascorbic acid is seen during the BHT-induced process of liver enlargement in the rat. Urinary ascorbic acid is proposed as a test to distinguish hyperfunctional liver enlargement from pathological liver enlargement. The livers of primates are not capable of producing ascorbate, but this phenomenon might prove useful by following the excretion of L-xylulose and/or D-glucaric acid [Gaunt 1965]. These observations would suggest the ascorbate be included with BHT, especially in the dose titration phase of BHT therapy, when hepatic enlargement would be occurring. In additions, ascorbate’s strong antioxidant and free radical-scavenging effects might prove helpful in lowering the toxicity or concentration of BHT-hydroperoxide, which has been reported to be the most toxic of the BHT metabolites [Yamamoto 1980]. Sulfur antioxidants, such as cysteine, N-acetylcysteine and glutathione, should also be mentioned in this regard. Vitamins and coenzymes which support the generation of reducing agents (NADH, NADPH and FADH2) may have significant effects on the redox recycling of ascorbate and glutathione. Nutritional factors with critical roles in the production of reducing power include vitamins B1, B2, B3, tryptophan, lipoate and coenzyme Q.

In rat experiments, the liver adjustment phase was established at less than one week, which was in accord with the induction phase of BHT-oxidase [Gilbert 1967], the enzyme responsible for oxidizing BHT to BHT-alcohol. No permanent liver changes are seen at a dietary level of 0.1% BHT (approximately 100 mg/kg).

In monkeys, 50 mg/kg and 500 mg/kg produce no clinical abnormalities, yet many ultrastructural abnormalities are visible under an electron microscope at the higher dose [Allen 1972]. “The degree of liver enlargement and induction of drug-metabolizing enzyme activity increases linearly with dose until, at very high doses, no further increases occur” [Crampton 1977].

Several differences in liver metabolism between primates (monkeys) and rodents (rats) have been reported. The greater liver enlargement with BHT over BHA in rats is reversed in monkeys. There is an enterohepatic circulation of BHT in the rat that is not present in man.
An early report of t-butyl-oxidized metabolites in man [Daniel 1968] was not confirmed by another researcher [Ryan 1971]. Further research using high-performance liquid chromatography has confirmed that t-butyl oxidation is a major metabolic pathway in man [Wiebe 1978] and a minor one in rats and mice [Matsuo 1984].

The liver’s drug-metabolizing enzyme systems have undergone a naming-convention change during the span of the cited literature. Originally, they were referred to as P450 enzymes, or cytochrome P450 enzymes, each of which now has a new “CYP” designation.

Increased Thyroid Weight

In rats, BHT has been reported to increase thyroid weight and iodine uptake at both 500 and 5000 ppm (0.05-0.5%) BHT. Electron microscopy “revealed microfoliculation and increased height of the follicular epithelial cells” [Sondergaard 1982]. The significance of this finding to the health of the rat, or man, is not clear.

Lung Damage

In single doses of 400 mg/kg, BHT causes lung damage and cell proliferation in all strains of mice [Williamson 1978, Witschi 1978, Omaye 1975, Saheb 1975, Marino 1972]. This effect is not observable at doses below 200 mg/kg and is also completely blocked by cedar terpene administration (from cedar shavings or cedrol injections), even up to doses of 2500 mg/kg BHT or with a two hour delay in terpene administration [Malikson 1979]. Lung damage is specific to BHT, or an early metabolite, as other antioxidants (BHA, α-tocopherol, pyrogallol, propyl gallate), other substituted phenols (2,4-di-tert-butylphenol, 4-phenylphenol, 4,5-butylphenol; and 2,4,6-trimethylphenol), and BHT metabolites (BHT-alcohol, BHT-acid, and BHT-quinone) did not have that effect [Malikson 1979]. The lack of sensitivity to BHT of young mice (who do not have fully active livers) suggests that a metabolite of BHT is the toxic agent in lung damage. The structural features responsible for lung toxicity are a methyl group in the para-(4)-position and hindering alkyl groups in the ortho-(2,6)-positions on a phenolic ring [Mizutani 1982]. This suggests the quinone-methide, or a close metabolite, as the toxic moiety. BHT-hydroperoxide is another possible candidate. However, an increase in the non-enzymatic conversion of BHT to BHT-hydroperoxide that would be expected from exposure to 100% oxygen is not seen [Williamson 1978]. It is possible that unchanged BHT itself is responsible for lung damage in mice. If a radical intermediate is responsible for the toxic effect, it should diminish with the addition of appropriate free-radical scavengers. Although BHT-induced lung damage is observed in all mouse species, it has yet to be seen in any other species.

BHT’s Effect on the Kidney

High doses of BHT also have effects on the kidneys. In rats, 500 mg/kg oral doses of BHT cause decreases in food intake and water consumption, and an increase in urinary volume, followed eventually by an increase in water consumption. Sodium and potassium excretion dropped but not in proportion to the drop in food intake. Urine osmolality was consistently lower [Ford 1979a]. Other renal functions were also affected. The ability of renal cortical slices to accumulate an organic acid (p-aminohippurate) was reduced on days 1, 2, and 4 but was comparable to control levels by day 6. “The attenuation of this effect despite continual administration of the antioxidant may be related to the induction of hepatic metabolism” [Ford 1979b]. Transport of an organic base (n-methylnicotinamide) was unaffected.

BHT and Radiation

Many antioxidant and reducing (anti-oxidizing) chemicals have radio-protective effects including ascorbate, reduced sulfhydryl compounds, selenium and tocopherol. When 400 mg/kg BHT was given by single injection to induce cell proliferation in mice, changes in radiation sensitivity were observed for both x-rays and fission neutrons. At day two after BHT, the LD50 dose had dropped 72% (from 959 rad to 269
rad) for x-rays and 80% (from 476 rad to 98 rad) for fission neutrons. At day six, however, the LD$_{50}$ had risen above control levels, to 150% (1445 rads) for x-rays and 120% (575 rads) for neutrons [Ulrich 1982]. BHT must therefore be considered to be a radiation-modifying compound and appropriate care taken in adjusting the dose and timing of cancer radiation treatments to avoid potential complications and maintain radiation effectiveness.

Similar tests with mice on a 0.75% BHT diet for 28 days found radiation potentiation in C31F1 mice and radiation protection in BALB/c mice [Clapp 1975, Cumming 1973]. Both effects were statistically significant. If BHT is demonstrated to be radio-protective over long-term exposure, it may have application in high-radiation jobs, like extended space missions and nuclear power plant decommissionings.

BHT also modifies the effects of ultraviolet radiation on skin. This has been widely reported in terms of resistance to sunburn and in lowering skin-cancer risks. This effect may be due to actual reduction in the transmission of ultraviolet radiation through the upper layers of skin into the basal cells below [Koone and Black, 1986]. In essence, BHT deposited in the skin acts like a mild sunblocker.

**BHT and Blood Clotting**

BHT also causes a lowering of prothrombin index in rats at doses as low as 0.017% of diet after one week [Takahashi 1978]. This effect also attenuates with time. After four weeks, prothrombin index was lowered only at dietary intakes of 0.25% and 0.50% BHT. At levels of 1.0-1.5%, dietary BHT causes hemorrhagic death in male rats [Takahashi 1981, 1978b, 1976b]. This effect may be due to inhibition of phylloquinone epoxide reductase [Takahashi 1981c] which allows accumulation of a prothrombin precursor in the microsomes of treated rats. The hemorrhagic effect is completely blocked by phylloquinone (vitamin K) or phylloquinone oxide [Suzuki 1979, Takahashi 1979].

There have been no published data concerning this effect in man or monkey, but we have received numerous anecdotal reports of delayed clotting in humans. It is possible that supplemental vitamin K may prove of benefit. Special caution is indicated with concurrent use of other substances with anti-coagulant activity, especially during the titration phase of BHT therapy. These include warfarin, aspirin, ginkgo biloba and cold-water fish oils—which contain EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid).

**Acknowledgements and Final Comments**

This section must begin with an acknowledgement of John A. Mann, who wrote his first booklet on BHT back in 1981 and with whom I co-authored the first edition of *Wipe out Herpes with BHT* in 1983. In many respects, John was my mentor and taught me how to write effectively, a skill that I managed to neglect during four years of high school and five years attending one of the best liberal arts colleges in the world. But then again, there is a huge difference between writing because of duty, a class assignment for example, and writing because you have something to say. As you might guess from reading this book, I definitely had something to say.

The second acknowledgement goes to Durk Pearson and Sandy Shaw, who were the first people that I knew to fully appreciate BHT’s therapeutic potential as an antiviral agent and the first people to popularize this idea in writing (in their 1990 best seller *Life Extension: A Practical Scientific Approach*). Durk and Sandy were information pioneers way back then, and are still doing cutting-edge analysis today.

The third acknowledgement goes to Ward Dean, who had the vision to see BHT’s value as a therapeutic agent from within the medical profession and the courage to put his vision into practice. The earliest insights into adjunctive therapies were his.

The last acknowledgement goes to the faculty and students at Reed College, who had a huge effect on my thinking. Although Reed College is truly a liberal arts college bestowing only Bachelor of Arts degrees, it
has outstanding scientific departments that rival the best technical colleges and universities. My coursework in chemistry and biology at Reed (and the impromptu discussions with fellow students) taught me how to think, how to analyze, how the scientific process is supposed to work, and how to properly question and evaluate scientific research. These are the skills upon which my professional career has been built.

I also came to understand how ideology, belief and financial reward can slant the process of discovery, leading to what might be called “self-fulfilling prophecy.” Scientists who set out to prove a belief often accomplish that end, but not without sacrificing the scientific process which would have validated their work. When we work from within ideology and belief, we tend to find what we expect to find.

Another way to say this is we more easily ignore facts, observations and data which conflict with our expectation. Expectation has a dark side—disappointment. That may sound strange but think about it for a moment. We cannot be disappointed unless we have an expectation. And disappointments are something most of us would rather not have in our lives. This is not rocket science; this is plain, ordinary wisdom. Regarding financial conflicts, there is little need of explanation. Pretty much everybody understands the power of money to corrupt any supposedly impartial process. Whether we are talking about bribes for police or regulators, a salary for employment, or a grant for a scientific investigation, the political or ideological agenda of the employer can become part of the transaction. With science, the degree to which political influence corrupts the grant process is the degree to which the scientific process is undermined before the research actually takes place.

Following my graduation from college in 1975, I have found many instances—probably far more than most people suspect exist—of scientific research that has been conducted with faulty premises, with flawed methodology, with incomplete analysis and/or with overt bias. Despite the minimal scientific value of such flawed research, some of it is touted by experts and media alike as 1) scientifically valid and 2) a sound basis for public health policy. I am not afraid to disagree with such findings, people and institutions, and have regularly done so in my writings. The full extent of such criticisms will be largely outside the scope of this book, which must necessarily remain focused on viral diseases. But interested readers are invited to read for themselves various newsletter articles on other subjects on the web (http://www.ceri.com) and blogs at Project Wellbeing (http://www.projectwellbeing.com). Or listen to the many interviews and podcasts out there on the Internet.

Some of the topics presented here are also presented as elements of “Reversing Alzheimer’s Disease,” a nine-part series presented on YouTube. For example, the effect of aromatase and IDO on hormones and serotonin levels are laid out in detail. You can find the video series on the swfowkes channel on YouTube (see title page for links).

Regarding BHT and herpes, the issue is not about scientific fraud. The issue is why isn’t there ongoing research? How can BHT be so effective and not be on the TV nightly news? A significant number of readers might ask themselves, “How can I have suffered for ten years with herpes and become lesion-free in ten days, at a total cost of $5?” Many people with herpes problems have been to doctors and been told, “Live with it” or “Take acyclovir.” Some people with herpes, shingles, hepatitis or cytomegalovirus may have been to a dozen doctors, with the same lack of results.

I hope that my criticisms of medical and regulatory institutions are not taken personally by medical doctors and regulators. There is a huge difference between institutions and individuals. Pride in being a scientist does not require endorsement of the NIH. Pride in being a doctor does not require appreciation of the AMA. And just as one’s spiritual development does not require a Church, one’s continuing medical education does not require CME credits.

70 There is a new bias emerging, the advancing of unsupported conclusions in the summary section of journal articles. It is disheartening to see this overt lack of professionalism among scientists, reviewers and editors.
There are many paths. Find and make your own.

**References**

The following references have been published over the last 50 years and present a broad spectrum of scientific inquiry into the properties of BHT. Many papers have been included that do not deal directly with BHT, however they do discuss aspects of aging, free radical activity, liver enzyme induction, viruses, infectious and inflammatory mechanisms, and vitamin therapeutics.

Due to the sequential writing of the above material and the merging of new files with old files, some dating back 30 years, many if not most of the following references are not cited directly in the above text. Although citations are being slowly added as editing advances, any assistance by readers is welcome. If you track down a reference and want other readers to have the benefits of your effort, please send me the citation and location in the text. Thank you.

Nouran Abd-ElMoemen, et al. Ebola Outbreak in West Africa; Is Selenium Involved? *Int J Pept Res Ther* 22: 135-41, 2016. Published online 15 Sept 2015. **Abstract:** One of the current international public health emergencies is the outbreak of Ebola virus disease (EVD), requiring extraordinary response. The current outbreak in West Africa is the most dangerous since Ebola was first discovered on 26 August 1976. Till January 6th 2015, it resulted in 13,387 laboratory confirmed human cases and 8274 deaths. Ebola virus has 5 strains, 4 are pathogenic in humans while the 5th strain Ebola reston strain is not. The current outbreak is caused by Ebola most pathogenic strain, Ebola Zaire strain whose genome differs from that of Reston Ebola virus strain, by the existence of several open reading frames containing large numbers of UGA codons. These codons act as stop codons and in addition they may encode for selenocysteine, the 21st amino acid, which is essential for the formation of selenoproteins. Selenoproteins are integral to the metabolism and have been linked to the progression of certain viral diseases. In this review, we discuss the relation between selenium and the progression of the current EVD in Africa supported by geographical distribution of Se and genetic evidence.


E Cameron and G M Baird. Ascorbic acid and dependence on opiates in patients with advanced disseminated cancer. IRCS Letters to the Editor, August 1983.


Hal Drakesmith and Andrew Prentice. Viral infection and iron metabolism. Nature Reviews Microbiology 6: 541-52, July 2008. This is a review article discussing viral manipulation of iron mechanisms and viral virulence relating to iron levels. “Some viruses selectively infect iron-acquiring cells…” and “Other viruses alter the expression of proteins involved in iron homeostasis…” and “In HIV-1 and hepatitis C virus infections, iron overload is associated with poor prognosis and could be partly caused by the viruses themselves.” And concluding with, “Understanding how iron metabolism and viral infection interact might suggest new methods to control disease.”


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K M Abdel Hamied and A F Abdel Mola. The protective role of the food additive butylated hydroxytoluene in the pancreas of aflatoxicosed adult albino rats: a histological, immunohistochemical, and morphometric study. The Egyptian Journal of Histology 39(3): 217-27, September 2016. Conclusion: "Treatment with AFB1 for 5 weeks has a direct toxic effect on the exocrine and endocrine pancreas that can ultimately affect its performance. The use of BHT markedly attenuates these effects and can be considered an effective chemoprotective agent against AFB1 toxicity."


P J Hughes, H McCellan, DA Lowes, SZ Kahn, JG Bilm, SC Tovey, RE Godfrey, RH Michell, CJ Kirk and F Michelangeli. Estrogenic alkylphenols induce cell death by inhibiting testis endoplasmic reticulum Ca(2+)-pumps. *Biochem Biophys Res Commun* 2000 Nov 2;277(3):568-74. Abstract: Industrial alkylphenols in the environment may act as "xenoestrogens" to disrupt testicular development and decrease male fertility. Among possible targets for these compounds are testicular Sertoli cells, which nurture the developing sperm cells. We demonstrate that SERCA 2 and 3 Ca(2+)-pumps are relatively abundant in rat testis microsomal membranes, and also in Sertoli, myoid, and TM4 cells (a Sertoli cell line). A number of estrogenic alkylphenols such as nonylphenol, octylphenol, bisphenol A, and butylated hydroxytoluene all inhibit testicular Ca(2+) ATPase in the low micromolar concentration range. These agents also mobilize intracellular Ca(2+) in intact TM4 cells in a manner consistent with the inhibition of ER Ca(2+) pumps. Alkylphenols dramatically decrease the viability of TM4 cells, an effect that is reversed by either a caspase inhibitor or by BAPTA, and is therefore consistent with Ca(2+)-dependent cell death via apoptosis. We postulate that alkylphenols disrupt testicular development by inhibiting ER Ca(2+) pumps, thus disturbing testicular Ca(2+) homeostasis.

GD 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.”

GD 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.”


R S Lanigan and T A Yamark. Final Report on the Safety assessment of BHT. *International Journal of Toxicology* Supplement 2: 19-94, 2002. Abstract: BHT is the recognized name in the cosmetics industry for butylated hydroxytoluene. BHT is used in a wide range of cosmetic formulations as an antioxidant at concentrations from 0.0002% to 0.5%. BHT does penetrate the skin, but the relatively low amount absorbed remains primarily in the skin. Oral studies demonstrate that BHT is metabolized. The major metabolites appear as the carboxylic acid of BHT and its glucuronide in urine. At acute doses of 0.5 to 1.0 g/kg, some renal and hepatic damage was seen in male rats. Short-term repeated exposure to comparable doses produced hepatic toxic effects in male and female rats. Subchronic feeding and intraperitoneal studies in rats with BHT at lower doses produced increased liver weight, and decreased activity of several hepatic enzymes. In addition to liver and kidney effects, BHT applied to the skin was associated with toxic effects in lung tissue. BHT was not a reproductive or developmental toxin in animals. BHT has been found to enhance and to inhibit the humoral immune response in animals. BHT itself was not generally considered genotoxic, although it did modify the genotoxicity of other agents. BHT has been associated with hepatocellular and pulmonary adenomas in animals, but was not considered carcinogenic and actually was associated with a decreased incidence of neoplasms. BHT has been shown to have tumor promotion effects, to be anticarcinogenic, and to have no effect on other carcinogenic agents, depending on the target organ, exposure parameters, the carcinogen, and the animal tested. Various mechanism studies suggested that BHT toxicity is related to an electrophilic metabolite. In a predictive clinical test, 100% BHT was a mild irritant and a moderate sensitizer. In provocative skin tests, BHT (in the 1% to 2% concentration range) produced positive reactions in a small number of patients. Clinical testing did not find any depigmentation associated with dermal exposure to BHT, although a few case reports of depigmentation were found. The Cosmetic Ingredient Review Expert Panel recognized that oral exposure to BHT was associated with toxic effects in some studies and was negative in others. BHT applied to the skin, however, appears to remain in the skin or pass through only slowly and does not produce systemic exposures to BHT or its metabolites seen with oral exposures. Although there were only limited studies that evaluated the effect of BHT on the skin, the available studies, along with the case literature, demonstrate no significant irritation, sensitization, or photosensitization. Recognizing the low concentration at which this ingredient is currently used in cosmetic formulations, it was concluded that BHT is safe as used in cosmetic formulations.


Paul B McCoy, M Margaret King and Jan V Pitha. Evidence that the effectiveness of antioxidants as inhibitors of 7,12-dimethylbenz(a)anthracene-induced mammary tumors is a function of dietary fat composition. Cancer Res 41(9): 3745-8, 1981.


The mean plasma selenium levels achieved with supplementation were 492.2 ng/ml (SD 1457-60, 11 September 1998. “...we show that the alterations of the antioxidant status in [non-obese diabetic] mice is efficaciously counteracted by BHT.”


C S Ramanathan and E W Taylor. Computerized genomic analysis of hemorrhagic fever viruses. Viral selenoproteins as a potential factor in pathogenesis. *Biol Trace Elem Res* 56(1): 93-106, January 1997. Abstract: A number of distinct viruses are known as hemorrhagic fever viruses based on a shared ability to induce hemorrhage by poorly understood mechanisms, typically involving the formation of blood clots (“disseminated intravascular coagulation”). It is well documented that selenium plays a significant role in the regulation of blood clotting via its effects on the thromboxane/prostacyclin ratio, and effects on the complement system. Selenium has an anticlotting effect, whereas selenium deficiency has a proclotting or thrombotic effect. It is also well documented that extreme dietary selenium deficiency, which is almost never seen in humans, has been associated with hemorrhagic effects in animals. Thus, the possibility that viral selenoprotein synthesis might contribute to hemorrhagic symptoms merits further consideration. Computational genomic analysis of certain hemorrhagic fever viruses reveals the presence of potential protein coding regions (PPCRs) containing large numbers of in-frame UGA codons, particularly in the -1 reading frame. In some cases, these clusterings of UGA codons are very unlikely to have arisen by chance, suggesting that these UGAs may have some function other than being a stop codon, such as encoding selenocysteine. For this to be possible, a downstream selenocysteine insertion element (SECIS) is required. Ebola Zaire, the most notorious hemorrhagic fever virus, has a PCR with 17 UGA codons, and several potential SECIS elements can be identified in the viral genome. One potential viral selenoprotein may contain up to 16 selenium atoms per molecule. Biosynthesis of this protein could impo...


A report of high-dose selenium supplementation: response and toxicities. M E Reid et al. A report of high-dose selenium supplementation: response and toxicities. *J Trace Elem Med Biol* 18(1): 69-74, 2004. Abstract: “Concerning the toxicity of selenium has limited the doses used in chemoprevention. Based on previous studies, intakes of 400 microg/day and plasma selenium of 1000 ng/ml (Dietary Reference Intakes, Academy Press, New York, 2000, p. 384) were established as the no observed adverse effect level (NOAEL). This investigation summarizes the plasma response and toxicity reports from 33 men with biopsy-proven prostate cancer who were randomized to either 1600 or 3200 microg/day of selenized yeast as part of a controlled clinical trial testing selenium as a chemopreventive agent for prostate cancer progression. Subjects were on these doses for averages of about 12 months. Plasma selenium levels were monitored throughout the course of follow-up. Symptoms of selenium toxicity were assessed by patient interview with specific questions regarding breath, hair and nail changes. Several liver and kidney function tests and hematologic changes were measured at 6-month intervals. 8 subjects were randomized to the 1600 microg/day and 16 to the 3200 microg/day group. The mean plasma selenium levels achieved with supplementation were 492.2 ng/ml (SD = 188.3) and 639.7 ng/ml (SD = 490.7) for the 1600 and 3200 microg/day doses, respectively. The 3200 microg/day group reported more selenium-related side effects. Blood chemistry and hematologic results were all within normal limits for both treatment groups. More subjects on 3200 microg/day reported symptoms of selenium toxicity; however, these reports did not correspond to peaks in plasma selenium levels. We observed no obvious selenium-related...
serious toxicities. As selenium is used in more chemoprevention and therapeutic settings, additional information on selenium species, sequestration of selenium in specific organs, excretion, and toxicities is needed.”


K Sikorska et al. The role of iron overload and HFE gene mutations in the era of pegylated interferon and ribavirin treatment of chronic hepatitis C. Med Sci Monit 16(3): CR137-43, Feb 2010. “Iron overload was frequently detected in patients with [chronic hepatitis C], and was associated only with C282Y alleles. Biochemical markers of iron overload and HFE gene mutations were negative prognostic factors of antiviral treatment.” The HFE gene is hereditary hemochromatosis.


Dorrit Sondergaard and Preben Olsen. The effect of butylated hydroxytoluene (BHT) on the rat thyroid. Toxicology Letters 10(2-3): 239-44, February 1982. “Iodine uptake was significantly increased in those animals given BHT in the diet, but the half-life of thyroxine was unchanged or slightly prolonged. Thyroid weight was enhanced at both 500 and 5000 ppm BHT, while liver weight was increased only in the latter.”


Buxiang Sun and Morio Fukuhara. Effects of co-administration of butylated hydroxytoluene, butylated hydroxyanisole and flavonoids on the activation of mutagens and drug-metabolizing enzymes in mice. Toxicology 122(1–2): 61-72, 26 September 1997. Abstract: Effects of co-administration of food additives and naturally occurring food components were studied on the activation of mutagens. Male mice (ddY) were given diets containing butylated hydroxytoluene (BHT) or butylated hydroxyanisole (BHA) and flavone or flavanone (2,3-dihydroflavone) for two weeks and the ability of hepatic microsomes to activate aflatoxin B1, benz[a]pyrene and N-nitrosodimethylamine was determined by the mutagenicity test. Co-administration of an antioxidant (0.1% BHT or 0.2% BHA in diet) and a flavonoid (0.1% flavone or 0.1% flavanone) resulted in additive effects on the activation of aflatoxin B1 and benz[a]pyrene, while the activation of N-nitrosodimethylamine was not elevated significantly by the co-administration. To understand the mechanism for the additive effects, induction of specific isoforms of cytochrome P450 involved in the activation of the mutagens was studied. Co-administration of BHT (0.1%) and flavone (0.1%) increased markedly the levels of proteins and the activities of the enzymes related to the isoforms of CYP2A2 and CYP2B2, while co-administration of BHA (0.2%) and flavanone (0.1%) elevated those related to CYP2A1. Further, the activation of aflatoxin B1 and benz[a]pyrene in hepatic microsomes was inhibited by the antibodies against these isoforms, which suggested that the enhanced activation of the mutagens by the co-administration might be mediated by the induction of these isoforms.


O Takahashi and K Hiraga. Inhibition of phyloquinone epoxide-dependent carboxylation of microsomal proteins from rat liver by 2,6-di-tert-buty1-4-methylene-2,5-cyclohexadieneone. *Food Cosmet Toxicol* 19: 701-6, 1981c.


E D Weinberg. Iron toxicity: New conditions continue to emerge. Oxid Med Cell Longev 2(2): 107-9, Apr 2009. “…four additional disorders have been recognized to be enhanced by iron: aging muscle atrophy, viral replication, rosacea and pulmonary alveolar proteinosis.”


Mike C Wolf, et al. A broad-spectrum antiviral targeting entry of enveloped viruses. *Proceedings of the National Academy of Sciences* 107(7): 3157-62, 28 January 2010. Abstract: “We describe an antiviral small molecule, LJ001, effective against numerous enveloped viruses including influenza A, filoviruses, poxviruses, arenaviruses, bunyaviruses, paramyxoviruses, flaviviruses, and HIV-1. In sharp contrast, the compound had no effect on the infection of nonenveloped viruses. In vitro and in vivo assays showed no overt toxicity. LJ001 specifically intercalated into viral membranes, irreversibly inactivated virions while leaving functionally intact envelope proteins, and inhibited viral entry at a step after virus binding but before virus–cell fusion. LJ001 pretreatment also prevented virus-induced mortality from Ebola and Rift Valley fever viruses. Structure–activity relationship analyses of LJ001, a rhodamine derivative, implicated both the polar and nonpolar ends of LJ001 in its antiviral activity. LJ001 specifically inhibited virus–cell but not cell–cell fusion, and further studies with lipid biosynthesis inhibitors indicated that LJ001 exploits the therapeutic window that exists between static viral membranes and biogenic cellular membranes with reparative capacity. In sum, our data reveal a class of broad-spectrum antivirals effective against enveloped viruses that target the viral lipid membrane and compromises its ability to mediate virus–cell fusion.”

A S Wright, D A Akintonwa, R S Crowne and D E Hathway. The metabolism of 2,6-di-tert-butyl-4-hydroxymethylphenol (ionox 100) in the dog and rat. *Biochem J* 97: 303, 1965.


Ibrahim Yıbrık, Yeter Deger, Handan Mert, Nihat Mert and Veyser Ataseven. Serum Concentration of Copper, Zinc, Iron, and Cobalt and the Copper/Zinc Ratio in Horses with Equine Herpessirus-1. *Biological Trace Element Research* 118(1): 38-42, July 2007. “In conclusion, copper and zinc concentrations of the infected group were lower than the control group (p<0.001), whereas iron concentration and the copper/zinc ratio of the infected group were higher than the control group (p<0.05 and p<0.001).” Therefore, proportional loss of copper was the elemental signature of EHV-1. Whether this was caused by the infection or an underlying risk factor for the infection is not known.


