

RAPID VIRUS RECOVERY

No need to live in fear!

Thomas E. Levy, MD, JD

Forewords by Richard Cheng, MD, PhD
and Ron Hunninghake, MD

Over 600 Citations from Peer-Reviewed Journals

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Thomas E. Levy, MD, JD



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Dedicated to definitively ending
COVID and all future pandemics, and
to all who have needlessly suffered
and died

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To my good friends and colleagues, Keith Skinner, Ron Hunninghake, and Richard Cheng, who continue to help me discover and analyze new information that needs to be shared with the world

Foreword

Dr. Levy keeps on surprising me. Until I met Tom several years ago, I felt lost in medicine. Not only did he help restore my confidence, and he also rekindled my interest in medicine. He showed me correctly that the primary motive for medicine should be (and actually could be) about healing rather than making a profit.

I still remember many years ago when I read his book *Death by Calcium*, an important book that proves calcium supplements don't improve osteoporosis, but rather increase our risks for chronic conditions like coronary heart disease. The scientific research data and clear, irrefutable conclusions he presents are so convincing and interesting that I was up reading the book past midnight. Seldom do I stay up to read a book and since medical school I have **never** stayed up that late to read a medical book.

I have learned more about medicine from Dr. Levy than anyone else in the medical field. He's a true genius and the best mentor I have ever had. His ability to comb through the vast amount of scientific literature, connect the many dots, and arrive at clinically useful

information that is logical, irrefutable, and truly based on science is more than impressive.

There seems to be a serious disconnection between basic medical research and the practice of clinical medicine. We can find ample research data on various topics. We can also analyze these data and arrive at plausible clinical suggestions. However, research results are hardly ever reflected in medical practice. Dr. Levy is one of the few who are connecting the latest research with practical clinical application.

One of the many problems in Western medicine is its singular focus on the observable expressions of disease. As a result, clinicians treat superficial symptoms while scientists study specific molecular mechanisms which may become patentable targets for drug development. None of them look at the root causes of diseases. Fortunately, there are alternative medicine practitioners who usually take an integrative, holistic approach to health and disease management. But few of them really understand health and disease at the basic biochemical and cellular physiology levels. Dr. Levy has that understanding and is able to bring that knowledge to practitioners and patients alike.

I am learning from him that our health at the cell level is determined by the molecules that are normally found in the microenvironment in our body in the right amount and right relationship to each other. This concept is what we refer to as orthomolecular medicine. When this microenvironment is disturbed, either by imbalance in quantity or relationship (e.g., too much calcium, not enough magnesium) of normally present molecules, or the introduction of foreign toxins, disease may ensue. Study of this micro-

environmental biochemical balance and how our body responds to insults will greatly enhance our ability to optimize health and heal illnesses. This is a critically important concept, a concept that is missing or largely ignored in western medicine today.

Take viral infections for example. Mainstream medicine focuses nearly entirely on finding specific antiviral drugs and vaccines for prevention and treatment. We all know that for most of the viral infections, there are no specific antiviral drugs, despite great efforts devoted to viral research. The global chaotic, confusing and ineffective management of the Covid-19 pandemic fully reflects the shortcomings of this approach. When Covid-19 broke out, there weren't any SARS-Cov2 specific drugs or vaccines available. Nearly one year later with millions of Covid-19 deaths and billions of dollars of financial losses, vaccines started to show up at a historically record speed for vaccine R&D. But the knowledge of hydrogen peroxide (HP), vitamins C, D, magnesium, other antioxidants and nutrients has been available. If these safe, inexpensive and effective treatments were applied early, as we have been appealing worldwide from Day 1, at the onset of this pandemic, Covid-19 could have been way behind us. Millions of lives and billions of dollars could have been saved. Mainstream medicine nearly totally ignores the God-designed defense mechanism of our human body. We have ample mechanisms to protect us from pathogen invasion. These are universal mechanisms against all invading pathogens. HP is at the center of this defense system. It has been well researched — there are over 80,000 research papers in the literature — and it has been used in the treatment

of various infections. Even so, HP has never achieved wide recognition. This is not unusual, given the fact that if a treatment is not expensive, there is no interest in promoting it. The same fate we have seen over and over happened to vitamins C, D and other inexpensive antioxidants and nutrients. When profit is put ahead of health, people suffer.

Rapid Virus Recovery is a genius work and can frankly save the humanity. Most of us don't realize the threat from viral epidemics/pandemics. Worldwide epidemics and pandemics are rising at an alarming rate. Wikipedia has a list of epidemics and pandemics. In the 19th and 20th centuries, the site listed a total of 98 epidemics/pandemics, but in the short 20 years of 21st century, there has been already 63 epidemics/pandemics! That's about 600% increase! No doubt, epidemics and pandemics are on the rise! Covid-19 is not over yet, are we ready for future pandemics?

I have had many discussions with Dr. Levy and read many of his books. I have even translated and published several of them into Chinese, because the messages in those books are so important to our health that I felt a responsibility to share with my Chinese readers. It's not just the information in his books that attract me, it's a new way of thinking, a truly discovery way of looking into the intricate fabric of our cells and how the natural molecules in our body work together for optimal health. With a Ph.D. degree in biochemistry, I found it quite easy for me to understand the concepts he was describing in his various books. Dr. Levy is a board-certified cardiologist, a clinician. But I was quite surprised, to be honest, by his deep knowl-

edge in biochemistry, something that most other clinicians forgot as soon as they go into clinical practice.

I love Dr. Levy's writing style. There are many health books on the market. Dr. Levy's books stand out from the others on one basic and critical factor: his books are giant scientific reviews. They are fully science based and the conclusions that he arrives in these books are hard to refute. His writing style, which reflects both his medical and legal training, is factual, logical, and compelling. He presents scientific research and conclusions like a masterful lawyer defends his case, with facts and conclusions clearly laid out. This latest book is no exception. Truly, there is an easy, inexpensive way for us to return to normal living. Employ the truths and protocols in this book. The path to freedom is in your hands. Take it, share it, and return to a life without fear.

*Richard Cheng, M.D., Ph.D., ABAARM
Fellow and Diplomate of the American Academy of
Anti-Aging and Regenerative Medicine (A4M)*

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Foreword

There are plagues, and there are victims, and it's the duty of good men not to join forces with the plagues.

– Albert Camus, quote from *The Plague*

I first met Dr. Thomas Levy in early October 2009... in a pyramid.

Dr. Levy had flown into Wichita, Kansas to serve as our keynote speaker at the 1st Annual Riordan IVC and Cancer Symposium. My mentor, Dr. Hugh Riordan, the clinic's founder, had died four years earlier. Dr. Riordan is now well-known for his ground-breaking 90's research in the use of high dose intravenous vitamin C ("IVC") in the adjunctive care of cancer patients.

Dr. Levy was vitamin C famous too. His *Curing the Incurable: Vitamin C, Infectious Diseases, and Toxins* was first published in 2002. Now, in its 4th edition, it has been translated into 7 languages. The title of Dr. Levy's symposium presentation was *The Optimization of Vitamin C and Antioxidant Therapy*. He made his presentation in the iconic two-story pyramid that sits alongside the nine geodesic domes that symmetrically highlight the 93-acre Riordan Clinic campus.

I introduced Dr. Levy to about 120 attendees as an M.D. who was "medically and intellectually well-

grounded” with certification as both a cardiologist and an attorney. Having read several of his now 12 books, I commented on their crispness and clarity of thought. One other major characteristic stood out: this doctor/attorney was not afraid to challenge a pervasive medical bias against proven nutrient-based therapies.

Dr. Levy had flown up from Colombia, South America to make his presentation that evening. I mentioned that he had “come a long way” to be with us. He had also *come a long way* in his scientific thinking in order to break free of the rigid medical school paradigm that all too often squelches original thought and insight.

That evening in the pyramid was the beginning of a great friendship that evolved into a speaking partnership. Over the next decade we were invited to travel to more than a dozen countries on four continents to educate open-minded clinicians on the numerous benefits of IVC. Coincidentally, the more we talked about high dose ascorbate, the more we questioned our basic understandings of just how IVC worked its medical magic. This evolving dialogue on the foundations of what we now know as Redox Medicine led Dr. Levy to pen several new books (including this one) that have dramatically *pushed the envelope of our scientific understanding* of both acute and chronic illness.

His profound insights into the real ROOT CAUSES OF ILLNESS have resulted in the development of an array of simpler, more effective, and much less expensive treatments and lifestyle practices that cut at the etiologic heart of ALL respiratory viral illnesses, most infectious diseases, and the vast majority of complex chronic illnesses.

Dr. Levy's understanding of redox biology led him to a deep study of the Fenton reaction. This amazing chemical transaction explains much of the human immune system's ability to kill pathogens and even cancer cells. In the presence of adequate doses, vitamin C transfers electrons to oxidized iron molecules. This reactivates the iron, so that it is capable of interacting with hydrogen peroxide (HP) to create a powerful, pathogen-killing molecule called the hydroxyl radical. This is the Fenton reaction. Your immune cells would be impotent without it.

With this understanding of the power of properly dosed HP, Levy has revitalized the procedure of nasal nebulization with HP. Yes, properly administered nasal HP nebulization gives ALL OF US the power to kill nasal pathogens — including the “novel” coronaviruses — without damaging the sensitive inner lining of the nose, sinuses, or upper airway. The byproducts of this procedure: simply life-giving oxygen and water to further enhance the immune-protecting mechanisms in our bodily tissues.

The bottom-line application of this scientific reality is that nebulizing HP as outlined by this book could end the current pandemic in a couple of weeks if applied on a global scale. In addition, it would provide some great relief, if not outright cures, for many troublesome chronic infections by attacking the chronic pathogen colonization (CPC) that seeds and feeds them.

Do Not Join Forces with the Plagues

I write this foreword in the early months of 2021. The world lies shaken under the staggering burden of Covid-19. Our generation has painfully partaken of

untold human suffering wrought by this pandemic. As during the Black Plague of the middle ages, or the Great Influenza Epidemic of 1918, we have lost friends, family, jobs, homes, and businesses. Yet, with all this devastation and loss, the more serious threat is not to human life, but to our way of life...our human freedoms! The bigger problem is not a contagion that can take life, but an epidemic of fear that can destroy our freedom to live life on our own terms, without medical or corporate interference or possible malfeasance.

As Albert Camus wrote in his famous 1947 novel, *The Plague*, it truly IS our duty as good men (and women) to NOT “join forces” with this current plague. We need to be free to think and act on behalf of ourselves, to make use of safe, simple, and scientifically effective means of strengthening our immune systems and warding off illness.

Rapid Virus Recovery is a call to arms. Each one of us is responsible for our own health and well-being. We should not have to submit our power of choice to corporate medicine who threatens to dictate our medical choices through the coercive use of experimental, expensive, and potentially damaging methods.

You hold in your hands a comprehensive guide-book that outlines a proven path to better health, superior immunity, and to the potential elimination of all life-threatening viral illnesses from your future. I encourage you to read and to try Dr. Levy's simple, yet ground-breaking strategies.

*Ron Hunninghake, MD
Chief Medical Officer
Riordan Clinic
Wichita, Kansas*

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Introduction

There is NO need to be afraid

Acute viral infections have always presented a major challenge to the practitioners of traditional medicine. To this day, there exist no prescription agents that mainstream healthcare providers can utilize that have the ability to quickly and definitively **cure** a viral syndrome. The most common and clinically severe of such viral infections are acquired via the respiratory route. When such viruses happen to be exceptionally contagious, the stage is set for the escalation of such infections to the level of an epidemic, or at its worst, a pandemic.

These common respiratory viral syndromes primarily fall into the broad array of viruses that cause the common cold and influenza (“flu”). Depending on the strength of the immune system of the person infected, such an infection can be brief and self-limited, or it can be chronic and resistant to resolution. Under some circumstances acute respiratory viral infections can quickly become life-threatening. The potentially fatal viral infections invariably evolve into influenza and influenza-like syndromes that disseminate

nate throughout the body, causing generalized illness rather than the focal symptomatology seen with the typical nose and throat symptoms of a cold.

Nearly all acute viral syndromes are very responsive to a number of different treatment approaches, as will be discussed in this book. Not only do most of these treatments work extremely well to lessen the severity and duration of the viral illness once it has taken hold, they also greatly enhance the body's immunity against contracting a virus at all. Some of the protocols that will be discussed can also quickly and unequivocally cure these infections in most patients.

The training of medical doctors makes them very reluctant to ever use the word "cure" in referring to the effectiveness of any treatment for any condition, no matter how effective such a treatment might actually be. Indeed, just a cursory review of the medical literature on PubMed quickly reveals a common theme in the clinical studies.

No matter how profoundly clear-cut the studied treatments resolve various infections or medical conditions, the authors of such studies will **NEVER** assert that treatment X was able to cure condition, or infection, Y in the Results/Conclusion part of the study. In fact, some of the most impressive studies will only assert that treatment X "appears" to be of benefit for condition Y, and that more studies are needed. It's as if there are certain unwritten commandments for all medical research scientists that require them to avoid any definitive statements — even when the results are glaringly clear. The mantra of all research, especially clinical studies: Evaluate, measure, test, analyze, but never, never, never definitively conclude.

Researchers are similarly unwilling to report a clearly defined cause-and-effect relationship between two things, such as a treatment and a disease, or a medical condition and another disease. For example,

The training of medical doctors makes them very reluctant to ever use the word “cure” in referring to the effectiveness of any treatment for any condition, no matter how effective such a treatment might actually be.

although numerous studies demonstrate a definite causal relationship between periodontitis and heart disease, the conclusions always suggest there is a “link” between periodontitis and heart disease but never state that periodontitis is actually a cause of heart disease.

Certainly, much of the time it is completely appropriate to only conclude that the presence of one thing appears to have a consistent association or relationship to another thing, especially when few other studies exist that have looked at that association. However, it is also apparent that no matter how clear it is that one thing is causing another, that conclusion will never be asserted.

The universal reluctance shown by researchers to assert that one health condition has a cause-and-effect relationship with another is astounding. Many examples of this “Non-Conclusion Convention” are presented herein where researchers refuse to report the cause-and-effect relationship between chronic

pathogen colonization (CPC) and/or focal infections and any of a host of chronic diseases. For example, the literature often notes that the resolution of advanced chronic periodontitis is followed by one or more medical conditions dramatically lessening or disappearing completely. Even when a recurrence of periodontitis is accompanied by a return of those same conditions researchers still refuse to conclude that the periodontitis might actually be a cause for those conditions. Of course, the ultimate studies to “prove” such cause-and-effect relationships would be completely unethical, since they would involve giving healthy participants one disease to see whether another disease appeared.

Simply put, the “research game” is fixed to never conclude that anything reliably causes something, or that anything can ever be cured.

Properly-dosed and properly-administered vitamin C has been curing nearly all acute infectious diseases, especially viral ones, for over 80 years now. Many different articles have documented its benefits in this regard. Yet no current medical textbook even mentions vitamin C as a potential or possible treatment for infectious diseases. While not a topic to be dealt with in any detail in this book, it must still be pointed out to the reader confused as to why vitamin C, or any other viral cure, is not widely used is revealed in the following sobering facts:

Healthcare is primarily a business.

**Any commonly used agent/therapy
must generate substantial and even
unconscionable revenue for the**

**pharmaceutical companies, the hospitals,
and the physicians, or it will never be
used or even properly investigated.
More likely, it will be suppressed.**

**There is more politics in medicine
than there is in politics.**

Proof of these assertions abounds. For example, there was a total refusal by doctors and hospitals all over the United States to provide early treatment for the millions of Americans who tested positive for COVID but were told to “go home and only come back if having difficulty breathing.” In other words, come back when the patient is so sick that they can admit him to the ICU and put him on a ventilator. This “standard of care protocol” generated an automatic \$39,000 check from the government as well as huge revenue for an intensive care hospital stay, and grossly

The inhalation of HP by nebulization has been shown to be extremely effective for the rapid elimination of any pathogen presence in the sinuses, nose, throat, and deep into the lungs.

inflated the fatality rate at the same time.

Many trusting patients simply cannot bring themselves to understand that **their** doctor or **their** hospital is much more interested in profits than their

health interests and overall welfare. Certainly, there are exceptions to the blanket nature of these assertions. But those exceptions are decidedly rare. Until the reader fully grasps the heinous truth of these assertions, it will remain impossible to understand why an easy and cheap answer to a grave infection or illness will always be disparaged, suppressed, and perhaps even **made illegal**.

Even many seemingly well-meaning physicians will almost reflexly reject and denigrate a therapy like HP nebulization, since it undercuts much of the significance of their many years of sacrifice, expense, and training in obtaining medical licenses. Many different rationalizations can be used by doctors to not even consider HP nebulization as a therapy. Nobody who has undergone extensive training in any field wants to feel that the huge amount of time and money invested may be irrelevant or even harmful in the treatment of a substantial number of their patients. Imagine a physician with special ICU and respiratory medicine training, and most of the COVID patients that previously flooded the ICUs no longer appear. How can he/she embrace an intervention that greatly reduces the number of people who need their expertise and that threatens their ability to make a significant income — especially when the traditional medical community is loudly dismissing the intervention as being scientifically unfounded or even frankly fraudulent?

The entire intent of this book is to make it clear that nearly all acute viral syndromes are curable, especially those contracted via the respiratory route, and **rapidly so**. This includes COVID and any future

pathogens that may emerge from mutation or from a laboratory.

In order to stop any pandemic, COVID included, it is essential that a cure not only exists, but that it is extremely cheap, available everywhere, easily administered, completely nontoxic, and not requiring the intervention of a doctor or hospital in order to be obtained. At this time, there is really only one such treatment that fulfills all of those requirements. It is:

Hydrogen Peroxide Nebulization

The reasons why hydrogen peroxide (HP) nebulization is such a perfect therapy for any acute respiratory virus is detailed in the book, and does not need to be repeated here. In extremely poor areas of the world, a nebulization machine, which still only involves a one-time expense of \$30 to \$40, can be shared by a household, or even a community that would use it at a local clinic or treatment center. Treatment times need only last 5 to 15 minutes in most circumstances. A pint of regular 3% HP, to be used as is or with variable degrees of dilution with water or saline solution depending on patient tolerance, has a retail cost of 1 to 2 dollars throughout the planet. The amount of HP for a treatment can be between 1 and 3 cc of the 3% solution. One pint has 473 cc of solution.

This means that a cure, or effective kickstart to the rapid resolution of a respiratory virus, including COVID, could cost as little as $\frac{1}{4}$ of a penny.

The amount of electricity needed to run the nebulizer, which would be inconsequential as well, would

be the main “expense.” It should also be emphasized that HP nebulization quickly and effectively resolves the common cold and all variety of influenza viral syndromes, depending on long how the patients have been infected when the nebulization is initiated. It also means that $\frac{1}{4}$ of a penny can do what many extended hospitalizations costing many hundreds of thousands to millions of dollars do not do: heal the patient, spare the patient much suffering and possibly death, and permit the patient or family of the patient to avoid complete financial devastation.

HP is the prototypical Bio-Oxidative therapy, as will be discussed in the book. Vitamin C, ozone, ultraviolet blood irradiation, and hyperbaric oxygen therapy also come under this category. They all share final common pathways in their eradication of infecting pathogens. When available, any of these other Bio-Oxidative therapies will synergize with HP nebulization to achieve virus resolution even more rapidly. This is especially

Considering its essential role in normal metabolic function, along with the effective storing and release of oxygen, HP actually serves as an important nutrient in the body.

important when the infection is already too advanced to be resolved quickly by simply eradicating the virus in the nose and throat areas, as can be readily accomplished with the HP nebulization alone.

Whenever available, vitamin C therapy, especially via intravenous application, should accompany the HP nebulization if any virus-related symptoms are already present. Vitamin C and HP are natural anti-pathogen partners, and one literally fuels the impact of the other. Furthermore, all infections deplete vitamin C stores in the body, which are essential for immune support and tissue integrity. In particular, acute respiratory viral syndromes like COVID can rapidly induce a state of acute scurvy in the body if not promptly resolved. This body-wide vitamin C depletion can often turn a readily curable infection into a protracted or even incurable infection if not promptly addressed.

Finally, as wonderful as it is to have multiple effective therapies for COVID, there is another benefit as or even more significant as having the ability to cure oneself of this infection. Simply put:

**Life can once again be lived without
continuously worrying about
loss of health, dealing with great
suffering, and even dying.**

Very many people have become deathly afraid of contracting COVID, in spite of the fact that the mortality resulting from this infection has declined substantially, and is now on the order of one tenth of a percent (1 in 1,000). Not only is the fear palpable, it is irrational. For almost a year now, when stopping at a traffic light many drivers “socially distance” in their cars and maintain more than a car length from the cars ahead of them until the light changes. Scientifically, this is ludicrous, but it does underscore the amount of fear that has been instilled in a very large number of

people. Some people are paralyzed with fear when they see someone's mask drop below the nose for a moment as if the offender were an assassin. Some will literally attack such an individual. Currently, the fear of the virus is ***much more debilitating*** to the population as a whole than the virus itself.

Of note, it is very likely that the increased consumption of the right supplements (vitamin C, vitamin D, magnesium, zinc, and others) early on in 2020 has accounted for much of this decline in COVID mortality from the earliest data. Very shortly after the virus began its rapid spread, bottles of vitamin C and other supplements were quickly in short supply. Bottles of HP rapidly disappeared as well.

Although not the primary message of this book, it turns out that HP nebulization, along with a few other interventions, effectively restores a normal, or near-normal, gut microbiome in many people who end up nebulizing on at least a semi-regular basis. This is of ENORMOUS consequence to general health, and if the world was not in the clutches of the COVID pandemic, it would be the primary message of this book.

The information in the book demonstrates that a continued fear of COVID or any other respiratory virus is irrational. Owning a good nebulizer and having a fresh bottle of HP readily at hand will keep COVID from destroying ***mental*** health before it has the chance to threaten ***physical*** health. That means a return to normal life is possible, safe, rational, and easy to achieve.

Important Note About the Book's Claims

Most of the significant scientific assertions made in this chapter and throughout the book are directly referenced to the peer-reviewed scientific literature. Much of the data comes from the most trusted journals in the scientific world. And with services such as are offered on the PubMed website, the information is easily and quickly accessible. The only requirement is that the individual, especially the clinician, has enough interest to take a few seconds (literally) to check out the validity of an assertion. PMID numbers are included with nearly all of the citations. Just typing in this number and nothing else in the search box on the webpage (<https://pubmed.ncbi.nlm.nih.gov/>) quickly displays the abstract and oftentimes the full article on the research being cited. The reasons why such important and significant research never really makes its way into the clinical practices of most physicians around the world is discussed above.

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How to Completely Cancel COVID

Is the title of this chapter too good to be true? Be assured that it is not. But readers will need to reach that conclusion on their own. Let's start with the following unqualified assertion:

Applying a protocol of hydrogen peroxide (HP) nebulizations [the repeated inhalations of a fine HP mist] after an exposure to COVID or after the initial symptoms of COVID are noted offers a prompt and definitive cure in nearly everyone.

Although many other therapeutic agents can be used in tandem with HP nebulization, this treatment is so effective that most people do not need anything else to stop COVID, or any other cold or flu syndrome soon after exposure or contraction. If treatment does not begin until the infection is well advanced, additional therapies will be important and should be employed. These adjunctive therapies and additional therapeutic agents will be discussed at length later in the book. In

no case, however, is panic or fear in order. Virtually all acute respiratory infections, including COVID, are preventable and curable with the appropriate use of HP nebulization as a monotherapy or in conjunction with other proven therapies for more severe infections.

What About Vaccinations?

Whether or not a vaccination for COVID should be taken will not be addressed in this book. Much has been written already about the potential benefits and harms that can result from any vaccination, including the various COVID vaccinations.

The only voices that are allowed to be heard in the current culture are using FEAR to motivate the population to totally change the way we live, work, and play — to STAY SAFE we all must wear a mask, avoid large groups of people, social distance, and get an admittedly

Why is a vaccine necessary when the targeted infection or toxin can simply be prevented, or if contracted, easily cured?

experimental vaccine. But what if there were an easy and ridiculously inexpensive way to prevent, treat, and stop the virus that causes COVID dead in its track as this book contends?

Perhaps before deciding on whether to take a vaccination, the reader should first ask this question:

Why is a vaccine necessary when the targeted infection or toxin can simply be prevented, or if contracted, easily cured?

Fortunately, the reader can review the information in this book and decide if the information and suggestions are correct, and whether an irrational fear of a viral infection will be allowed to forever destroy their quality of life.

HP is the Ideal Agent for Treating Clinical Infections

Nearly everyone knows that HP is a powerful disinfectant that can readily decontaminate counters, floors, walls — as well as most surfaces outside the body — of any pathogens that are present. Also, in high enough concentrations and long enough exposures, HP is also known to have a whitening, or bleaching, effect on whatever surface it is administered. It is also commonly used by physicians and laypersons around the world to help eliminate the pathogens growing in open, infected wounds. This anti-pathogen effect of HP has been well-documented in the research literature.¹⁻⁴

When one considers that the definition of a disinfectant is that of an agent used to destroy pathogens on an external surface, it is understandable that any internal application of such a substance would be assumed to be inappropriate and even toxic. Therefore, somewhat understandably, traditional medicine generally opposes the intake of HP by any route into the body for any reason.

Since HP is technically a disinfectant which has widespread applications in killing pathogens outside of the body, it is just **assumed** that it would also

be highly toxic to the cells of the body if used in any internal application. Those who oppose therapeutic applications of HP never base their position on scientific arguments. Rather, their opposition is based on assertions, devoid of any scientific substantiation, that any agent with disinfectant and/or bleaching capabilities must be inherently poisonous and therefore should never be administered inside the body. Admittedly, this is absolutely true for most cleaning or disinfecting agents. But for HP, this assertion is not even remotely valid. Quite the opposite, in fact, is true.

The very unique properties of HP actually make it an ***ideal*** agent to treat internal infections, especially those acquired via the respiratory route. The inhalation of HP by nebulization has been shown to be extremely effective for the rapid elimination of any pathogen presence in the sinuses, nose, throat, and deep into the lungs. As well as being highly effective, HP nebulization is also nontoxic, extremely cheap, and readily available to just about anyone on the planet.

Until the COVID pandemic hit the United States and the rest of the world, the primary drugs offered by traditional medicine for a cold, influenza, or any other respiratory virus only alleviated clinical symptoms or provided general supportive care. The pandemic, however, has spawned an enormous amount of research worldwide, and the scientific journals have stepped up and fast-tracked the publication of much quality clinical research from years to just months. There are now multiple treatments that have been documented to rapidly resolve viral respiratory syndromes, including COVID, with virtually no clinical

failures. They can be considered as near-absolute cures when properly administered.

HP nebulization simply stands out as the singular most impressive therapy for such viruses. Of course, its negligible cost means that there will never be any large prospective, double-blind, placebo-controlled trials of

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HP nebulization. This fact alone will forever prevent it from being accepted by mainstream medicine. Such research usually costs millions of dollars. Industries, including the pharmaceutical industry, are in business to generate large profits. Dumping millions into something that can never produce a large monetary return simply does not occur.

Amazing Health-Related Properties of HP

HP has the following properties that account for its powerful and positive clinical impact:

1. Documented antiseptic (disinfectant) abilities when applied externally
2. Completely nontoxic when appropriately administered to an individual

3. Tiny in size, nonionic, and permeable to all cell, intracellular organelle, and pathogen walls and membranes
4. Present everywhere in the body, continually produced both inside and outside the cells⁵
5. Chemically stable and not readily reactive like other ROS (reactive oxygen species), as it requires specific cofactors to have its pro-oxidant, pathogen-killing effect
6. Rapidly produces hydroxyl radicals (pro-oxidant effect) in the presence of unbound iron (Fenton reaction)
7. Can spontaneously be generated from water to a limited degree
8. Increased production in the face of infection and inflammation⁶
9. Generated in massive amounts into the extracellular space by phagocytes in order to respond to pathogen presence^{7,8}
10. Secreted continually by the cells lining the airways, serving as a natural defense mechanism against newly inhaled pathogens
11. Naturally present in exhaled breath of healthy human subjects⁹
12. Increased presence in exhaled breath when antioxidant status is improved in COPD patients with nebulized N-acetylcysteine¹⁰
13. Pulmonary infection and inflammation results in a compensatory increased production of HP, as measured in the exhaled air

14. Naturally present in the urine, helping to minimize the occurrence or persistence of infections there
15. Metabolically breaks down into water and oxygen after pathogens are killed
16. Improves blood oxygenation when inhaled by nebulization
17. Effectively serves throughout the body as an effective storage form of oxygen
18. Extracellular production of HP is massively increased in the presence of highly-dosed vitamin C
19. Can be considered a nutrient by virtue of its effects on metabolism and ability to mobilize stored oxygen
20. Activation of lymphocytes ¹¹
21. In an insulin-like fashion, HP can markedly enhance glucose transport/uptake in fibroblasts ¹²
22. HP has a mucolytic (mucus-dissolving) effect that is also of great benefit in mobilizing and eliminating any secretions associated with a pulmonary infection being treated¹³

Not Only Safe, but Important for Optimal Health

There are few molecules in the body that play a more important role in maintaining optimal health than HP. It is essential for the ongoing defense against pathogens while also playing a pivotal role in the regulation of normal metabolism. HP is present throughout the body, continually generated both inside and outside of all cells.¹⁴⁻¹⁶ HP is literally a ***naturally-generated***

ated antibiotic that gives the body its best chance at preventing an infection or at resolving an infection once contracted.

Designed by nature, it is also intimately involved in vital messaging and signaling functions between different cells as well as inside the cells themselves in order to sustain normal metabolic function. Furthermore, HP is present in the body in sizeable amounts, with some data indicating that as much as 5% of oxygen intake is utilized in the generation of HP.¹⁷ An extremely simple molecule, HP presents no toxicity to the body except when arbitrarily administered in very high doses and concentrations, conditions that can literally make any substance toxic. Considering its essential role in normal metabolic function, along with the effective storing and release of oxygen, HP actually serves as an important nutrient in the body.

Although HP reliably and completely kills all known pathogens, it seems to be counterintuitive to many people, including scientists and physicians, that something with such potent anti-pathogenic properties could be so completely nontoxic when utilized in an internal application. Yet, this is precisely the case. The primary reason for this is that HP breaks down to **water and oxygen** after completing its pathogen-killing task.

Water and oxygen are arguably the two most important molecules in the body. They are both essential for life, not to mention their ongoing roles in healing and good health. It is hard to imagine any molecule having more optimal and beneficial metabolic byproducts than water and oxygen, yet this is the case with HP. After the pathogens in an infected area of

the body have been eradicated, the two most important factors in helping the damaged tissue to completely heal are water and oxygen, which directly support optimal hydration and oxygenation in that tissue. This

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metabolic outcome is assured when HP is used as the anti-pathogen treatment.

Hydration helps to dilute the acidity seen focally in areas of damaged tissue (helping to normalize pH). It also optimizes the chemical interactions required for healing and ongoing health. Increased tissue oxygenation promotes the additional production of energy needed to support good healing. Oxygen is recognized as the single most important factor for healing and optimal tissue repair.¹⁸ In fact, the metabolic role played by HP along with its ability to break down into water and oxygen can legitimately allow it to be regarded as an important nutrient, or at least as a substance with significant nutritive impact.

The vital role of HP in the metabolism of the body is substantiated by the fact that a great deal (as much as 5%) of oxygen intake ends up in HP synthesized inside the body. In cell culture, greater oxygen exposure clearly results in increased HP production.^{18,19}

This effect is especially pronounced inside the mitochondria, the primary sites of energy production in the cells.²⁰ In reality, the HP molecule actually serves as a vital oxygen **storage system** in the body, releasing oxygen into the microenvironments where and when it is needed most.

Not only is HP present in and around all the cells of the body, it is also secreted from some cells that line areas that are technically outside of the body. The outer surfaces of the sinuses, throat, airways, and all the way down through the length of the gut are physically inside the body, but they share their immediate surroundings with the external environment and not the internal environment. When these lining cells produce HP and secrete it, the HP does not move into the extracellular space. Rather, it becomes a coating on the outer surface of these cells. Even when no infection or inflammation is present, the pulmonary epithelial cells actually express this HP in order to coat the cellular surface facing the air flow. This coating serves to help protect the lungs from the new pathogens that are always present to some extent in each breath.²¹ When infection and/or inflammation are present, this HP production is significantly increased by the cells lining the airways, as determined by the significantly increased amount of HP measurable in exhaled breath.²² This is consistent with the concept that the body naturally increases HP production and presence wherever pathogens need to be confronted. HP is also consistently found in the urine, suggesting that it serves to help maintain a non-infected urine as well.²³

Mitochondria are the energy-producing “generators” inside each cell, and by extension the entire

body, and they require both oxygen and water to do their work. Interestingly, the highest concentrations of HP found in the body are present in the mitochondria which strongly suggests that the ability of HP to break down into water and oxygen plays an important, if not essential, role in the normal metabolic function of the cell. But that is not the only place where HP is found. It is also literally present everywhere in the body which indicates that it plays a multitude of significant metabolic and physiological roles in the body. As with other signaling molecules that modulate metabolic functions, the microenvironment in which HP is found determines whether HP directly provides pro-oxidant (toxic)

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or antioxidant (nutritive) impact, or whether it remains chemically inactive and stable as a storage form of oxygen.²⁴

The ubiquitous nature of HP is further supported by its widespread presence outside of the body and throughout nature. HP is present naturally in drinking water, rain water, and sea water. In fact, in the **absence** of other agents, HP has been documented to be spontaneously generated in microdroplets of water,

with greater production resulting as the droplets get tinier.^{25,26} Some HP is assimilated from the diet as well.

Although some would consider HP to be a pharmaceutical agent, in truth it is a naturally occurring molecule that can be found in the food we eat, water we drink, and the air we breathe. These facts give even further support that it is a safe substance for use inside the body. Unlike even the best of pharmaceutical agents, including antibiotics, that can harm or kill — and they do so on a regular basis — nobody dies or suffers any toxic effects from HP properly applied in the right dose and form of application, particularly via nebulization.

On the other hand, statistical data indicates that about 2,000,000 patients hospitalized in the United States have serious adverse prescription drug reactions every year. Furthermore, over 100,000 patients in the United States die every year from prescription drugs that have been ***properly-dosed and prop-***

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erly-administered.²⁷ Even more deaths occur with such drugs that have not been dosed and administered appropriately.

The worst side-effect from HP nebulization administered within the boundaries recommended in this

book can occur when the treatment is too prolonged or too concentrated. In these cases, minor, self-limited side effects (nose and throat irritation) can occur. However, just as dilute acids are an important part of the normal diet, concentrated acids can rapidly kill if ingested. It is important to note that extremely concentrated HP can be fatal but a dilute solution of 3% or less is exceptionally safe.

Perhaps in an effort to protect the profits of the pharmaceutical companies and hospitals, traditional researchers publish reports on deaths and severe side effects resulting from totally inappropriate applications of excessively concentrated HP while almost completely neglecting reports on its positive impacts, except for such applications as tooth whitening or wound disinfection.^{28,29}

Isn't hydrogen peroxide a pro-oxidant? How could that be good for the body?

Hydrogen Peroxide (HP): Facts and Misconceptions

HP is frequently characterized as an unstable substance presenting significant toxicity to all cells. In fact, the opposite is true. Except under certain limited circumstances, HP is very stable and largely nonreactive, with its ongoing presence inside cells being essential for supporting critical biochemical pathways and resulting in no toxicity whatsoever.

HP is also referred to in the scientific literature as a ROS (reactive oxygen species) molecule. While technically accurate, HP is neither ionic nor a free radical, characteristics that define the nature of other common ROS, such as superoxide and hydroxyl radical. However, when in the presence of transition metal ions such as iron or copper, HP can generate the highly

pro-oxidant ROS species known as hydroxyl radical, in a chemical pathway known as the Fenton reaction.¹ This hydroxyl radical is one of the most reactive pro-oxidants known to exist, never accumulating or migrating since it immediately oxidizes whatever is next to it at the moment of its formation.²

It would probably be more accurate to consider HP as a “ROS-in-waiting.” By itself, it is nonreactive, but it is ready to promote pro-oxidant impact when called upon. Increased pro-oxidant activity is critical to killing pathogens, but such increased activity is not desirable inside the healthy cell. The “standby” ability of HP to generate hydroxyl radical only when needed accounts for its unique cell-protective properties. Furthermore, it is very important to realize that an increased presence of HP can initiate responses that can limit or **repair** oxidative damage. This further enforces the concept that HP serves as a central regulating molecule in the body, ready to promote either pro-oxidant or antioxidant impact depending on the “microenvironment” in which it is found at any time.³

This selective reactivity of HP in the presence of unbound iron allows it to selectively target pathogens, which accumulate and literally thrive on iron. Pathogens, with their exceptionally high iron content, literally put a target on themselves that allows HP to directly attack them with massive amounts of oxidation while leaving normal, uninfected cells alone. It cannot be overemphasized that appropriate HP therapies just augment and bolster the normal defense mechanisms that the body uses to naturally kill pathogens and eradicate infections.

Activated phagocytes, the pathogen-scavenging white blood cells that are routinely summoned to any site of infection and inflammation, naturally generate massive amounts of HP into the extracellular space.^{4,5}

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This allows access of the HP to wherever it is needed to oxidize and kill the pathogens. Similarly, these phagocytic white blood cells have also been documented to have exceptionally high levels of vitamin C, which strongly stimulate the conversion of HP into the powerful oxidizing agent, hydroxyl radical, inside these cells. This allows these phagocytic cells to readily oxidize the pathogens that have already been assimilated or “ingested.” The phagocytosis of pathogens proceeds in a manner analogous to what happens to food after it is swallowed. The HP acts on the pathogens like the digestive enzymes act on the food.

While vitamin C has long been documented to be enormously effective in the clinical resolution of all viruses and most other infectious diseases when administered appropriately, it is the ability of vitamin C to convert HP into hydroxyl radical that accounts for its direct ability to kill pathogens. Indirectly, vitamin C also has a wide array of properties that all act together to strengthen immune function in order to prevent or resolve nearly all infectious diseases.

It is when high doses of vitamin C are given intravenously that a large amount of HP is generated in the extracellular space^{6,7} At the same time, large amounts of vitamin C are also reaching the intracellular space. And the HP in the extracellular space is free to diffuse into the intracellular space. This means that at the same time vitamin C is converting HP into hydroxyl radical inside the cells, it is helping to generate new HP to enter the cells and continue to feed the production of hydroxyl radical until the pathogen-killing goal has been completely realized. This is analogous to vitamin C burning the gas in a car while simultaneously working to keep the tank full.

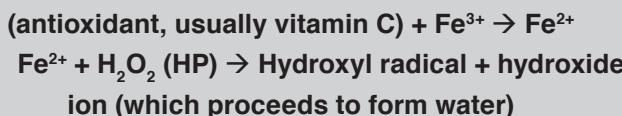
The Fenton Reaction

The Fenton mechanism or process, more commonly known as the Fenton reaction, produces hydroxyl radicals from HP.^{8,9} Named after the scientist, Henry Fenton who first discovered it, this naturally-occurring process is occurring in the body around the clock as part of the continued pathogen removal by a healthy immune system. This amazing, iron-requiring chain of chemical interactions is able to kill pathogens and the cells they have heavily infected. There is no residual toxicity associated with the reaction as the byproducts are simply oxygen and water. In healthy cells, a low concentration of iron as well as the presence of an enzyme (catalase) that breaks down HP directly into water and oxygen, protects them from hydroxyl radical damage. Administration of HP either via nebulization or by vein simply increases this natural, disease-resolving activity.

Fenton Reaction Chemistry

For those readers who are interested in a more technical explanation of the Fenton reaction, HP forms hydroxyl radicals when electrons are donated to it. In the body, it is nearly always ferrous ion (Fe^{2+}) that donates these electrons, reverting back into ferric ion (Fe^{3+}) in the process. When there is predominantly ferric ion present inside the pathogens or cells, another electron donor is needed to first convert the ferric ion to ferrous ion. This donation is very commonly, but not exclusively, achieved by the electron-donating capacity of vitamin C. Other transition metals aside from iron that also have multiple valence states (degrees of electron saturation) can also fuel the Fenton reaction when they are present to an excess degree, promoting different diseases depending on how much excess oxidative stress is being sustained in the affected tissues.¹⁰ The basics of the Fenton reaction can be viewed in this manner:

Electron donor



HP not participating in the Fenton reaction can remain intact or naturally break down into water and oxygen.

When upregulated to a massive degree, the metabolic breakdown of HP into hydroxyl radicals also

appears to be the final common pathway utilized by all of the other highly effective bio-oxidative therapies in the killing of pathogens. This includes vitamin C, ozone, ultraviolet blood irradiation, and hyperbaric oxygen therapy. The mechanisms and metabolic pathways leading up to the utilization of the Fenton reaction can vary, but no pathogen or pathogen-filled cell can be **selectively** destroyed without the massive increases in oxidative stress generated by an optimized Fenton reaction.

Anything that can non-specifically upregulate oxidative stress to a massive degree can destroy cells and pathogens, such as chemotherapy or a very potent toxin. However, the incredible utility of the anti-pathogen activity of HP is its ability to target pathogens and pathogen-infected cells, while leaving normal cells and beneficial microbes untouched.

The Fenton reaction requires not only an optimal supply of HP for hydroxyl radical production, it also needs a transition metal to donate electrons to the HP in order to achieve this production. Typically, this transition metal is always iron, with the ferric ion being reduced to ferrous ion in order to pass the electron on the HP.

The natural fuel of pathogens is free iron, and the more iron they accumulate, the more virulent and clinically aggressive they become. In contrast to pathogens, non-pathogenic microbes do not accumulate iron. Because of the essential role played by iron in stimu-

lating the Fenton reaction to produce hydroxyl radicals, pathogens ***self-target themselves for destruction*** by virtue of their high iron content. Clinically, large doses of vitamin C continually convert the ferric ion to its ferrous form, and HP is then able to destroy the pathogens with large amounts of hydroxyl radicals formed from the electrons passed along by the ferrous ions.

In many patients, highly-dosed vitamin C is also effective in killing cancer cells and often completely resolving a cancer. This is because cancer cells, like pathogens, thrive on and actively accumulate iron. Additionally, cancer cells also have low levels of catalase, which is the enzyme that naturally functions to break down HP into water and oxygen. With HP levels high (from low catalase), iron levels high, and large amounts of vitamin C being administered, the stage is set for a continued high production of hydroxyl radicals until the cancer has been destroyed. This Fenton reaction activation does not occur in healthy cells in the body, as they do not accumulate iron, and they have normal catalase levels that keep the presence of HP at a minimal level. Because of this, a high dose of vitamin C is helping to strengthen the natural immune defenses of the normal cells at the same time it is selectively killing the cancer cells. The use of chemotherapy does also kill cancer cells, but it is not selective and it damages/kills normal cells at the same time.

At the same time high doses of vitamin C are fueling the Fenton reaction, they are also directly generating large amounts of HP outside of the cells, which is then free to diffuse into the cells and provide more HP to be converted into hydroxyl radicals. HP

inside the cells also helps to mobilize stored iron (ferritin) into free iron that can also work to sustain the Fenton reaction until no pathogens remain to be killed, as noted above. HP and vitamin C are natural physiological partners, and together they are the optimal tandem agents for dealing with any infections. Of course, additional therapies can always be added, but properly administered HP and vitamin C should nearly always do the job.

Nebulization: Old Treatment, New Applications

Nebulization is a process that converts a liquid form of a therapeutic agent into a fine mist that can be readily inhaled. This allows the direct contact of such an agent with the cells lining the sinuses, oral cavity, throat, and respiratory tract.¹

This form of drug administration has a long history in the annals of medicine, dating back more than 3,500 years to ancient Egypt.² Also known as inhalation therapy,^{3,4} nebulization offers a number of unique benefits not realized with other forms of drug administration, especially for pulmonary conditions. These benefits include the following:

- ✓ Provides a direct route of drug administration to affected cells/tissue without needing to circulate throughout the body, such as with agents that ease breathing or that kill pathogens.

- ✓ Moistens the areas nebulized, helping to mobilize and expel tenacious mucus and other secretions, such as infected sputum.
- ✓ Reliably lessens cough long-term after tenacious secretions get mobilized and expelled.
- ✓ Permits the use of lower amounts of the therapeutic agents needed in the lungs than would be needed if given systemically, which would lessen any potential toxicity of those agents as well.
- ✓ Permits a limited degree of systemic assimilation of the nebulized agent throughout the body.
- ✓ Permits a direct attack on areas of chronic pathogen colonization (CPC) in the sinuses, oral cavity, throat, esophagus, and airways (aerodigestive tract).

Inhalation Medicine

Inhalation medicine focuses on the proper applications of agents via nebulization. Although a well-established form of treatment, nebulization remains little-used in clinical medicine, especially when any condition other than pulmonary in nature is being treated. Nebulization delivers a mist of very tiny droplet size into the lungs by inhaling it with a mouth-piece or full mask. Depending on the type of nebulizer and the solution being atomized, a particle size as tiny as 3 to 7 micrometers can be inhaled (some mesh ultrasonic nebulizers). Generally, most air jet nebulizers

deliver a slightly larger droplet size. Both devices are very effective clinically.

A variety of agents can be applied in this manner for the purpose of treating lung infections, improving lung function in the absence of infection, and even to treat some non-pulmonary, systemic conditions.⁵⁻⁷ The therapeutic agents being administered are dissolved in a solvent, typically water or saline solution, and then converted into a fine mist or even a smoke-like vapor, depending on the nebulization device. Generally, saline is probably a better choice than water as a solvent, especially when repeated and prolonged nebulizations are anticipated. Inhalation of pure water is known to induce cough, and except for the expectoration of mucus and secretions, it is not desirable to provoke a non-productive cough, which can result in further irritation of the throat after pathogens have been eliminated.⁸ Furthermore, the chloride anion from the saline solution appears to have its own anti-pathogen impact.

The solution is then inhaled with normal breathing, readily reaching deep into the lungs. When inhaled using a full facial mask, the nebulized agent is also reaching inside the sinuses, along with all the mucosal surfaces in the nasopharynx and upper oropharynx. Nebulization of various agents has been applied to patients already being supported on mechanical ventilation, and it has been shown to be effective in the prevention of pneumonia in such patients.⁹ It also appears that nebulization might finally be receiving the appreciation it deserves, as an increasingly number of drugs and other agents are being delivered

to patients by this route versus oral or intravenous administration.¹⁰

A New Medical Discipline?

Even though patients have been inhaling a wide variety of medicines and nutraceuticals for a very long time now, the therapeutic applications of this approach have never been given sufficient attention or emphasis. With the exception of inhalers for such conditions as asthma or chronic lung disease, inhaling a therapeutic agent for any other medical condition is rarely given consideration, and certainly it is never done so in a routine or “accepted” manner.

Very many doctors have never had their patients nebulize or inhale anything, in spite of the substantial amount of published data showing this is an excellent adjunct in the treatment of many conditions. In some conditions, inhalation therapy can offer the greatest chances of a positive clinical impact. It may well be the best way to administer antibiotics and other anti-pathogen agents for lung infections, as an animal study showed that inhaled vancomycin provided ***higher lung tissue concentrations*** than was achieved by intravenous administration.¹¹ But like the example of intravenous vitamin C, discussed elsewhere, a proof of benefit in the scientific literature, however impressive or extensive, does not assure the use of a positive intervention. And it does not matter that it has been shown to be even ***more*** effective than any of a multitude of other “accepted” treatments. Perhaps the emergence of inhalation medicine will be better received, since it does not exclude the use of prescription agents in deference to using only natural nutrient agents.

Documented Use

A number of examples of applications of nutrient agents and prescription drugs via inhalation/nebulization from a limited review of the medical literature includes the following:

- ✓ Vitamin A supplementation has been administered via aerosol spray in asthmatic children.¹² In an animal study, the administration of vitamin A by inhalation was shown to be more effective than by intramuscular administration for the prevention of hyperoxia-induced lung injury, a type of lung injury that can occur in premature birth babies.¹³
- ✓ Vitamin D nebulization was shown to enhance lung maturation in rat pups.¹⁴ In asthmatic children vitamin D supplementation was shown to improve their clinical status, suggesting vitamin D nebulization might also offer an excellent therapeutic outcome in such children.¹⁵
- ✓ Vitamin E nebulization has been successful in an animal model at lessening the lung injury after burn and smoke inhalation. It improved pulmonary oxygenation and markedly reduced the time needed for assisted ventilation.¹⁶ Another study determined that the nebulization of different carriers of vitamin E was successful in targeting the lungs.¹⁷
- ✓ Vitamin C has been successfully nebulized, appearing to enhance smoking reduction and cessation.¹⁸

- ✓ Sodium bicarbonate solution nebulization was found to rapidly and effectively reverse the symptoms secondary to accidental chlorine gas inhalation in a 9-year-old boy. It was concluded that the sodium bicarbonate quickly neutralized the hydrochloric acid that was locally produced in the lungs of this boy after the chlorine was inhaled.¹⁹ A study on 86 patients with chlorine gas inhalation who received nebulized sodium bicarbonate revealed a good clinical response in most of them, with no evidence of a negative impact in any of them.²⁰ In three males aged 19 to 20 years of age the nebulized administration of a 3.75% sodium bicarbonate solution promptly relieved the cough, chest pain, and shortness of breath that resulted from an acute chlorine gas inhalation.²¹
- ✓ Sodium bicarbonate nebulization has also been used in the treatment of acute severe asthma.²²
- ✓ Multiple studies have shown that the nebulization of corticosteroids was effective and well-tolerated in the management of acute and chronic asthma in young children.^{23,24}
- ✓ Coenzyme Q10 nanosuspensions have been successfully nebulized and appear to offer a direct antioxidant delivery to the lungs.²⁵
- ✓ Nebulization of a combination of vitamin C, glutathione, and sodium bicarbonate appeared to help mobilize the clearance of mucus from the airways.²⁶

- ✓ Nebulization of N-acetylcysteine in elderly patients with ventilator-associated pneumonia significantly controlled the degree of lung infection and shortened the time for mechanical ventilation and lessened the amount of antibiotic usage.²⁷
- ✓ Nebulization of 7% sodium chloride solution has been shown to be an effective adjunctive therapy for the management of cystic fibrosis-related symptoms.²⁸
- ✓ Nebulization of warmed 0.9% saline was beneficial in the mobilization of purulent sputum in patients with bronchiectasis.²⁹
- ✓ Nebulization of vancomycin was effectively administered in mechanically ventilated patients with methicillin-resistant *Staphylococcus aureus* pneumonia. Maximal lung concentrations of the antibiotic were superior to that achieved with intravenous administration.³⁰
- ✓ Nebulization of tobramycin was shown to be effective in the eradication of *Pseudomonas aeruginosa* in the sputum specimens of cystic fibrosis patients.³¹
- ✓ Nebulization of special bacteria-infecting viruses known as bacteriophages has successfully improved the survival of rats infected with methicillin-resistant *Staphylococcus aureus*.³²
- ✓ Nebulization of interferon in treating infants with bronchiolitis successfully

alleviated coughing and decreased the duration of wheezing.³³

- ✓ Nebulized immunoglobulin treatments were able to successfully reduce the incidence of upper respiratory tract infections in three brothers with antibody deficiencies.³⁴
- ✓ Nebulization of ketamine has successfully served as premedication before the induction of anesthesia in young children.³⁵
- ✓ Therapeutic levels of milrinone were achieved by pre-surgical nebulization in cardiac patients in order to successfully reduce the severity of pulmonary hypertension during surgery.³⁶

The possibility of nebulizing hydroxychloroquine, an effective anti-COVID prescription drug, in the treatment of early COVID lung infection has also been explored. Early results show that this form of HCQ administration is very well-tolerated, and it appears to deliver therapeutic amounts of HCQ in the respiratory cells where the virus is primarily located in early infections.³⁷

All of the citations above underscore a common phenomenon in “modern” medicine. Even though something like nebulization can be repeatedly shown to be a superior form of treatment administration, that is not the determining factor as to whether it will become commonly used. Generally, if something is not an expensive pharmaceutical, it does not reach the medicine textbooks or clinical medicine manuals. And the average clinician has no interest in being a “trail-

blazer” for any remedy lacking the complete support of his/her colleagues.

How does HP resolve respiratory infections?

Nebulization with HP is truly an ideal therapy for dealing with a number of conditions, but especially acute respiratory viral infections. This therapy has at least six characteristics that no other treatments fully share, although there are a number of therapies that do have two or three of these characteristics. These six characteristics of HP nebulization are:

1. Highly effective clinically
2. Directly promotes tissue healing after pathogen eradication because byproducts are water and oxygen
3. Completely nontoxic when used as recommended
4. Of inconsequential cost to even the poorest of patients
5. Readily available, and not requiring prescription
6. Very easy to administer and not requiring assistance of a physician, clinic, or hospital

Respiratory viral infections, including influenza, the common cold, and even COVID are ideally addressed by nebulizing agents that are known to readily inactivate viruses and kill cells already having a high virus content. Furthermore, while it has been raised as a concern in the literature, nebulization of a potent anti-pathogen agent will not promote the spread of any infectious source in the lungs.³⁸ But if a

patient is heavily infected and agents that have no anti-pathogen properties are vigorously nebulized, there could be an increased release of infected droplets/mist around the patient.^{39,40} However, it can be anticipated that any potential contagious spread from an infected patient will promptly be **lessened** when an anti-pathogen agent such as HP is being nebulized, which results in an immediate and rapid decline of

Just as HP easily eradicates pathogens when applied to a contaminated surface outside of the body, it eradicates those pathogens just as readily when the contaminated surface is the mucosa and cells lining the nose, throat, and lungs.

viable pathogens that would otherwise be potentially available for spread.

While nebulizing appropriate agents with a proper protocol can often serve as an effective monotherapy in managing and resolving such respiratory infections, it is usually best to utilize such nebulization as a **natural adjunct** to other therapies documented to be effective in resolving acute viral infections, such as properly-administered vitamin C. Regular nebulization with the correct agents can also resolve other lung conditions, and it can help to normalize the gut microbiome, which in turn positively impacts virtually all medical conditions affecting other areas of the body.

With acute respiratory viral infections, the virus is not only contracted by gaining a foothold in the nose and throat, it also replicates primarily in this area. Although enough virus disseminates throughout the body to cause the systemic symptoms (fever, chills, headache, malaise, etc.), the proliferation of the virus in the nose, throat, and lungs makes this area the ideal place to target with a potent antiviral therapy. An early elimination of the virus presence here acts to “cut the head off of the viral snake,” and then the body can readily mop up the remaining virus elsewhere in the body. How quickly this occurs relates to how many other things are being done to support the immune system and/or kill the virus (vitamin C, vitamin D, magnesium, zinc, etc.).

Just as HP easily eradicates pathogens when applied to a contaminated surface outside of the body, it eradicates those pathogens just as readily when the contaminated surface is the mucosa and cells lining the nose, throat, and lungs. And unlike nearly all traditional house cleaners and disinfectants, using HP nebulization promotes and supports the normal defense mechanisms of the body, leaving behind no toxic contaminants or byproducts after killing the pathogens.

Viruses, Basic Facts

A virus is an infectious agent that by itself is not technically alive, since it is incapable of replicating without a host cell. The host cell can be of animal, plant, or even bacterial origin. While there are many variations in the structure of a virus, the basic makeup of a single viral particle, or virion is simple. Genetic

material (RNA or DNA) is contained or wrapped up inside a shell of protein, known as the capsid.

The relative size of most viruses compared to cells and many other biological structures is also quite tiny. Most virions range between 20 and 500 nanometers in diameter. By comparison, a red blood cell generally is between 6,000 and 8,000 nanometers in diameter. Most bacteria are also vastly larger than individual viral particles.

Viruses cause a variety of infectious diseases, and they are transmitted by several different routes. These routes include the following:

- ✓ From the gastrointestinal tract (via food or water),
- ✓ Via blood and bodily secretions (transfusions, accidental needle sticks, unprotected sex, etc.), and
- ✓ Via respiratory exposure (common cold and influenza, including COVID) [statistically most common route].

Once inside the host cell, viruses, including COVID, utilize enzymes known as RNA polymerases to replicate the viral nucleic acid needed to produce new viruses.^{41,42} If these polymerases cannot function, the virus cannot multiply and the infectious “process” is effectively terminated as the body can then “mop up” the remaining viral presence.

Intracellular zinc has been shown to directly inhibit the activity of these viral polymerases.⁴³ When this activity is significantly inhibited, clinical resolution of the viral infection promptly ensues. Several effective anti-COVID therapies have resulted from the use

of agents that optimize intracellular levels of zinc. An augmented or facilitated transport of zinc into cells has even been shown to reverse antibiotic resistance otherwise seen with some pathogenic bacteria.⁴⁴ Increasing intracellular zinc levels in this manner has also been shown to have significant anti-cancer properties with

Once inside the host cell, viruses, including COVID, utilize enzymes known as RNA polymerases to replicate the viral nucleic acid needed to produce new viruses.

increased induction of cell death in such cells.^{45,46} In addition to striving for the therapeutic goal of normalizing intracellular levels of magnesium, vitamin C, and glutathione, these zinc properties make it clear that normalizing intracellular zinc levels is also critical to optimizing cellular health.

These effective anti-COVID therapies have centered on agents known as zinc ionophores. An ionophore is a chemical species that reversibly binds an ion such as zinc, releasing it into the cytoplasm after it is transported across the cell membrane. Many agents in their pure ionic form such as zinc have difficulty in passing through the membrane. Increased zinc supplementation can increase the amounts of zinc that end up inside the cells, but the binding of zinc to an effective ionophore is a far more efficient way to deliver zinc intracellularly. Furthermore, chronically high dosing of zinc in most of its supplemental forms does eventually result

in some toxicity. Nevertheless, it is very important to avoid being in a state of chronic zinc deficiency. Good ionophore-facilitated transcellular transport eliminates the need for pushing zinc supplementation to such

While the degree to which mask-wearing is vital to prevention is a point of increasing contention, general sanitary measures and avoiding crowds are nevertheless common-sense ways that will always diminish the concentration and total amount factors of virus exposure.

potentially toxic levels.

Both prescription and supplemental zinc ionophores exist. Hydroxychloroquine and chloroquine are the two most prescribed zinc ionophores in the treatment of COVID.⁴⁷ Both drugs have been shown to be highly effective in resolving COVID infection, especially early in its clinical course.⁴⁸ Not surprisingly, they are also both highly effective in preventing the contraction of COVID, although there has been much pushback politically to convince doctors and the public that they are of little to no value and possibly presenting a significant chance of great harm. But to be clear, both of these drugs are **exceptionally safe**, especially in the doses recommended for either treatment or prevention.

Quercetin is a naturally-derived polyphenol supplement that appears to serve as a zinc ionophore. In fact, much of the positive impact of quercetin as an anti-

oxidant and a signaling molecule inside the cell is felt to correlate directly with its ability to raise intracellular zinc levels.⁴⁹ A liposome-encapsulated form of zinc supplementation should also have this impact, although studies have not yet been performed that would document this conclusion.

Respiratory Virus Prevention and Treatment

In approaching the prevention and/or treatment of a respiratory tract viral infection, it is first appropriate to consider the factors that determine the likelihood of contracting such an infection. Factors critical to acquiring such an infection include the following:

- ✓ The concentration of viral particles in a given exposure
- ✓ The total amount (duration) of virus exposure
- ✓ The inherent strength of the defense mechanisms of the exposed individual, especially the overall immune capacity

Addressing all of these factors in concert will result in the most effective avoidance of a respiratory viral syndrome taking hold. While the degree to which mask-wearing is vital to prevention is a point of increasing contention, general sanitary measures and avoiding crowds are nevertheless common-sense ways that will always diminish the concentration and total amount factors of virus exposure.

Since complete avoidance of a significant virus exposure is difficult and even largely impossible to achieve during a pandemic with a highly contagious

pathogen, the best way to prevent such an infection is to strengthen and maintain a strong immune system. This is also the best protection against significant morbidity or even death resulting from an infection already contracted.

Effective Viral Therapies

Although it has become an accepted mantra in medicine, among both physicians and the public alike, that the common cold and influenza are incurable infections that simply must take their course, nothing could be further from the truth. The following therapies all have reliably cured these conditions (only a partial list):

- ✓ High-dose vitamin C (most reliably when given intravenously or taken orally in a quality liposome-encapsulated form)⁵⁰
- ✓ Ozone applications, especially autohemotherapy
- ✓ Ultraviolet blood irradiation
- ✓ Hydroxychloroquine and chloroquine, along with zinc supplementation
- ✓ HP applications, especially via nebulization or by vein

As a potent antiviral agent with a large and well-established track record in effectively curing a wide variety of viral syndromes, nothing comes close to vitamin C. Had it been given for as many different

infections and childhood illnesses as vitamin C, ozone would probably have been proven to be just as effective.

Vitamin C, by intravenous and/or oral administration has been documented in the scientific literature to cure the following viral infectious diseases:⁵¹

- ✓ Polio
- ✓ Acute viral hepatitis
- ✓ Measles
- ✓ Mumps
- ✓ Viral encephalitis
- ✓ Chickenpox
- ✓ Viral pneumonia
- ✓ Influenza⁵²
- ✓ Zika fever⁵³
- ✓ Chikungunya virus⁵⁴
- ✓ Ebola, as part of an ozone protocol⁵⁵

Not surprisingly, the most important factors in assuring an optimal and rapid cure of a given viral syndrome with vitamin C are:

1. Total dose,
2. Frequency of administration (keeping blood levels of vitamin C in a therapeutic range), and
3. Route of administration and form of vitamin C (intravenous, oral regular form, oral liposome-encapsulated form).

The importance of vitamin C in the treatment of any viral syndrome is further highlighted by the fact that viral infections rapidly consume whatever vitamin C is in the body. And since circulating vitamin C is rapidly excreted in the urine and is not significantly

stored anywhere in the body, variable degrees of acute scurvy can reliably be induced by an especially rapidly-replicating virus. The viral hemorrhagic fevers are caused by viruses that can replicate so rapidly that vitamin C is no longer detectable in the urine, a direct indication that very little vitamin C is left in the body. And it is precisely because of this accelerated consumption of vitamin C that bleeding complications are seen in the terminal stages of these viral infections. The body requires an ongoing presence of significant amounts of vitamin C to maintain vascular strength and integrity. The microvasculature rapidly loses its structural capacity to contain the blood, and the oozing/hemorrhage of a severe vitamin C depletion is a major reason for the eventual fatal outcome in such infections. Many Ebola viral infections result in a fatal outcome because of this severe induced vitamin C depletion throughout the body.

Of course, when one is already significantly ill with one of these viral syndromes, including COVID, there is no need to limit therapy to one therapeutic agent or intervention only. Indeed, there appears to be an increasing number of patients following COVID infection resolution that have some persistent symptoms, including malaise and loss of smell and taste (“long-haul COVID”). Whether this represents chronic virus-induced damage or is reflective of some persistent presence of the virus is not established. Furthermore, some patients continue to have difficulties with an excess coagulability of the blood and proneness to both venous and arterial blood clots.

There is evidence that the more clinically severe COVID patients have a greater chance of acquiring

a vasculitis (blood vessel inflammation), which has the potential to become chronic post-infection condition if measures are not taken to resolve the increased oxidative stress and damage that resulted from the

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acute infection.⁵⁶⁻⁵⁹ Because of this, an extended course of highly-dosed vitamin C should be administered post-infection after all acute symptoms and evidence of active COVID infection have disappeared. This would probably not be needed if high-dose vitamin C had been used at the outset to resolve an acute COVID infection, but any other therapy that could eradicate the virus without having any substantial intrinsic anti-oxidant capacity should be followed with a course of vitamin C for optimal long-term clinical outcome.

In early 2020, at the outset of the pandemic in the United States, the reported chances of severe clinical disease and even death from COVID were substantially greater than they were in late 2020. While a number of factors could account for the lessened morbidity and mortality secondary to this virus, one likely factor for this favorable change is that many more people began consuming supplemental vitamin C, as well as vitamin D, magnesium, zinc, and other supplements.

At about the same time that toilet tissue disappeared from the store shelves almost overnight (for whatever reason), bottles of supplemental vitamin C began to rapidly disappear as well. Furthermore, for the first time in history, many hospitals in the United States began initiating trials of intravenous vitamin C for the treatment of advanced COVID. Results were very positive in these trials, even though suboptimal doses of vitamin C were generally being administered (<http://orthomolecular.org/resources/omns/index.shtml>). The fact that these trials began taking place so early in the pandemic further enhanced the “reputation” of the important role of vitamin C in treating infections, which in turn highlighted the benefits of regular supplementation.

While there is no way to know for sure without extensive polling to research the issue, it would appear that more people in the United States are now taking regular oral vitamin and mineral supplements than ever before. Even older individuals who contracted COVID **late** in 2020 typically had only a few days of illness with very minimal symptoms. This is what would be anticipated when someone has been taking regular nutrient supplements such as vitamin C, vitamin D, magnesium, and zinc.

Cold, Flu, and Coronavirus Infection Prevention

This is a general guide only. While all of the listed supplements are recommended, any of them can be expected to provide some significant benefit by supporting good immune function.

- ✓ Vitamin C powder (sodium ascorbate or ascorbic acid): 1 to 2 grams three times

daily orally, or 1 to 3 grams daily orally of liposome-encapsulated vitamin C.

- ✓ Magnesium supplementation, at roughly a dose of 500 mg orally daily, given in divided doses (many forms available; chloride, glycinate, gluconate, and threonate are especially good forms).
 - Magnesium chloride is felt to be the optimal form for virus prevention/treatment.⁶⁰
 - Depending on age and body size, 10 to 50 cc orally twice daily of a 2.5% magnesium chloride solution (25 grams in 1,000 cc of water); tablets or capsules orally are also acceptable. Each dose can be further diluted in juice to optimize taste.
- ✓ Vitamin D (as vitamin D₃—cholecalciferol), 25,000 units daily for two weeks (if never previously supplemented); then 5,000 to 10,000 units daily.
- ✓ Zinc (as picolinate or citrate), 50 to 75 mg daily for two weeks (if never previously supplemented), then 25 mg daily.
- ✓ Iodine/iodide supplementation, 12.5 mg daily (available as Iodoral 12.5 mg tablets).
- ✓ Take, or continue to take, any other quality nutrient/antioxidant supplements that you desire and can afford to take.
- ✓ HP nebulization, 1 to 3 minutes daily, or at least when a significant virus exposure is suspected, utilizing a 3% or less concentration of the solution.

What are chronic pathogen colonizations (CPCs) and why are they a threat to my health?

Clinical Pathogen Presentations

Pathogens in the body generally present clinically in one of the following forms:

- ✓ Systemically present throughout the body, such as is seen with influenza, or with an infecting bacterial species as in sepsis
- ✓ Very focal and concentrated, commonly seen with infected teeth, gums, or tonsils, but present anywhere an abscess can develop
- ✓ Chronic pathogen colonization (CPC). This is an accumulation of pathogens that is not present throughout the body, but generally

extends over a larger area of tissue lining a space or cavity. The concentration of pathogens in a CPC site is also much lower than with an identifiable abscess.

Chronic Pathogen Colonization Presentations and Biofilms

Active growth of the pathogens in an area of CPC is limited, and the tendency of such areas to spread over larger areas of tissue is relatively limited. CPC is also characterized by the presence of overlying biofilms, an organized coat of extracellular polysaccharides that effectively protects those pathogens from any type of disruption. This biofilm barrier also completely blocks access by all known prescription antibiotics.¹⁻³ Although slow-growing, these CPC pathogens are metabolically active and therefore produce byproducts and exudates on a continual basis. Biofilms will be found not only in all CPC sites, but also in all sites of focal infection, even where abscesses have formed. Whenever pathogens are present in the body and not promptly killed and eliminated, biofilms will form in an effort to help the pathogens survive.

Virtually all chronic infections involve the formation of biofilms.⁴ Indeed, it is the formation of the biofilm that assures the chronicity of the infection. Areas of CPC and associated biofilms, are often involved in the etiology and development of the following conditions:

1. Esophageal cancer⁵
2. Gastric cancer⁶
3. Colorectal cancer⁷⁻⁹

4. Pancreatic cancer¹⁰
5. Lung cancer¹¹⁻¹²
6. Tongue cancer¹³

Areas of CPC, presumably with protective biofilms helping to sustain their presence, are also often seen throughout the body, existing in the tissues affected by various diseases or medical conditions, causing or significantly contributing to a specific pathology by maintaining focally increased levels of oxidative stress at those sites. Examples include:

- ✓ Coronary atherosclerotic plaques¹⁴⁻¹⁷
- ✓ Carotid atherosclerotic plaques¹⁸
- ✓ Blood clots causing acute myocardial infarctions^{19,20}
- ✓ Blood clots in the arterial and the venous system²¹
- ✓ Pericardial fluid of patients with chronic coronary artery disease²²
- ✓ Cerebral aneurysms²³
- ✓ Malignant breast tissue²⁴
- ✓ The affected brain tissue of patients with Alzheimer's disease²⁵⁻²⁷
- ✓ The synovial fluid and synovial tissue in patients with rheumatoid arthritis²⁸⁻³²
- ✓ The placentas of women prone to adverse pregnancy outcomes³³

While focal infections and abscesses, as well as systemic infections throughout the body, are well-

known manners of pathogen presentation, CPC remains little-recognized by physicians and other healthcare providers in mainstream medicine. And this is of particular importance, as CPC is ***much more***

The presence of these slow-growing sites of pathogens allows them and their toxins to be released and swallowed into the gastrointestinal tract “24/7.”

common than focal and systemic infections, as everybody has unknowingly developed CPC at some point in their lives. The vast majority of people never resolve it once it has developed unless very specific treatment measures are taken. Because of this, most people just live with it without ever realizing its existence, much less its impact on their general health. The negative health consequences of CPC end up being part of the chronically compromised health that is considered to be the “normal baseline” of so many people.

While it can potentially be found in any tissue or on virtually any cells lining an open space, cavity or hollow organ, the most clinically significant sites of CPC are largely limited to periodontal tissue, the mucosal surfaces of the sinuses and oral cavity, and even the epithelial cells lining the airways in the lungs. The presence of these slow-growing sites of pathogens allows them and their toxins to be released and swallowed into the gastrointestinal tract “24/7.” Of course, this substantially amplifies their clinical impact over whatever effects they might have locally, as the

intestinal microbiome becomes pathogen-laden and the gut becomes leaky. This then results in the new seeding of new sites of CPC throughout the body. More rapid proliferation of the pathogens will result in larger amounts of these pathogen/toxin mixtures sloughing off and getting swallowed.

Periodontitis has long been recognized as having an association and/or cause-and-effect relationship with a wide variety of diseases and medical conditions throughout the body. One of the most common periodontal pathogens in periodontitis that has been shown to play a prominent role in the development and evolution of many diseases is *Porphyromonas gingivalis*.³⁴⁻³⁵ This pathogen has also been implicated in the formation of bacterial plaque, which effectively acts as a **hardened equivalent of the pliable biofilms** that characterize CPC elsewhere on mucus membranes and in the tissues.³⁶

The Cause of All Chronic Disease?

When researchers have looked for a connection between particular diseases and an abnormal microbiome, such a correlation is frequently found and clear-cut in nature. Many of the tissues involved in various chronic diseases and conditions have been found to harbor the chronic presence of pathogens, typically of oral cavity origin. Furthermore, it is difficult to find any chronic medical condition not directly aggravated by chronic periodontitis. This demonstrates the powerful clinical impact associated with the body-wide spread of pathogens and toxins from this part of the oral cavity, as well as from the abnormal microbiome it has helped to generate and sustain.

There is a significant interrelationship between all of these pathogen-disease studies:

- ✓ The pathogens and toxins generated by oral cavity infections (gums, teeth, tonsils, sinuses) are continually swallowed as they are generated, promoting ongoing inflammation and oxidation in the gut. This both directly results in a loss of the intestinal cellular barrier integrity (leaky gut) while changing the microbiome into a less diverse, more pathogen-dominated flora. Once the microbiome has been altered in this fashion, it can then supply **even more** pathogens and pathogen-related toxins that can reach the rest of the body through the leaky gut.
- ✓ The literature documents that many of the tissues involved in different disease states colonize these pathogens from the leaky gut. Pathogens not gaining access to the rest of the body through the leaky gut can also spread via direct blood and lymphatic dissemination from the oral cavity infections mentioned directly above.
- ✓ The incredibly wide array of medical conditions associated with and aggravated by chronic periodontitis underscores the clinical significance of chronic pathogen exposure from any source.
- ✓ Chronic pathogen colonization of the nose, sinuses, throat, and lungs without grossly manifest infection further contributes to the chronic poisoning of the lower gut and

is **typically present without being clinically apparent.**

A great deal of indirect but compelling evidence of pathogen colonization in the affected tissues of many different diseases can be found in the literature. Specific examples include the following:

- ✓ In Alzheimer's disease and other forms of dementia, significant periodontitis was present in all of the patients in a study group. And even though periodontal pathogens were not found in the blood or cerebrospinal fluid, antibodies to those pathogens were found in the spinal fluid of a substantial number of those patients.³⁷
- ✓ Circulating inflammatory reaction agents to *Porphyromonas gingivalis* have been associated with Parkinson's disease.³⁸
- ✓ Circulating antibodies to periodontal pathogenic bacteria were found to be associated with a higher disease activity in systemic lupus erythematosus, including in juveniles with this disease.³⁹⁻⁴⁰
- ✓ Circulating antibodies to periodontal pathogens were found to be associated with increased cardiovascular disease mortality.⁴¹
- ✓ Circulating antibodies to the periodontal pathogen *Porphyromonas gingivalis* were associated with oral squamous cell carcinoma.⁴²

- ✓ Periodontal pathogens have been found in the placental tissue of women with adverse pregnancy outcomes.⁴³
- ✓ Circulating antibodies to periodontal pathogens were found to be associated with stroke.⁴⁴ Antibodies to a greater number of pathogens were independent predictors of unfavorable outcomes in ischemic stroke patients.⁴⁵
- ✓ Circulating antibodies to *Porphyromonas gingivalis* were associated with carotid intima-media thickness. Periodontal therapy decreased this thickening significantly, and this is regarded as a model of atherosclerosis reversal.⁴⁶
- ✓ Circulating antibodies to two periodontal pathogens were found to be associated with atherosclerosis at different sites in patients with rheumatoid arthritis.⁴⁷
- ✓ Circulating antibodies to periodontal pathogens were found to be associated with pancreatic cancer.⁴⁸
- ✓ Increased circulating antibodies to *Porphyromonas gingivalis* were found to be associated with an increased prevalence of heart failure.⁴⁹
- ✓ Circulating antibodies to *Porphyromonas gingivalis* were found to be associated with elevated liver enzymes.⁵⁰
- ✓ Circulating antibodies to *Porphyromonas gingivalis* were found to be associated with cerebral hemorrhage growth.⁵¹

All of the above studies show that periodontitis, with the eventual exposure of the periodontal pathogens to the systemic circulation, results in an immune response. This response often results in detectable antibodies to those pathogens. And this immune response is always reflected in a systemic inflammatory response. This chronic inflammatory response in turn predisposes the body to all diseases depending upon where the most inflammation gets situated and the most biomolecules end up oxidized. The measurement of the degree of this inflammatory response is very often reflected in elevated levels of C-reactive protein (CRP).⁵²⁻⁵³ Further support for the concept that periodontal pathogens are actually the **cause** of many diseases and not just “associated” with them is the observation that effective treatments of periodontitis frequently reduces the severity of the chronic disease under consideration.

What are biofilms and why do they make CPCs so hard to resolve?

Overview

Biofilms refer to collections of microbes that adhere to a surface and to each other in an encasement of an extracellular polymeric substance (EPS). This EPS is produced by the microbes themselves and consists of polysaccharides, proteins, and lipids, with further variations of content depending on the microbe and the microenvironment it which it forms.^{1,2} The microbes can adhere to living tissue or to non-biological surfaces, such as indwelling catheters or implanted joint prostheses. However, they can also exist as free clumps of bacteria bound together in an EPS. Bacteria-related biofilms are also facilitated in their formation by the release of extracellular DNA from the bacteria which binds with the polysaccharides and proteins to help give additional structural integrity to the EPS.^{3,4}

Up to 97% of a biofilm is composed of water and the rest is densely packed cells. Nutrients can flow

through the water portion of the encasing matrix. Some authors have asserted the water channels through the biofilm in a manner somewhat analogous to a circulatory system. Because of this, some feel that biofilm-coated microbe communities are akin to primitive multicellular organisms.⁵ This adaptation/evolution of groups of microbes could also indicate that the formation of biofilms is simply a natural protective mechanism to allow microbes to survive and even prosper in otherwise hostile environments.⁶ Certainly, once a biofilm-encased site of chronic pathogen colonization (CPC) has taken hold, the survival of those microbe colonies can be measured in years. Not only does CPC serve as a long-term reservoir for pathogens, it also permits the continued regeneration of pathogens and the long-term formation and release of pathogen-related toxins.⁷

Most of the biofilm literature addresses those biofilms that are generated by bacteria. However, many clinically important fungi, including *Candida*, *Aspergillus*, *Cryptococcus*, *Trichosporon*, *Coccidioides*, and *Pneumocystis*, produce biofilms as well.⁸ A virus (human T-cell leukemia virus type 1) has also been found to encase itself in a carbohydrate-rich matrix that bears a similarity to bacterial biofilms.^{9,10}

Bacterial biofilm formation can also be enhanced by the presence of an infecting virus.¹¹ Furthermore, many non-bacterial pathogens often colonize and replicate along with a bacterial pathogen, and they end up encased together in the associated bacterial biofilm. A combination of virus and fungus has been found to be entrapped together in the fungus-generated biofilm, and this has resulted in the virus being more resistant

to antiviral agents.¹² Bacterial biofilms are frequently polymicrobial, involving the containment of viruses, protozoa, and fungi along with the bacteria.¹³ This is the typical microbial community seen in the complex biofilm of dental plaque.¹⁴

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Biofilm Characteristics

Generally, bacteria exist in one of two forms during growth and proliferation. The planktonic, or free-floating, form is where the bacteria exist as single, independent cells, especially in the blood. The sufficient and rapid proliferation of this bacterial form can be expected to result in an acute infectious disease. The other form is known as the biofilm growth phenotype, where the bacterial proliferation is much slower and groups of pathogens stick together, resulting in the prompt formation of biofilms.¹⁵ Sometimes cells can become detached from a CPC-associated biofilm, and an acute systemic infection can result.¹⁶

All biofilms are not covering pathogens. Microbes that are beneficial or just non-pathogenic in nature can often exist in biofilm-protected microbial communities. However, when a microbe is a potent pathogen (CPC), the presence of the biofilm simply facilitates its prolonged survival, along with the ability to express

pathogen-related toxins or to break off in smaller aggregates to seed other areas of the body.

Aggregates of biofilm-protected bacteria are highly resistant to all known prescription antibiotics, and resolving many chronic infections with established biofilms remains a big challenge for practitioners of traditional medicine.^{17,18} The antibiotic resistance of biofilms involves multiple factors, including:

- ✓ Antibiotics are slowed in their rate of transit/transport across the biofilm
- ✓ Antibiotics can chemically react/combine with some of the EPS components.¹⁹
- ✓ The antibiotics are not helped by the immune system in dealing with biofilm-coated pathogens as much as with free circulating pathogens. The presence of the EPS severely limits the ability of macrophages (phagocytic immune cells containing very high concentrations of vitamin C) to penetrate through the biofilm and kill/digest the underlying pathogens. This impaired ability to reach such protected pathogens also hinders some of the immune response that is needed for eliminating the pathogens from the body.²⁰ The CPC biofilm also works to decrease the detection of, and response to, the pathogen-generated metabolic signaling factors needed to activate the pathogen-killing immune response.
- ✓ Biofilm-coated pathogens have substantially reduced growth rates over free pathogens. Antibiotics tend to work

much more effectively with rapidly replicating pathogens.

- ✓ Biofilms result in an oxygen-deprived microenvironment, which can further decrease the uptake and effectiveness of an antibiotic.
- ✓ Older biofilms are more resistant to treatment than newly-formed biofilms.

The resolution of an acute bacterial infection with appropriate and properly administered antibiotic therapy only eliminates the free, unattached bacteria in the blood, lymph, and extracellular fluid.²¹ However, following such clinical resolution that might appear to be a complete cure, various tissues in the body will often be left with residual areas of biofilm-protected CPC. When appropriate antibiotic treatment is administered early enough in the course of the infection, clinical resolution without any residual CPC could occur. However, when the acute bacterial infection remains unaddressed for a long enough time, areas of CPC can be expected to remain indefinitely even though the acute infection might appear completely cured. In other words,

Chronic infection assures the presence of biofilms.

While not all sites of chronic infection have been evaluated for the presence of biofilms, its presence has been documented or insinuated at in a wide array of chronic infections or sites of CPC. These areas of infection include the following:

- ✓ Periodontal tissues when inflamed and infected (periodontitis). The dental plaque that causes and sustains periodontitis is the prototype for biofilm, typically composed of a diverse microbial/pathogen community.²²
- ✓ Chronic otitis media (middle-ear infection) had mucosal biofilms in over 90% of the specimens taken at a surgical procedure visualized on laser scanning microscopy.²³
- ✓ CPC sites were found in the brain tissue of Alzheimer's disease patients.²⁴⁻²⁷
- ✓ Chronic wounds, such as diabetic foot ulcers, pressure ulcers in immobile patients, and venous leg ulcers, typically heal very slowly and sometimes not at all. Scanning electron microscopy found biofilms in a majority (30 of 50) of chronic wounds examined. Biofilm was detected in only 1 of 16 acute wounds.²⁸
- ✓ Tonsil tissue surfaces harbor biofilms, often in children with chronic otitis media. Adenoidectomy has been shown to be effective in the treatment of chronic otitis media, indicating the adenoids may also serve as reservoirs for the otitis-related pathogens.^{29,30} Infected tonsils have biofilms internally and on external surfaces.
- ✓ In bacterial endocarditis, the primary infection is a biofilm-protected growth of bacterial pathogens on a cardiac valve. This also allows for biofilm pieces to periodically break off (embolize) and seed a

new site of infection via the arterial system to another area of the body.³¹

- ✓ In atherosclerosis, or hardening of the arteries, bacterial biofilms have been identified by microscopy. It is logically suggested that such biofilm not only promotes the evolution of atherosclerosis, but might also contribute to an enhanced risk of plaque rupture.³²⁻³³
- ✓ In the salivary glands, calcified stones can develop and the gland can become infected. Such infections often have biofilms, and healthy control glands demonstrate no biofilm.^{34,35} It is hypothesized by some that biofilm formation actually initiates such glandular stone formation.^{36,37} If true, biofilm may well be a ***foundational etiology*** for stone formation in other organs and tissues throughout the body. This would further underscore how significant CPC can be in the development of other medical conditions associated with calcifications.
- ✓ Biofilms appear to form on gallstones, particularly in the presence of *Salmonella* infections causing typhoid fever.³⁸ This type of bacteria readily develops biofilms *in vitro*.³⁹⁻⁴⁰
- ✓ Inflammatory bowel disease involves an adherence of pathogenic bacteria (biofilm) to the mucosal lining of the bowel. The more advanced the bowel disease, the more and greater the biofilm mass.⁴¹⁻⁴³

- ✓ Substantial evidence now indicates that biofilms promote colorectal carcinogenesis at its early stages.⁴⁴
- ✓ Bacterial vaginosis, a common relapsing genital tract infection in women, features a polymicrobial biofilm primarily composed of clusters of a type of anaerobic bacteria, *Gardnerella vaginalis*.⁴⁵⁻⁴⁶
- ✓ The *Gardnerella vaginalis* biofilm seen in bacterial vaginosis frequently extends into the endometrium and even the fallopian tubes.⁴⁷
- ✓ Chronic sinus infections can harbor biofilm-coated pathogens, often coating viral presences as well.⁴⁸ In one study, a group of tested control patients who were operated for deviated septum showed no biofilm presence.⁴⁹
- ✓ Chronic laryngitis often has detectable biofilms.^{50,51}
- ✓ Many of the pathogens involved in the chronic lung infections seen in cystic fibrosis patients are biofilm-protected areas of CPC.⁵²⁻⁵⁵
- ✓ **Intracellular** bacterial biofilms have been described in patients with chronic urinary tract infections.⁵⁶ The presence of biofilms inside of infected cells has only really been significantly addressed for chronic urinary infections, and it is unclear how common intracellular biofilms might be in the cells of tissues affected by the

extracellular CPC noted above in many different disease states.

- ✓ The development of chronic bacterial prostatitis has been suggested by multiple researchers to be often caused by biofilm-protected pathogens.⁵⁷⁻⁶⁰

Furthermore,

When laboratory testing indicates an infecting pathogen is highly sensitive to a given antibiotic, and the appropriate administration of that antibiotic does not result in a prompt resolution of an infection or infectious disease, it can reliably be assumed that much of the pathogen load is already biofilm-protected.

The actual amount of time it takes for an acute infection to start coating sites of CPC with biofilm is not clearly established. However, one animal model study clearly showed the presence of biofilm by at least 48 hours after bacterial inoculation (*Staphylococcus aureus*) into a wound.⁶¹ Specimens less than 48 hours after inoculation were not tested. Multiple factors could make this a significantly different period of time for different patients. However, the biofilm literature would indicate that it does not take long for biofilms to start forming as foci of pathogens accumulate on different tissue surfaces or on the surfaces of implanted prosthetic devices. Sex and thyroid hormone status and inherent immune capacity, along with the adequacy of vitamin C, magnesium, zinc, and vitamin D levels would all be factors that would strongly impact how

quickly and tenaciously biofilms might take hold for a certain type of infection in one patient versus another. Clinically, it has been shown that oral bacteria-seeded plaques in the coronary arteries stabilize dramatically and lose much of their ability to block off those arteries and cause heart attacks when thyroid function is adjusted and maintained optimally.⁶² And this stabilization will often occur when the infected teeth, tonsils, or gums are left alone and **never addressed clinically**. Perfect thyroid hormone status, along with optimal sex hormone status, are **extremely** critical in keeping focal infections focal when they are not otherwise resolved.

The major practical clinical takeaway point from all of this information on infection, CPC, and biofilms is straightforward:

**The complete resolution of any state
of infection or infectious disease
MUST include agents known to destroy
biofilm as well as to penetrate deeply
enough into the involved tissues to
exert their impact on the “islands”
of CPC throughout the body.**

All effective agents for resolving biofilm-protected pathogens are natural substances, unmodified by a laboratory. Discussed in more detail in Chapter 14, such agents are optimal when they can achieve the following clinical goals:

- ✓ Prevention of biofilm formation,
- ✓ Dissolution of existing biofilm, and
- ✓ Killing of exposed pathogens.

Some agents accomplish all of these goals, while others are less broad-based in achieving all three prongs of CPC eradication. Some of the most effective natural therapeutic agents for preventing and/or eliminating CPC include the following, applied in ***one or more*** modalities, and in any of a number of combinations:

Agent Formation	Prevents Biofilm Formation	Dissolves Pathogens	Kills/Inactivates Pathogens
Hydrogen Peroxide	YES ⁶³	YES ⁶⁴⁻⁶⁶	YES ^{67,68}
Ozone	YES ⁶⁹	YES ^{70,71}	YES ⁷²
Vitamin C	YES ⁷³⁻⁷⁵	YES ^{76,77}	YES ⁷⁸
Magnesium	YES ⁷⁹		
DMSO (dimethyl sulfoxide)	YES ⁸⁰	YES ⁸¹	Anti-Pathogen Activity ^{82,83}
Antioxidants (in general)	YES ^{84,85}	Possibly	In varying ⁸⁶ degrees
Zinc	YES ⁸⁷⁻⁸⁹		
Vitamin E	YES ⁹⁰		
Iodine		Penetration of biofilms in wound treatment ⁹¹	

Recap

Biofilms are clusters of microbes, often pathogenic bacteria, encased in a protective matrix that they produce themselves when enough of them have

had a chance to gather on a tissue surface in the body. Generally, it is the presence of this biofilm that not only makes various diseases more severe and difficult to treat, it also permits the long-term survival of pathogens in the body.

Acute infections that are treatable by agents such as antibiotics can rapidly become refractory to such treatment if they are not administered promptly and appropriately. This is due to the ability of many pathogens to form protective biofilms after they accumulate on tissue surfaces throughout the body. Virtually all chronic infections are chronic **because** biofilm formation quickly forms and protects the involved pathogens from being eradicated.

The effective treatment of ***all chronic diseases*** is optimized by taking measures to eradicate sites of CPC in the tissues of the body that are presumed to be present when identifiable sites of focal infection and CPC, usually in the aerodigestive tract, have been present for a long enough period of time. Eradication of CPC outside of the gastrointestinal tract will only be optimized when the ongoing sources of infection seeding and re-seeding in the oral cavity and gastrointestinal tract are first eradicated or contained as completely as possible.

How do oral and nasal CPCs create and exacerbate chronic disease?

Although CPC infections can exist throughout the body in the cells lining hollow organs, cavities, and even open spaces, the CPCs that result in the most clinically significant health issues are generally found on the mucosal and epithelial surfaces of the oral cavity, sinuses, pharyngeal areas, and the lungs. The location of these CPCs above the beginning of the gastrointestinal tract allows their harbored pathogens and toxins to continually drain into the gut. Depending of the health and diversity of the “bugs” in the gut and the integrity of the gut lining, some of these pathogens/toxins will also enter the lymphatics and bloodstream, eventually infecting various systems and organs throughout the body.

Periodontitis also disseminates a large variety of pathogens/toxins into the GI tract as well as directly into the bloodstream. This chronic oral infection (a

classic form of CPC) has long been associated with a wide variety of diseases and medical conditions throughout the body. As mentioned earlier, one of the most common and aggressive of periodontal patho-

Many authorities consider periodontal disease to be the **most prevalent infectious disease in the world.**

gens, *Porphyromonas gingivalis*, is known to have a significant part in the development and evolution of many diseases.^{1,2} This pathogen also been implicated as having a prominent role in the formation of dental plaque. The hard protective barrier seen with dental plaque is largely just a more rigid and calcified version (calculus) of the pliable biofilms that characterize CPC elsewhere on mucus membranes and other cellular surfaces.³

Even with mild chronic inflammation of the gum and periodontal tissue, CPC will already have formed, and the hardened plaque seen with advanced periodontitis just represents an advanced form of CPC. Of note, periodontitis is an extremely common condition, as are the many diseases with which it has been documented to be associated. The negative health impact of smoking is largely due to the variable degrees of periodontitis that are ALWAYS present in such individuals.

It should always be expected that CPC in the throat and lungs will be present upon the clinical resolution of a cold or flu as a result of the infecting pathogens. The development of those throat and lung areas of

CPC are often hastened and exacerbated by an ongoing exposure to the pathogens and toxins from preexisting CPC from periodontitis, focal infections, and other sites in the aerodigestive tract.

Without really being recognized as a common site of CPC that continually assaults the gut with new pathogen and toxin exposures with every swallow, an enormous amount of literature has already been generated showing that periodontitis is associated with, and often shown to have a ***cause-and-effect*** relationship with, diseases and medical conditions throughout the body. It would appear that the effect of periodontitis on the health status of the gut and microbiome is also a primary reason why this “local” condition in the mouth so dramatically impacts health in all areas of the body.

Periodontitis, including just mild gum disease (gingivitis), is very prevalent in the adult population.⁴ Many authorities consider periodontal disease to be the ***most prevalent infectious disease in the world.***⁵ Consistent with this high prevalence, the association of so many different diseases and medical

Of note, periodontitis is an extremely common condition, as are the many diseases with which it has been documented to be associated.

conditions with periodontitis makes a great deal of sense, as the chronic seeding of pathogens and toxins into the tissues throughout the body would be expected to do nothing but harm. Not surprisingly, the following

list of medical conditions associated with periodontitis appears largely the same as the list of conditions noted to be associated with an abnormal gut microbiome. Pathogens and toxins, from wherever they arise, clearly cause many diseases and conditions, although the literature largely maintains that only “associations or correlations” can be confirmed, and only the possibility of cause-and-effect relationships exist, which would often involve unethical research to be performed for definitive proof. Medical conditions found to be associated with periodontitis include the following:

- ✓ Increased all-cause mortality⁶
- ✓ Cardiovascular diseases⁷
 1. Hypertension⁸⁻¹⁰
 2. Atherosclerosis and coronary heart disease^{11,12}
 3. Acute myocardial infarction¹³
 4. Heart failure¹⁴
 5. Metabolic syndrome and lipid abnormalities^{15,16}
 6. Arterial calcification¹⁷
- ✓ Cerebrovascular disease and stroke¹⁸⁻²⁴
- ✓ Intracranial aneurysms^{25,26}
- ✓ Brain abscess²⁷
- ✓ General vascular disease^{28,29}
- ✓ Diabetes^{30,31}
- ✓ Obesity^{32,33}
- ✓ Pulmonary inflammation and infection³⁴⁻³⁷
- ✓ Asthma^{38,39}
- ✓ Inflammatory bowel disease⁴⁰
- ✓ Migraine⁴¹
- ✓ Alzheimer’s disease^{42,43}

- ✓ Memory loss and neuroinflammation in mice⁴⁴
- ✓ Parkinson's disease⁴⁵
- ✓ Seizure disorders⁴⁶
- ✓ Depression⁴⁷
- ✓ Bipolar disorder⁴⁸
- ✓ Dementia^{49,50}
- ✓ Rheumatoid arthritis^{51,52}
- ✓ Ankylosing spondylitis⁵³
- ✓ Juvenile idiopathic arthritis⁵⁴
- ✓ Osteoarthritis⁵⁵
- ✓ Osteoporosis^{56,57}
- ✓ Liver cirrhosis⁵⁸
- ✓ Cancer
 - 1. Pancreatic^{59,60}
 - 2. Lung⁶¹
 - 3. Liver⁶²
 - 4. Colorectal^{63,64}
 - 5. Esophageal⁶⁵
 - 6. Oral cavity and throat (aerodigestive tract)⁶⁶⁻⁶⁸
 - 7. Head and neck⁶⁹
 - 8. Gastric cancer⁷⁰
 - 9. Prostate, blood, and skin cancers⁷¹
 - 10. Cancer in general⁷²
- ✓ Adverse pregnancy outcomes^{73,74}
- ✓ Anemia^{75,76}
- ✓ Hypothyroidism⁷⁶
- ✓ Eye diseases (inflammation and infection)⁷⁸
- ✓ Glaucoma⁷⁹
- ✓ Loss of hearing⁸⁰
- ✓ Psoriasis⁸¹

- ✓ Polycystic ovary syndrome⁸²
- ✓ Erectile dysfunction⁸³
- ✓ Female infertility-associated conditions⁸⁴
- ✓ Chronic kidney disease⁸⁵⁻⁸⁷
- ✓ Autoimmune diseases⁸⁸
- ✓ Systemic lupus erythematosus^{89,90}
- ✓ Multiple sclerosis⁹¹
- ✓ Elevated C-reactive protein levels⁹²⁻⁹⁴
- ✓ Depressed vitamin D levels⁹⁵
- ✓ Depressed vitamin C levels⁹⁶

The presence of aerodigestive tract CPC also sets the stage for contracting a new acute respiratory infection (typically viral). Any cells lining the nose and throat that are already coping with CPC are much more susceptible to succumbing to a new pathogen compared to areas free of CPC. Furthermore, areas of CPC can more readily re-manifest (“flare up”) as a new acute infection when the body has been subjected to a sufficient immune challenge. This is another good reason for undertaking a regular program of periodic therapeutic nebulizations even when no symptoms are present, as keeping the throat and airways completely free from CPC greatly reduces the susceptibility of contracting a new respiratory infection.

A CPC site with an especially high concentration of viral, bacterial, and/or fungal pathogens and associated toxins is the tongue⁹⁷ Chronically pathogen-colonized tongues are very common, and they are especially well-protected with biofilms. The anatomy of the tongue makes it an ideal site for pathogens to get trapped along with food particles supporting their growth. The tongue is not a smooth surface at the

microscopic level. Rather, there are many fissures and tiny papillae (finger-like projections) literally waiting to trap pathogens, food, mucosal secretions, and saliva.

Also, once “trapped” in a thick enough CPC biofilm on the tongue, many of the otherwise innocuous

...keeping the throat and airways completely free from CPC greatly reduces the susceptibility of contracting a new respiratory infection.

aerobic bacteria find themselves in an anaerobic environment, triggering the production of enormously potent toxins, just as are found in root canal-treated teeth and other infected teeth.^{98,99} Also, CPC in one area reliably seeds CPC in another area. Most individuals with CPC anywhere else in the alimentary tract will be found to have an abnormal tongue flora.

Just as any other part of your body can become soiled and require cleaning, the same is even more true for the tongue, with its many pathogen- and debris-trapping nooks and crannies. You cannot garden with your bare hands and expect them to clean themselves of the dirt and soil remaining on your hands and even under your nails. Unless you want them to be dirty “for life” you need to wash them. The same is true of your tongue. It must be cleaned regularly, at least once a day for optimal benefit. Additional cleaning would be required after a thick creamy soup versus, say, a garden salad. Use common sense to decide when cleaning is indicated.

In some people, the lack of attention to tongue hygiene can lead to a coated tongue, resulting in a chronic whiteness of the tongue. This occurs when pathogens are trapped with putrid food debris, along with dead tongue cells that have not been able to shed or be exfoliated. This can also result in variable degrees of tongue inflammation, in which a chronic swelling or hypertrophy of the tongue papillae results from the trapped waste.

Another similar tongue condition is known as geographic tongue. While largely regarded as a “harmless” condition, it nevertheless involves variable degrees of inflammation of the tongue, with an array of well-circumscribed smooth and rough areas on the surface of the tongue. The more technical name for geographic tongue is benign migratory glossitis. The suffix “-itis” always means inflammation.

Whether your tongue appears coated, inflamed in patches, or just soiled and stained with your previous few meals or drinks, specific measures need to be taken to clean it. While some doctors regard these tongue conditions as being of no significant consequence, nothing could be further from the truth. Individuals with the thickest and most readily apparent tongue coatings have actually been shown to have significantly higher CRP (C-reactive protein) levels than patients with thinner coatings.¹⁰⁰ As increased CRP levels reflect increased body-wide levels of oxidative stress, it appears to be clear-cut that a well-contained and “localized” area of significant CPC, as with a coated tongue that remains unaddressed, can chronically increase oxidative stress throughout the body.

Not surprisingly, the effective cleaning of the tongue along with the maintenance of a clean tongue, resolves many cases of persistent bad breath, or halitosis.^{101,102} The metabolism of pathogens, especially

Just as any other part of your body can become soiled and require cleaning, the same is even more true for the tongue, with its many pathogen- and debris-trapping nooks and crannies.

when trapped anaerobically with food particles and other secretions, readily produces the bad odors involved in many cases of foul breath. Fortunately, the metabolism of normal microbial flora does not do the same thing. When chronic periodontal disease is present, it is the pathogens involved in this condition that are often the prominent sources of the halitosis-producing bacteria colonizing the tongue.^{103,104}

Tongue cleaning is quite straightforward. Brushing the tongue as thoroughly as possible when brushing the teeth is one option. However, this is probably better for maintaining good tongue hygiene rather than for trying to restore a chronically coated and even inflamed tongue back to normal. An optimal approach to maintaining a healthy and clean tongue includes the following:

- ✓ Use any commercially available tongue scraper to mechanically remove as much biofilm, pathogen, and pathogen-related debris as possible. While it is a very

simple procedure, there are many YouTube videos that demonstrate how to do this for anyone feeling uncertain how to proceed. This scraping should not cause discomfort. If it does, a lighter scraping of the tongue can be achieved with a toothbrush. If this is still not well-tolerated, swishing 3% HP (not swallowing) in the mouth should be done several times a day for a week or so to quell any prominent inflammation, and then tongue scraping can be carefully reinitiated.

- ✓ The rate of reformation of tongue coatings indicates that daily cleaning is indicated.¹⁰⁵ Optimally, a few seconds should be taken to clean the tongue after every meal. However, twice a day also works well (first thing in the morning, last thing at night). If it is only done once day, then doing it directly before going to bed at night would be the best time.
- ✓ When scraping the tongue along with brushing the teeth, it is probably best to scrape before brushing to optimize breath-freshening impact unless a mouthwash swish is part of the routine.
- ✓ For general gum care, utilize an oral water irrigation device. This is more effective at removing retained food particles than flossing, and it cleans away small particulates that flossing simply cannot reach. Furthermore, a suboptimal flossing technique can damage the gums while removing larger food particles. When the water irrigation tank has 10 to 15 cc of 3% HP added to about a cupful of water, gums are much easier to

maintain in optimal health, and the overall reservoir of pathogens in the oral cavity is greatly minimized.

- ✓ When water irrigation is initiated in a mouth with advanced periodontitis, bleeding is to be expected, even at the lowest pressure levels. This typically dissipates over time, and most individuals can work their way up to maximal pressure levels in several weeks to a month or so. This is reliably reflected in healthier-appearing gums along with a substantial reduction in the amount of infection between the teeth and gum tissue detectable upon probing by your dentist. Optimally, irrigation should be done after each meal to restore and preserve gum health.

Along with maintaining good tongue hygiene, a regular program of nebulization with any of a variety of pathogen-eradicating agents should be undertaken as well for any CPC in the nose and throat. While not needing to be done on a daily basis when there is no reason to believe a condition of CPC is present, one should not hesitate to nebulize for a few minutes with an appropriate agent to help protect against a potential seeding of new pathogens after a suspected or possible exposure via the respiratory route.

CPC is responsible for the following consequences:

- ✓ Prevents a normal microbial flora from colonizing the nose, mouth, and throat
- ✓ Facilitates the contraction of a new acute respiratory infection
- ✓ Promotes chronic sinus infections

- ✓ Promotes chronic pulmonary infections and diseases
- ✓ Can promote carcinogenesis in the lungs
- ✓ Promotes chronic halitosis and chronic inflammatory tongue conditions
- ✓ Permits the continual swallowing of pathogens and pathogen-related toxins, which promotes:
 1. A chronic inflammation of the intestinal epithelial cells lining the gut, resulting in a chronic leaky gut syndrome
 2. A less diverse and more pathogen-predominant microbiome in the gut
 3. The generation of even more pathogens and toxins from the altered microbiome to further sustain the gut inflammation and to provide an even greater variety of pathogens and toxins to “leak” through the intestinal barrier and disseminate throughout the body
 4. The development and maintenance of nearly all chronic diseases by the feeding of body-wide oxidative stress from the leaked pathogens and toxins
 5. The development of CPC with associated biofilms in different organs and tissues throughout the body by the seeding of pathogens gaining access through the leaky gut

How can I interrupt, minimize, and even eliminate CPCs?

The foremost intent of this book is to show that HP nebulization and other clinical interventions will completely stop the pandemic dead in its tracks. Yet perhaps there is another lesson of greater long-term benefit. It now appears that there are clear-cut ways to lessen and **even eliminate** some chronic diseases felt not only to be incurable, but also clinically unimprovable. This is due to the fact that the primary and secondary effects of chronic pathogen colonization (CPC) and chronic focal infections in the oral cavity and aerodigestive tract are the reasons for many diseases starting in the body, as well as for all diseases worsening or not readily responding to different therapies.

Ironically, the most important of the interventions needed to eliminate

CPC and minimize its effects on the microbiome of the gut, which then lessens the dissemination of pathogens and toxins through a leaky gut is the HP nebulization.

At the same time HP nebulization is knocking out your cold, flu, or COVID, it is substantially (sometimes completely) **resetting your gut flora back to normal**, and greatly helping to restore overall gut function to normal as well. This “gut reset” allows the leaky gut to heal and thereby prevent easy access of pathogens and toxins to organs and structures elsewhere in the body. As this healing takes place, **ALL** chronic diseases come under better control, and a few of them may even completely resolve clinically if not present for too long a period of time already.

Chronic Pathogen Colonization (CPC)

Discussed in Chapter 4, the bulk of CPC in the body is located in the sinuses, nose, mouth and throat, airways, and esophagus (aerodigestive tract). A much lower, but chronically present, pathogen load is found in the tissues involved in different diseases throughout the body. Once the primary seeding sources of pathogens in focal infections and aerodigestive tract CPC have been effectively addressed or at least minimized as completely as possible, it is very important to normalize the microbial flora in these areas as completely as possible. Additional measures to help achieve this are presented in Chapter 14.

In some individuals, however, just completely eliminating the pathogens of focal infection and aerodigestive CPC goes a long way in allowing the tissues of the

body to finally starting resolving the CPC elsewhere in the body since they are no longer being reseeded “24/7”. Certain individuals with less advanced chronic diseases can show a complete clinical resolution of

At the same time HP nebulization is knocking out your cold, flu, or COVID, it is substantially (sometimes completely) ***resetting your gut flora back to normal.***

their medical conditions by just eliminating these sources of pathogen replication and stopping their ability to seed new sites of CPC while sustaining old sites. As always, the input of the integrative physician in conjunction with the biological dentist is essential in determining the optimal treatment protocol and long-term maintenance plan for such patients.

In the previously CPC-free aerodigestive tract, but after significant respiratory pathogen exposures have occurred, it is always appropriate to take the same measures for either keeping a cold or flu from taking hold or for treating it promptly after any characteristic symptoms occur. This is because CPC primarily appears and indefinitely thrives from residual pathogen colonization remaining after the acute and more clinically severe cold or flu has run its course. This residual colonization is not the exception but the ***rule***. And it should always be considered as being present in all or part of the aerodigestive tract after a cold or flu has clinically resolved, no matter how long ago that infec-

tion occurred, if no specific measures have been taken to eradicate it.

**Anti-pathogen nebulization for
pre-existing CPC is the most important
part of any chronic disease resolution
protocol, in addition to being essential
for the prevention and/or resolution
of any acute respiratory infection.**

As mentioned, HP nebulization is the single best agent and even monotherapy for dealing with acute respiratory infections or for dealing with pre-existing CPC in the aerodigestive tract. Individuals who nebulize for a cold, flu, or COVID infection are simultaneously “mopping up” much of the pre-existing CPC along with the acutely-contracted pathogen. As a result, the clinical perception of a rapidly improving sense of well-being is due to both the eradication of the acute bug along with the resolution of biofilm-protected pathogens that could have been present for years.

**Many such individuals come to realize
for the first time in years what a true
sense of optimal health can feel like after
properly-administered HP nebulization.**

In addition to HP, many other agents can be nebulized with good anti-pathogen impact and lack of toxicity. These include, but are not limited to: DMSO, vitamin C, magnesium chloride, zinc chloride, nascent iodine, sodium bicarbonate, N-acetylcysteine, and both normal and hypertonic saline. Depending on the goal of the nebulization protocol, insulin and/or hydrocortisone can be added as well. Both of these agents

work effectively to increase the intracellular uptake of vitamin C and magnesium, which is the primary physiological goal in normalizing the cells involved in any disease or infectious condition.

Individuals who nebulize for a cold, flu, or COVID infection are simultaneously “mopping up” much of the pre-existing CPC along with the acutely-contracted pathogen.

Furthermore, modern medicine has long been centered on giving therapeutic agents systemically. That means the entire body is exposed to the drug with the hope that the agent will eventually arrive where it is actually needed (the target tissue) in sufficient concentration. Nebulization offers the ability to deliver higher concentrations of drugs and nutrients to diseased or infected lung tissues than can usually be achieved with oral or parenteral administration. In some situations, it also allows therapeutic levels of drugs to disseminate throughout the body while remaining well below potentially toxic degrees of drug intake.

In dealing with acute respiratory infections and the prevention/resolution of CPC in the aerodigestive tract, many of the agents noted above can either be added to HP nebulization or nebulized following the HP nebulization for the purposes of healing the tissue after pathogen elimination. They can also soothe any possible irritation/inflammation that might have been

caused by extending HP nebulization too far beyond pathogen resolution. While exceptionally nontoxic, too much HP exposure does have a pro-oxidant impact and can manifest as nasal burning, irritation, runny nose, or sneezing, possibly with a minimally sore throat and a tendency to a non-productive cough.

Much lower concentrations of HP in the nebulization solutions can almost completely avoid such effects, but the closer to a 3% concentration in the solution, the much more rapid will be the pathogen kill and the clinical recovery.

The selection of HP concentration all depends on how sick someone is, and whether a few additional days of illness is really worth avoiding any of the minor side effects noted above. It should also be noted that very many acutely ill and heavily infected individuals who nebulize 3% HP solutions experience no irritation at all and even find the 3% concentration very easy to inhale and very calming on the ability to breathe easily. HP nebulization also offers an easy way to quickly improve blood oxygenation, which will not be nearly as pronounced with the nebulization of substantially lower HP concentrations.

The nebulization of 3% HP rapidly improves blood oxygenation, and it can readily serve as an emergency way to deliver oxygen to a critically ill patient when oxygen administration by mask or nasal cannula is not available.

Nebulization can be performed with a large, desktop jet nebulizer or a small handheld mesh nebu-

lizer. Both are effective, but the handheld version is very quiet and can deliver a finer, deeper-penetrating mist, which is especially important for dealing with infections already deep in the lungs. The jet nebulizer is quite noisy, but also very inexpensive and durable. It

[HP nebulization] Properly applied, it is also a simple way to help achieve and maintain a normal flora in the aerodigestive tract.

is also better suited for aerosolization of various multi-solute and more concentrated solutions in the nebulization chamber that is connected to the air compressor. The mesh nebulizer will be much more prone to getting clogged with such solutions, and much more attention would be needed for prompt and thorough cleaning after misting such solutions. But when using the mesh nebulizer to mist HP only a few moments of nebulizing water after the nebulization session will suffice to clean and maintain the unit. It is actually a good investment to have both units available for use.

While there is no etched-in-stone protocol for optimal HP nebulization, Chapter 11 provides some guidance for implementing a program of such nebulization. Adhering to such a program of HP nebulization effectively prevents and treats acute respiratory tract infections. Properly applied, it is also a simple way to help achieve and maintain a normal flora in the aerodigestive tract. This in turn can potentially completely normalize gut function and keep it normal.

What is “Leaky Gut” Syndrome?

Introduction

Leaky gut syndrome is a condition affecting the cells lining the intestines. There is a loss of the integrity of the connection between these cells, induced by chronic oxidative stress. This results in a loss of barrier function of these intestinal cells that normally prevents pathogens, antigens, and various toxins from bypassing their normal processing inside the intestinal cells, instead gaining direct access via the spaces between the cells into the lymphatic and circulatory systems.¹ Normally, the small intestine has structures known as epithelial tight junctions which play the major role in determining what molecules resulting from digestion are absorbed. In general, the smaller molecules can pass and the larger ones cannot.

Variable degrees of barrier function are lost in a number of diseases known to feature the leaky gut syndrome as part of their pathology. The diseases and medical conditions that involve a gut with a loss of this

protective barrier function against larger molecules include the following:

- ✓ Irritable bowel syndrome
- ✓ Inflammatory bowel disease
- ✓ Crohn's disease
- ✓ Celiac disease
- ✓ Food allergies and sensitivities
- ✓ Most autoimmune disorders, including lupus, multiple sclerosis, and rheumatoid arthritis

A major problem in trying to clinically resolve what is now commonly known as the leaky gut syndrome is that it still remains unappreciated as to why most affected patients have this condition in the first place. The diseases listed above, along with many others, can be considered to be resulting directly from some degree of loss of the intestinal barrier function needed to prevent pathogens and large, improperly digested molecules from gaining access to the rest of the body.

Quite simply, the intestine works well until it is overwhelmed with oxidative stress. In this regard, it is like any other tissue or organ in the body: too much oxidation, typically ongoing, results in chronic disease. In the case of the intestine, too much oxidation directly damages the barrier function discussed above. And when the source of the oxidative stress is ongoing and literally taking place “24/7”, the intestine has no capability of healing and regaining the barrier function that was lost.

The ongoing oxidative stress sustaining the conditions noted above by keeping the intestine in a chronically damaged state comes from the continuous

swallowing of pathogens and the toxins associated with those pathogens. When there is no ongoing ingestion of pathogens and toxins, the gut (except in the rarest of exceptions) will remain or revert to normal. And when

Minor degrees of leaky gut can disappear in days, and severely damaged intestinal barrier function can still completely heal over a span of weeks to months...

the continuous ingestion of toxins is stopped, even a severely leaky gut will often normalize completely. Minor degrees of leaky gut can disappear in days, and severely damaged intestinal barrier function can still completely heal over a span of weeks to months if ongoing chronic oxidative stress can be completely prevented.

Is gluten toxic?

In your circulation, yes. Inside your gut, no. Gluten is just another protein that presents no digestive obstacle in a normally functioning gut to being broken down to its constituent amino acids and then absorbed normally. The same is true of peanut-derived protein and any of the many antigenic (immune-provoking) proteins involved in many of the different food allergies. Some protein fragments are simply much more antigenic in nature than others. However, when they are properly digested and the barrier function of the intestine is intact, the constituent amino acids only provide nutrition. When the intestinal barrier

function can be completely restored, even a previously “gluten-sensitive” individual can consume gluten with no concerns.

There are many individuals who have genetic predispositions to diseases of the gut,²⁻⁵ nearly all involving the integrity of the intestinal barrier. However, if the chronic swallowing of oxidizing agents can be avoided, many such individuals will never manifest the disease to which they are genetically prone. And for those individuals who more readily manifest their disease symptoms, the complete elimination of swallowed toxins can still make even a genetic deficiency disease much more manageable and less symptomatic.

Restoring Normal Gut Function

The ability of the body to heal can be amazing. Anecdotally, the manifestations of irritable bowel syndrome and inflammatory bowel disease have virtually disappeared in days to weeks in some individuals when enough swallowing of toxins was avoided. Other unpublished reports have indicated that bowel function, at least in terms of being firm and well-formed, can often result from only a few days of HP nebulization. Such a clinical response certainly strongly suggests that very many people have significant areas of CPC chronically poisoning their intestines and their normalcy of bowel function. Properly performed, HP nebulization quickly breaks down biofilm, eradicates the underlying pathogens, and spares the microbiome enough ingested pathogens to normalize enough so that the stool composition can subsequently normalize as well. While this does not mean a leaky gut has

completely healed, it does indicate that improvement in the barrier function of the intestine can be anticipated as well, and that there is a good chance that the gut can eventually heal completely.

As discussed earlier, CPC eradication in the aerodi-

When the intestinal barrier function can be completely restored, even a previously “gluten-sensitive” individual can consume gluten with no concerns.

gestive tract is the necessary first step to eradicating CPC everywhere else in the body and achieving an optimal clinical improvement with any chronic disease. But when it is effective in completing normalizing the gut microbiome and in healing any leaky gut that might have been present, major improvements in nearly all chronic diseases can be anticipated.

While there are multiple intersecting factors involved, the most straightforward approach to reaching optimal health is as follows:

- ✓ Nebulization with HP or other biofilm-busting, anti-pathogen agents
- ✓ Resolution of focal infections in and around the oral cavity (periodontitis, infected teeth, infected tonsils)
- ✓ Maintenance measures to keep CPC from recurring in the aerodigestive tract
- ✓ Avoidance of other ingested toxins (for example, iron)

- ✓ Measures to optimize bowel transit time (proper food combining, see Chapter 14)
- ✓ Use of bio-oxidative therapies to obliterate CPC throughout the body outside of the aerodigestive tract (see Chapter 14)
- ✓ Optimal nutrient, vitamin, and mineral supplementation
- ✓ Optimization of sex hormone status and thyroid hormone status

Recap

The leaky gut syndrome plays a pivotal role in causing many diseases and making many diseases worse. For the most part, it is primarily caused and sustained by the continual swallowing of pathogens and toxins from focal infections and areas of CPC in the aerodigestive tract. Oftentimes nothing more than the regular, and sometimes just periodic, nebulization with HP can clinically normalize gut function and prevent its recurrence. Many other factors can be addressed to help HP nebulization reach this goal.

How is “leaky gut” related to respiratory infections and CPC?

The gastrointestinal tract (gut) harbors a very large presence of microbes, known as the microbiome. The entire gut is colonized, extending from the mouth to the anus (aerodigestive tract and gastrointestinal tract). Estimates in the scientific literature assert that the body has roughly 37 trillion cells, but the microbiome consists of about 100 trillion microbes, underlining the significant degree of its presence in the body.¹ This microbe presence is literally an ecosystem of its own, and it impacts physiological processes throughout the body. A normal balance of these microbes has a powerfully positive impact on health, and an excessive presence of pathogenic microbes can cause and/or worsen just about any chronic disease.

A diverse array of microbes, including bacteria, fungi, protozoa, and viruses, is found in the microbiome, and it is present in all mammals. The health and the integrity of the cells lining the gut are directly dependent on a normal microbiome. When the

pathogen presence is both substantial and chronic, a leaky gut ensues due to the ongoing oxidative stress inflicted by those pathogens and their associated toxins. Subsequently, the pathogens and their toxins

If the microbiome is never returned to normal, the seeding of pathogens and toxins throughout the body never stops.

gain access to the rest of the body.^{2,3} If the microbiome is never returned to normal, the seeding of pathogens and toxins throughout the body never stops. And currently there is no accepted medical therapy that can completely normalize the microbiome and maintain it in that normal state. Because of this, variable degrees of leaky gut rarely resolve completely.

It follows, then, that optimal energy homeostasis and cellular metabolism in general, along with the presence of normal immune function, absolutely require the maintenance of a normal gut microbiome. A failure to maintain the normal microbiome allows pathogens and toxins to continuously disseminate throughout the body.^{4,5} The ongoing physiological and pathophysiological interactions between the gut microbiome and the rest of the body has led some authors to refer to it as a “microbial organ,” as its impact on health is as significant as any other organ in the body.⁶

When microbiomes become less diverse, with increased levels of pathogens present, many diseases worsen. Numerous medical conditions have been documented to be associated with an unhealthy,

pathogen-dense microbiome. And although it is not definitively established, these associations would likely better be characterized as contributing **causes** rather than just correlations to these conditions. A partial list of these conditions and diseases includes the following:

- ✓ Breast, pancreatic, and colorectal cancer⁷⁻¹⁰
- ✓ Heart disease and metabolic syndrome^{11,12}
- ✓ Hypertension (high blood pressure)¹³
- ✓ Pregnancy complications^{14,15}
- ✓ Food allergies and allergic conditions^{16,17}
- ✓ Autoimmune thyroid diseases¹⁸
- ✓ Polycystic ovary syndrome¹⁹
- ✓ Neurological and central nervous system conditions (poor memory, migraine, depression, autism, Parkinson’s disease, stroke)²⁰⁻²⁶
- ✓ Irritable bowel syndrome²⁷
- ✓ Inflammatory bowel disease²⁸
- ✓ Liver and bile disorders²⁹⁻³²
- ✓ Abnormal lipid metabolism³³
- ✓ Diabetes³⁴
- ✓ Arthritis^{35,36}
- ✓ Rheumatic diseases³⁷
- ✓ Lung diseases³⁸⁻⁴⁰
- ✓ Eye diseases^{41,42}
- ✓ Periodontitis and halitosis⁴³⁻⁴⁵
- ✓ Obesity and weight gain⁴⁶⁻⁴⁹

This wide array of medical conditions and diseases that are worsened in the presence of abnormal gut microbiomes should make it clear how important it is to get and maintain the gut bugs in as normal a balance as possible. Pathogen presence should be kept minimal relative to the presence of the well-docu-

mented “friendly” gut bugs, and an overall greater diversity in such microbe types is also optimal.⁵⁰

As all disease gets started and continues to be sustained by an ongoing exposure to new oxidizing agents, the significance of the chronic swallowing of CPC-produced toxic waste cannot be underestimated. There are no oxidizing agents more potent than pathogen-produced toxins and metabolic byproducts. The presence of CPC in the nose, oral cavity, throat, and airways assures the continued presence of at least some degree of abnormal microbiome and compromised gut function until it is effectively remedied, and a normal flora is then able to replace it.

Most often, CPC is the low-grade infectious state that chronically remains after an acute respiratory viral syndrome has clinically resolved. Certainly, it can also arise from a sufficient pathogen exposure that never quite reached the point of clinical manifestation of a new cold or flu. However, the most common predisposing circumstances will be as the residual pathogen colonizations following the clinical resolution of a cold or case of the flu.

In fact, it appears that a significant majority of everyone who has had a cold or the flu will end up with a post-infection state of CPC. Furthermore, since CPC quickly protects itself with tenacious biofilms, such cases of CPC will ***persist indefinitely*** after a cold or flu until appropriate remedial measures are taken. This means that there are enormous numbers of people who keep this post-infection CPC for many years and sometimes for life. Of note, the “epidemic” of halitosis affecting so many is often secondary to CPC not otherwise suspected.

CPC in the throat and oral cavity can always be expected to negatively impact overall health, sometimes severely. Additional sites of CPC in the gut itself are also major contributors to the development and

Furthermore, since CPC quickly protects itself with tenacious biofilms, such cases of CPC will ***persist indefinitely*** after a cold or flu until appropriate remedial measures are taken.

maintenance of an abnormal gut microbiome, with all of its associated gastrointestinal and systemic disorders, cited earlier. Depending on the location of CPC sites, pathogens and their associated endotoxins and exotoxins are either continually swallowed or just directly produced in the gut. This results in a very large and chronic oxidative stress on the cells lining the intestines that will always result in breaches in the intestinal mucosal barrier.⁵¹ These resultant breaches are often referred to as a “leaky gut” because pathogens and toxins are now able to slip through the intestinal barrier surrounding cells and into the bloodstream and lymphatic system. The more pro-oxidants (toxins) that are continually swallowed or directly produced in the gut, the more permeable and leaky these cells become (and remain).

At the same time that the CPC is directly seeding and sustaining the development of an unhealthy microbiome, this pathogen-ridden microbiome starts chronically producing an ***additional*** number of pathogens

and pathogen-related toxins along with those which are being swallowed from above.

Wherever situated in the aerodigestive and gastrointestinal tract, sites of CPC literally cause that foundational condition (leaky gut) while simultaneously providing a continual supply of new pathogens and toxins to pass through that compromised gut barrier by the continual interplay of all the factors noted above. And once well-established, the abnormal microbiome that resulted from all of these factors independently produce even more pathogens and toxins to access the body through the leaky gut. A rough “schematic” of these interacting factors is as follows:

- ✓ Pathogens and toxins cause leaky gut.
- ✓ CPC in both the aerodigestive tract and the gut itself produces these pathogens and toxins.
- ✓ All CPC-associated pathogens and toxins access the body through the leaky gut.
- ✓ All CPC-associated pathogens increases the pathogen content and decrease the microbial diversity of the microbiome.
- ✓ That pathogen-altered microbiome produces more pathogens and toxins.
- ✓ Those additional microbiome-related pathogens and toxins further sustains the oxidative stress causing the leaky gut.
- ✓ Those additional microbiome-related pathogens and toxins then access the rest of the body across the leaky gut they have helped to form and sustain with the help of all sites of CPC.

- ✓ The increased pathogen and toxin content in the gut slows gut motility and more food ends up putrified rather than digested, and more pathogens and toxins are formed and are available to the rest of the body via the leaky gut. Additional toxin ingestion (such as iron, see below) and poor food combinations further sustain these gut abnormalities.

The negative impact of CPC on inflammation-induced loss of intestinal barrier integrity is further augmented by the exposure of the gut to any other pro-oxidant (toxic) substance that can not only inflict direct oxidative damage to these barrier cells but can also promote further microbiome damage. The most common toxin exposure of this nature faced by most individuals to some degree is due to the chronic ingestion of iron needlessly added to nearly all processed foods and even some organic foods as well. Although an essential nutrient agent, iron becomes highly toxic when ingested beyond the needs of maintaining a normal level of hemoglobin synthesis. It should never be administered or ingested in any form unless a documented iron deficiency anemia is present. And it should be promptly discontinued after the anemia has resolved.

This “nutrient/toxin” profile of iron is shared with copper and calcium. These three agents can accurately be regarded as toxic nutrients. They are absolutely necessary for sustaining normal physiology and good health. However, they quickly turn into three of the most toxic agents in an otherwise nutritious diet, or supplementation regimen, when the daily physiological

need for these three substances is chronically exceeded. All three of these agents, directly or indirectly, result in the significant production of oxidation and oxidative stress.

Iron in particular gets routinely added to many processed foods, often in the form of metallic iron filings, which only further increases the toxic impact of that ingested iron. This adulteration of our foods began in 1941.⁵² When both CPC and the regular ingestion of extra dietary iron (and from any supplements or prescriptions) are eliminated, an enormous improvement in gastrointestinal health, bowel function, and general health can very often be realized. Multiple animal studies have documented that the ingestion of iron in any form directly causes both gut inflammation as well as increased oxidative stress that provokes disease elsewhere in the body.⁵³⁻⁵⁷

The toxicity of iron supplementation is consistently present in one of the most supplemented subset of patients: the pregnant woman. While often supplemented appropriately in some individuals with clear-cut iron deficiency anemias, it is also common to supplement any pregnant woman to “prevent” an iron deficiency anemia from developing. The pregnant state itself is associated with increased oxidative stress and any iron supplementation only further adds to the degree of this stress.^{58,59} And even when an iron deficiency anemia is present in any woman, pregnant or otherwise, proper dosing and close clinical follow-up are essential, since iron supplementation can still cause some increased oxidative stress/harm even though some iron is needed to resolve the anemia.

The minimal iron dose and the minimal time that it is administered are important treatment goals.^{60,61}

The toxic (pro-oxidant) impact of iron in general as well as on the gut is substantial. Iron supplemen-

Iron in particular gets routinely added to many processed foods, often in the form of metallic iron filings, which only further increases the toxic impact of that ingested iron.

tation continues to receive unwarranted support by many healthcare providers for individuals with nonspecific anemias associated with chronic disease, such as cancer, even though lab tests indicate no iron deficiency and oftentimes iron excess. In fact, the great majority of adults in the United States do not have an iron deficiency, yet they are involuntarily subjected to a regular “supplementation” of this toxic nutrient because of the way most processed foods are treated.⁶² Now modern science has developed a way to genetically modify rice so it contains twofold to threefold more iron than in regular forms.⁶³ The general toxicity of iron and the negative impact of its excess dietary and supplemental intake is addressed elsewhere in greater detail.⁶⁴

Most people, including their physicians, simply accept that diarrhea, constipation, gastritis, bloating, abdominal discomfort/pain, inflammatory bowel syndromes, or almost any other gastrointestinal condition is largely inevitable and typically occurring for

seemingly no clear-cut reason, or just the inevitable consequence of growing older. The standard therapeutic goal aims to lessen the gut-related symptoms as best as possible with a variety of prescription and over-the-counter agents. Similarly, there is virtually no awareness that CPC sets the stage for the inflammation causing and sustaining some gastrointestinal cancers, even though chronic inflammation in the gut is recognized as an important factor in the development of cancers there.^{65,66} Medicine seems to accept chronic inflammation as a disease in and of itself without questioning how or why it arose in the first place.

Are there other effective ways to treat COVID?

Overview

Never before has something generated confusion, anxiety, and outright, gut-wrenching fear throughout the entire planet as the COVID pandemic of 2020. While only the scientific and clinical data will be emphasized here, it is nevertheless important to realize that politics and medicine have been intimately interwoven in dictating the best ways to prevent, treat, and conduct our lives with regard to each other during this pandemic.

The material presented in this chapter is intended to clearly demonstrate that true science has much better answers than those the political-medical coalition is currently pushing. Along with highly effective virus treatments that accelerate clinical recovery, multiple rapid **cures** that readily eradicate COVID or any other respiratory virus will be discussed. It is up to the reader to decide how much trust should be given to those in government and administrative agencies who

literally are turning billions of lives upside down in the United States and around the world.

While still unknown to most practitioners of traditional (“modern”) medicine, acute viral syndromes can be easily prevented most of the time. And once contracted, acute viral syndromes have been rapidly resolved (cured) for a very long time now. Furthermore, “new” viruses are still just as susceptible to the therapies to be discussed as any of the well-known viruses. Unless a patient is already too close to death or has too many underlying conditions compromising the immune system, a complete and rapid recovery from most viral syndromes, especially those contracted via the respiratory route, can be anticipated.

Of course, many doctors get attacked for promoting anything aside from prescription medicines as cures or simply recommending them as beneficial therapies for any infection or condition considered to be incurable. And while it is true that some non-traditional treatments embraced by alternative medicine are either fraudulent or of only nominal benefit, this cannot be allowed to negate clearly positive therapies that are not based on pharmaceuticals. Ethically, it cannot be over-emphasized that:

**Failing to assert the validity of a true
cure for a medical condition is just
as detrimental to the health of a sick
patient as it is to promote a false cure.**

Unfortunately, many doctors who use and promote inexpensive and natural therapies experience the threat of license revocation if they continue with such therapies. And when a physician’s entire practice and ability

to earn a living is threatened, the only real “option” is to comply and rely only on prescription drugs and traditional treatment approaches. Nothing is ever embraced that would significantly reduce the large

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profits of pharmaceutical companies, hospitals, insurance companies, and even the mainstream doctors themselves. And when a particular treatment is very effective, non-toxic, and cheap, it will often be actively **suppressed** rather than just ignored. Whenever it is impossible to fathom why a clearly valuable treatment is not being used, just calculate the revenue that might be lost — in billable drugs, tests, hospital stays, and procedures — if that treatment suddenly replaced the current “standard of care.” The reason for the avoidance/ridicule/suppression of that therapy will then become apparent.¹

The best example of such an enormously beneficial therapy that is still steadfastly avoided is intravenous vitamin C (IVC). While the literature of the last 70 to 75 years makes it clear as to how safe and **enormously** effective IVC is in a wide variety of conditions, it remains unappreciated by most physicians. Even more often, however, IVC is actively ridiculed by physi-

cians who have clearly made no attempt to educate themselves on its benefits, even when presented with massive amounts of peer-reviewed literature.

An especially flagrant example of this common malpractice is seen every time a dying, septic patient in the ICU is not offered — or worse — **denied** IVC. Conservatively, thousands of ICU patients around the world would be saved **every day** with the appropriate administration of IVC.² And outside of the ICU setting, the morbidity and mortality of nearly all infections and diseases would be lessened markedly with the proper use of vitamin C, orally or intravenously.

Hypocritically, traditional medicine consistently tries to invalidate most natural therapies by holding them to a standard that is not even met by most prescription drugs. That standard is the “prospective double-blind, placebo-controlled trial” conducted on hundreds and even thousands of patients. And although all of the therapies to be discussed would be solidly validated by such an approach, an enormous amount of money, sometimes in the millions of dollars, is required to conduct such trials.

In general, natural therapies and older drugs cannot be patented so there is no way to recoup the expense of clinical trials by establishing a legal “monopoly.” With the freedom to charge whatever the market will bear, a company can view clinical trials as an investment; without it there is no current way to cover those costs. Consequently, such validations are impossible and any positive reports, regardless of the number of them or the degree of success, are dismissed as “anecdotal.” And yet, an objective and scientific clinician realizes that even **one** overwhelm-

ingly impressive case report can clearly establish the benefit of a therapy that is already established to be extremely safe. When something promptly reverses the

In general, natural therapies and older drugs cannot be patented so there is no way to recoup the expense of clinical trials by establishing a legal “monopoly.”

condition of a rapidly declining patient with a raging infection back to good health, it simply cannot be disregarded as “anecdotal” and irrelevant from either a scientific or an ethical perspective.

In another example of hypocrisy, published case reports, even though they are simply “anecdotal” experiences of clinicians, are readily used by mainstream doctors to justify the ability of a prescription drug to produce a positive outcome. However, after any agent clearly saves the life of only one patient who is rapidly deteriorating, it is completely **unethical** to put a series of similar patients in a placebo-controlled clinical trial, especially when the agent used has never shown toxicity after many years of use by the public and countless integrative physicians (as is the case with vitamin C). Allowing patients in the placebo group to suffer needlessly and even die under such circumstances can never be justified.

The following therapies are highly effective against all respiratory viruses, including COVID. Most of them resolve or greatly improve any type of acute infection

anywhere in the body. For the reasons already outlined above, each agent has not been equally well-documented in its effectiveness in the scientific literature. Some have very strong support in the literature. Others, however, simply represent logical applications of protocols already proven to be highly effective against viruses, and they can be expected to positively impact COVID to a comparable degree. The ability of each therapy to **PREVENT, IMPROVE, or CURE** COVID will be specifically noted as well.

Pathogen Killing—The Common Denominator

Just as normalizing or even decreasing elevated IOS (intracellular oxidative stress) is the key to health [see Chapter 13], increasing IOS or keeping it elevated is the key to chronic illness, including chronic infection. And the only way to kill a pathogen or a pathogen-infected host cell is to increase the IOS of either or both to a level high enough to produce prompt programmed cell death (apoptosis) or frank cell rupture. Any therapeutic measures that effectively resolve infections are interventions that share the ***final common denominator*** of producing severely increased IOS in the pathogen and/or host cell.

The most effective anti-pathogen treatments are those which can rapidly increase pathogen and/or host cell IOS to very high, lethal levels. This is most effectively achieved by increasing HP levels in the pathogen or cell and then by breaking down HP into hydroxyl radicals (Fenton reaction). Pathogens and cells are always producing HP, and any HP surrounding them is also free to diffuse into them to further fuel the Fenton

reaction. A number of enzymes work to hold HP levels down when accelerated pro-oxidant activity is not desirable, as in the healthy, non-infected state.^{3,4}

And the only way to kill a pathogen or a pathogen-infected host cell is to increase the IOS of either or both to a level high enough to produce prompt programmed cell death (apoptosis) or frank cell rupture.

The most effective antiviral and general anti-pathogen agents come under the heading of Bio-Oxidative Therapies. This group of therapies works to imitate and/or augment the ways in which the healthy body naturally prevents or resolves infections.

Nearly all of the anti-pathogen impact of these bio-oxidative therapies comes from the eventual direct or indirect actions of HP on target pathogens and infected cells.

Furthermore, all of these therapies result in an **increased delivery of oxygen to the organs and other tissues**, although the mechanisms of action involve more than can be achieved with just inhaling supplemental oxygen.⁵ Also, the four therapies listed after HP all have their anti-pathogen impact at least in part by resulting in the **formation** of HP in the body.

The Bio-Oxidative Therapies to be addressed are:

- ✓ HP
- ✓ Vitamin C

- ✓ Ozone
- ✓ Ultraviolet blood irradiation (UBI)
- ✓ Hyperbaric oxygen therapy

Hydrogen Peroxide (*prevents, improves, cures*)

HP, especially administered via nebulization, is discussed at length in Chapter 11. It has been in use for very many years now as a common and reliable way to effectively treat a wide variety of infections. Its lack of expense is exceptional:

A clinically curative dose of HP, by either nebulization or intravenous infusion, literally costs 10 cents or even much less. This will always be the major impediment to its acceptance and widespread application by physicians and hospitals dedicated only to the administration of costly prescription drugs. The sad fact is that patients who stay sick and received “accepted” therapies generate enormous amounts of money for the “healthcare” industry.

Of note, during the influenza pandemic that started in 1918 and ultimately killed 500 million people worldwide, a protocol of intravenous HP given only to the **most critically ill** patients nevertheless dramatically decreased the death rate.⁶ A more aggressive protocol, in terms of dosing and frequency of dosing, would have been expected to result in a survival rate of greater than 95%.

Vitamin C (*prevents, improves, cures*)

As has been extensively documented for many years now in the medical literature, vitamin C is a potent,

broad-spectrum antiviral agent. There has never been a virus that vitamin C has been unable to inactivate in any *in vitro* (test tube) study. Similarly, it has also been documented to clinically resolve all acute viral

Vitamin C and HP are ***natural physiological partners*** in the body, especially when it comes to dealing with pathogens.

syndromes in patients who have received appropriately-dosed and correctly-administered vitamin C. If there is a virus that is “resistant” to aggressive vitamin C therapy, it is yet to be discovered. Furthermore, it would appear that COVID (coronavirus disease 19) is just as susceptible to vitamin C as any other virus that has come before it.

Vitamin C and HP are ***natural physiological partners*** in the body, especially when it comes to dealing with pathogens. Although vitamin C is an anti-oxidant looking to donate electrons and not oxidize anything, it is the donation of an electron to ferric ion (Fe^{3+}) to make it ferrous ion (Fe^{2+}) that allows that electron to be re-donated to HP in the cell or pathogen, which then directly results in the chemical breakdown of HP to produce the most toxic, oxidizing agent known in biology, the hydroxyl radical (Fenton reaction).⁷ So, it is actually an ***antioxidant activity*** that ultimately results in a pronounced ***pro-oxidant impact*** via the formation of this potent oxidizing agent. When iron or

another transition metal is not present, vitamin C is not chemically suited to easily donate the electron directly to HP, and the HP remains quite stable and poorly reactive.⁸

To keep any chemical pathway operating at top efficiency, there must be a continual supply of the substances participating in the reaction. Otherwise, the reaction will quickly “extinguish” itself as one or more of the reacting substances are used up. To keep the Fenton reaction functioning optimally and producing sufficient hydroxyl radicals to complete the destruction of a pathogen or infected host cell, three factors must be present:

- ✓ A continuing supply of electrons
- ✓ A continuing supply of free, reactive iron (not protein-bound or stored in ferritin)
- ✓ A continuing supply of HP
 - 1. A sufficient supply of electrons is satisfied by having enough vitamin C inside the pathogens and host cells. High-dose vitamin C administration assures the presence of enough new electrons to continue the Fenton reaction to completion.
 - 2. The pathogens themselves contain large amounts of iron, as they literally “feed” on iron and replicate more rapidly as more iron is assimilated. As such, iron-sated pathogens are already **self-targeted** for their destruction via the Fenton reaction. However, protein-bound and stored forms of iron cannot participate in the Fenton reaction. The entry of new HP into the pathogens/cells stimulates the release of iron from its storage

form in ferritin to its free, unbound, reactive form.⁹

3. HP is the primary “fuel” of the Fenton reaction, and new HP needs to continually be supplied in order to realize the complete destruction of the pathogen and host cell, as only water and oxygen remain after its metabolism to hydroxyl radicals. This is achieved by the massively increased amount of HP production that occurs when a large amount of vitamin C is in the extracellular space. This new HP is then free to move into the cells and pathogens, along with more vitamin C to serve as the needed source of electrons.
4. All of these properties explain why highly-dosed infusions of vitamin C are **uniquely suited** to rapidly cure acute viral syndromes, along with many other acute infections.

As the premier electron donor in the body, vitamin C works perfectly with HP by the ease with which it can donate its electrons. Other antioxidants are far less effective and efficient in fulfilling this function. And when the vitamin C has been administered in high enough doses so that a large amount of it is outside the cells or pathogens in the extracellular space, it works to naturally produce large amounts of new HP that can easily diffuse into the cells/pathogens and provide more fuel to the Fenton reaction until pathogen destruction is complete.¹¹⁻¹³

As such, when someone has an overwhelming and life-threatening infection, intravenous vitamin C in high enough doses can be **expected** to save the

patient. This is because vitamin C produces the HP, which then releases the stored iron to an unbound, reactive form. This iron then receives and passes the electrons of vitamin C origin to that HP, resulting in the production of sufficient hydroxyl radicals to complete the pathogen kill. No other antioxidant will come close to the importance of vitamin C in fueling the Fenton reaction and clinically resolving all variety of infections and infectious diseases.

Simply stated, vitamin C is the ultimate anti-pathogen agent since, directly or indirectly, it results in the continued supply of *electrons, free iron, and HP* that can produce hydroxyl radicals until pathogen/host cell death and destruction is complete.

Furthermore, vitamin C properly dosed and administered not only serves as the perfect monotherapy for dealing with pathogens via its broad fueling of the Fenton reaction inside pathogens and host cells, it also serves another extremely important function in dealing with infectious diseases. All significant infectious diseases, especially those which are life-threatening, rapidly deplete the body of its vitamin C stores. As mentioned with the viral hemorrhagic fevers, death actually occurs because of complications from the acute and severe scurvy that is inflicted on the patient. Therefore,

The treatment of all infectious diseases with vitamin C as a primary part of the protocol assures that there will also be no infection-

induced scurvy, and recovery will be further facilitated by the absence of this debilitating condition.

No other agent matches the power of vitamin C in dealing with acute viral infections and the complications they cause.

An additional elegant “designed-by-nature” cooperation between vitamin C and HP in eliminating pathogens is seen in phagocytes and monocytes, the pathogen-killing white cells of the immune system. These cells naturally gather at the sites of inflammation involved in any infection. Relative to other cells and the blood, these phagocytes contain and deliver **massive** amounts of both HP **and** vitamin C directly to the pathogens. This combined delivery immediately inflicts a large Fenton reaction-generated pro-oxidant killing effect on the iron-rich pathogens.¹³⁻¹⁸

In recent years, at least three other viruses emerged that infected many people and scared even more. Ebola virus, a rapidly fatal infection in many who contracted it, was readily cured by a combination of ozone and a supplement regimen that featured vitamin C.¹⁹ It should be noted that ozone treatments of the blood, to be discussed below, are also exceptionally effective in resolving viral syndromes. The primary drawback of ozone is a lack of widespread availability and its inability to address any virus-induced vitamin C depletion.

Even when vitamin C is not the primary antiviral therapy, as in this Ebola example, its addition to the

treatment is vital since patients infected with such an acute hemorrhagic fever viral syndrome rapidly consume vitamin C stores, and they can essentially be considered to be in a state of rapidly developing scurvy as the infection evolves. Such a deficiency of vitamin C greatly decreases the chances of survival, and it also greatly impairs the rate of any recovery that could otherwise result. In fact, death from such infections can easily be regarded as death from acute scurvy, with almost a complete destruction of any vitamin C content in the body. When little to no vitamin C is available to continue to synthesize new collagen and structural proteins to allow smaller blood vessels to maintain their normal strength, hemorrhage and death quickly follow.

Chikungunya virus infection results in a syndrome that can physically debilitate its victims for months, or longer, with a severe, persistent arthritis. Vitamin C infusions, as well as HP infusions and ozone treatments, have resolved this infection rapidly and completely. Even individuals who have residual arthritis after the acute infection typically experience rapid resolution of such arthritic symptoms with the proper application of vitamin C and/or ozone.^{20,21}

Zika fever is another virus that received a great deal of publicity, especially with reports of microcephaly in some babies born of women who contracted the virus. The fear of this virus resulted in many athletes opting not to go to the summer Olympics in Brazil in 2016. However, like the other viruses before it, Zika also proved to be no match for vitamin C given intravenously.²²

Vitamin C has also been shown to readily resolve viral influenza secondary to any of a number of respiratory viruses that initially multiply in the sinuses, nose and nasopharynx, oropharynx, throat, and upper airway.²³ Commonly known as the “flu”, the viral colo-

Clinical studies around the world have consistently indicated that appropriately-dosed vitamin C in COVID patients results in an accelerated resolution of the infection.

nization and multiplication in and around the nose and mouth that sustains and increases the viral load throughout the body is the same way that contraction of the COVID virus occurs and spreads.

Clinical studies around the world have consistently indicated that appropriately-dosed vitamin C in COVID patients results in an accelerated resolution of the infection. Over the course of March, 2020 in the United States, the use of vitamin C as a preventive measure as well as for the treatment of hospitalized COVID patients, particularly in the intensive care units, began to dramatically increase. At that time, all 23 hospitals in the Northwell Hospital Network in New York began giving IV vitamin C to their more advanced COVID patients. Bear in mind that prior to the COVID pandemic, receiving intravenous vitamin C for **any reason** in **any hospital** in the United States **simply did not occur** except for the rarest of circumstances.

In these New York hospitals, the typical vitamin C dose was still only 1.5 grams every six hours. This dosage schedule was chosen because of the success that it provided Dr. Paul Marik in virtually eliminating death in his ICU patients with advanced sepsis. And even though this dose is many-fold lower than the optimal doses given by many vitamin C-using physicians around the world in their clinics, very positive clinical responses were nevertheless reported.²⁴

Significant amounts of research using high, multi-gram doses of vitamin C intravenously for a variety of conditions, usually in the setting of the intensive care unit, has been taking place for many years now in China. Much less of this type of research has been done elsewhere, especially in the United States. A demonstration of the Chinese regard for vitamin C was reflected in their official policies after COVID infections began to increase. As of March 1, 2020, the government of Shanghai, in conjunction with the Chinese Medical Association, officially recommended the use of vitamin C in the treatment of COVID. Consistent with this policy, the Chinese province in which Wuhan is the capital city had 50 tons (45,360 kilograms) of vitamin C shipped there from a manufacturing plant elsewhere in China in early February, 2020. While the attitude toward vitamin C finally appears to be changing because of this pandemic, the United States simply would not have known what to do with 50 tons of vitamin C had it been delivered to any of their most infected cities.

In Wuhan, where the pandemic is believed to have begun, a large trial of intravenous vitamin C for the treatment of hospitalized COVID patients was initi-

ated on February 14, 2020. Designed for the patients with severe COVID pneumonia, a regimen of 24 grams of vitamin C infused over an eight-hour period each day was initiated. This study alone demonstrates the

And even though this dose is many-fold lower than the optimal doses given by many vitamin C-using physicians around the world in their clinics, very positive clinical responses were nevertheless reported.

glaring difference between China and the United States with regard to appreciating the vital role that vitamin C can play in treating any viral infection, along with the type of doses that should be administered.

Early on, the increasing acceptance of vitamin C for infectious disease came from *The Lancet. Respiratory Medicine* in March of 2020. It was asserted that “rescue therapy with high-dose vitamin C can also be considered” for the most advanced COVID patients with severe ARDS (acute respiratory distress syndrome) and supported by mechanical ventilation. For such a prominent and traditional medical journal to even suggest that vitamin C could be of help in “rescuing” patients with respiratory distress and late stage COVID infections is an enormous acknowledgement. Until the COVID pandemic began, the authors in ***nearly all*** traditional medical journals either ignored any role of vitamin C in treating just about anything, or they just showed derision that it could help with any

situation, much less a life-threatening one. And since that assertion, a large number of quality clinical studies have been conducted that continue to demonstrate the great value of vitamin C in treating COVID patients.

Vitamin C, especially when given in concert with a hydrogen peroxide application such as nebulization, is an especially good and synergistic treatment for COVID and any other respiratory virus.

Vitamin C is actually especially effective in dealing with ARDS of any origin, COVID infection included.^{25,26}

At the beginning of 2020, COVID patients in Shanghai were treated with IVC. Advanced COVID patients and “critical” COVID patients were treated with IVC doses ranging between 10,000 and 20,000 mg of vitamin C intravenously for a 7- to 10-day period. All patients who received IVC improved, and there was no mortality. Furthermore, no side effects of any kind secondary to the infusions were noted. It was also shown that the IVC clearly improved laboratory indicators of inflammation and organ deterioration (sequential organ failure assessment score).²⁷

Italy had also taken note of the clear-cut success of vitamin C in the treatment of patients with COVID. Salvatore Corrao, MD of the University of Palermo began a study on March 13, 2020. Entitled “Use of Ascorbic Acid in Patients with COVID,” the study sought to enroll 500 patients hospitalized with COVID

pneumonia and administer 10,000 mg of vitamin C intravenously daily in addition to whatever conventional therapy was being given.

Because of the pandemic and the early positive outcomes seen in patients with COVID given vitamin C, much of 2020 has seen the publication of a large number of clinical studies demonstrating the clear-cut positive impact of vitamin C on these patients. A good place to quickly review many of these studies is online.²⁸

Vitamin C, especially when given in concert with a hydrogen peroxide application such as nebulization, is an especially good and synergistic treatment for COVID and any other respiratory virus. The peroxide kills the pathogens, leaving behind water and oxygen, and the vitamin C repairs any damaged cells and tissues and oxidized biomolecules, while restoring any vitamin C deficiencies that had been induced.

Ozone (prevents, improves, cures)

Ozone is probably the most powerful pro-oxidant that can be administered in a treatment protocol. Certainly, no agent is a more potent pro-oxidant than ozone. Its chemical structure has a strong basic similarity to that of HP. HP could be regarded as hydrogen dioxide, and ozone could be regarded as oxygen dioxide. This dioxide or dioxide-like structure (two bound oxygens in the molecule) appears to be associated with, and perhaps causative for, significant pathogen-killing pro-oxidant capacity. Chlorine dioxide, nitrogen dioxide, sulfur dioxide, and even carbon dioxide are all significant pro-oxidant disinfectant agents.²⁹⁻³³ Titanium dioxide and silica dioxide have

actually been shown to be superior disinfectant agents to the highly effective dental disinfectant, chlorhexidine.^{34,35} Any molecule that can exert a toxic, pro-oxidant effect against pathogens while sparing normal

Like HP, ozone has been well-documented to increase blood levels of oxygen and oxygen delivery to the tissues in addition to its anti-pathogen effects.

cells and biomolecules will prove to be a powerful clinical agent for dealing with all variety of infections. No agent can resolve any infection without having a direct or indirect means of massively upregulating oxidative stress in the pathogen or host cell. How effectively this goal is achieved while leaving normal cells untouched determines whether a given anti-pathogen agent is an optimal choice.

Unlike HP in many areas of the body, ozone is very unstable and promptly oxidizes many of the molecules it encounters wherever it is applied or delivered. In the blood, ozone reacts with organic compounds containing double bonds, like polyunsaturated fatty acids. This results in the formation of compounds known as ozonides, which are also unstable and immediately metabolized to hydroperoxides and HP.³⁶ These hydroperoxides are much more stable molecules, and like their close relative HP, they can retain their oxygen content until acidic, infected microenvironments trigger the release of that oxygen to initiate

anti-pathogen activity.³⁷ Like HP, ozone has been well-documented to increase blood levels of oxygen and oxygen delivery to the tissues in addition to its anti-pathogen effects.³⁸ Tumor cells that are incubated in an ozonated medium have been shown to accumulate HP in the cytoplasm, further supporting the concept that HP is a major player in the cellular response to ozone therapy.³⁹ It would appear that ozone can help sustain intracellular levels of HP until the Fenton reaction has been able to completely destroy the pathogen and/or host cell.

Large clinical studies on the ozone treatment of COVID are yet to be done, but there is nothing to suggest that this coronavirus will be the first to be resistant to this therapy.⁴⁰ In fact, some small published reports already indicate that ozone autohemotherapy is just as effective with the COVID virus as with any of the other viruses and pathogens that have been effectively treated over many years now. Clinical recoveries were clearly accelerated, and asso-

No agent can resolve any infection without having a direct or indirect means of massively upregulating oxidative stress in the pathogen or host cell.

ciated abnormal blood tests measuring inflammation and proneness to blood clotting (CRP, IL-6, D-dimer) improved rapidly as well.^{41,42} Ozone autohemotherapy has also been shown already to significantly improve COVID patients with mild to moderate pneumonia.⁴³

Ultraviolet Blood Irradiation (*prevents, improves, may cure*)

Ultraviolet light irradiation (UBI) has been enormously effective in killing pathogens wherever they are found for many decades now. It has functioned as an effective disinfectant against airborne pathogens outside of the body.^{44,45} It has also been well-documented in killing multidrug-resistant pathogens found on surfaces in hospital settings.⁴⁶ Not surprisingly, it has already been documented that ultraviolet light irradiation completely inactivates the COVID virus, just like any other virus.⁴⁷ At least part of the anti-pathogen impact of UBI also appears to come from the production and clinical impact of HP.⁴⁸

Also known as photo-oxidation therapy, UBI has been documented to resolve advanced viral and bacterial infections. In a series of 36 cases of acute spinal type polio UBI was successful in curing 100% of these individuals. Viral hepatitis and bacterial sepsis have also been resolved with UBI.⁴⁹ In chronic hepatitis UBI consistently lowers viral loads.⁵⁰ UBI therapy has also been shown to dramatically improve the clinical response of pulmonary tuberculosis as part of a treatment protocol.^{51,52} In fact, pathogens resistant to ultraviolet irradiation have not been reported, and microbial strains that have developed antibiotic resistance are just as susceptible to ultraviolet irradiation as those strains that have not evolved a resistance.⁵³ It is questionable whether UBI would work well as a monotherapy to effect a cure in an advanced case of COVID. But there is no doubt that it would augment any multi-pronged protocol aimed at achieving a cure. Whenever

available, there should be no reluctance to utilizing this extremely valuable and exceptionally safe therapy.

While different mechanisms are involved in the killing of pathogens outside of the body by ultraviolet

In a series of 36 cases of acute spinal type polio UBI was successful in curing 100% of these individuals.

light, the treatment of the blood with ultraviolet light has nevertheless proven to be highly effective in eliminating any pathogens encountered. Outside of the body ultraviolet light directly breaks down pathogen structures, including their nucleic acid.^{54,55} Inside the body, the treatment of a limited amount of blood works to clear pathogens out of the entire volume of the blood quite rapidly. After only irradiating between 5 to 7% of the blood volume, an optimal anti-pathogen impact inside the body can be anticipated.

UBI has been found to enhance the concentrations of HP inside the phagocytic white blood cells. This means that this ultraviolet irradiation of the blood results in the ability of these immune cells to be even more effective in destroying the pathogens to which they are drawn by the infection-associated local inflammation. This also supports the concept that vitamin C, which directly partners with HP in killing pathogens, is ALWAYS an excellent and highly indicated adjunctive agent to give with any bio-oxidative therapy. There are also many other ways in which UBI impacts the immune system and the circulating immune cells.

Many complex mechanisms involved in this immune impact have been identified.⁵⁶

Hyperbaric oxygen therapy (*probably improves, may cure*)

Hyperbaric oxygen therapy (HBOT) is the breathing of pure oxygen inside a chamber that is usually pressurized between 1.5 to 3 times normal atmospheric pressure. HBOT has been well-documented to consistently resolve deep-seated and otherwise non-healing infections. This includes such notoriously refractory chronic infections like necrotizing fasciitis, osteomyelitis, and infective endocarditis.⁵⁷ Logically, HBOT should be as effective or even more effective in dealing with infections that are not deeply embedded in the tissues, as in any acute infectious disease like COVID. As with the other bio-oxidative therapies, HBOT appears to utilize HP in rendering its anti-pathogen properties.⁵⁸

Some authors are especially enthusiastic about the option of HBOT for COVID since decreased blood oxygen levels are seen in advanced infections, and HBOT has been long been known to elevate blood oxygen levels significantly.⁵⁹ While HBOT would likely never become a first-choice therapy due to availability with only a limited number of conventional metal chambers that can reach and exceed 1.5 atmospheres of pressure, it should prove to be a wonderful option for struggling patients who have HBOT access at their hospital or clinic and are not promptly responding to other therapies.

For the reasons of limited availability as well as significant expense, HBOT will not likely become a

routine therapy for COVID or other acute infections, especially with the other inexpensive and highly effective therapies already discussed. However, wherever it is available and other measures applied are not

HBOT should be as effective or even more effective in dealing with infections that are not deeply embedded in the tissues, as in any acute infectious disease like COVID.

resulting in a clear-cut and steady clinical improvement, strong consideration should be given to incorporating it into the treatment protocol.

Natural Adjunctive Agents

While the five bio-oxidative therapies described above can often serve as effective monotherapies in resolving COVID and other respiratory viral syndromes, it is always best to use them in combination with other known effective therapies, both natural and/or prescribed. No patient should suffer needlessly when multiple beneficial and safe therapies are available. In addition to reducing the chances of death, reducing the duration of suffering along with the need for hospitalization in COVID patients is a very important therapeutic goal as well.

There are many natural agents with solidly established benefits in the treatment and/or prevention of COVID that should really always be part of a treatment protocol. Some of the more important of these agents include vitamin D, magnesium chloride, zinc, and quer-

cetin. Vitamin C should always be included as well even if it is not being utilized as a primary therapy.

Vitamin D (*prevents, improves*)

Vitamin D has long been well-documented to strengthen immune function while decreasing the risk of infection from any pathogen.^{60,61} Not surprisingly, this protection has also been demonstrated for COVID infections. Patients with the highest vitamin D levels have substantially shorter and less symptomatic courses of infection, along with lower mortality rates.^{62,63} In nursing home patients during the COVID pandemic, vitamin D supplementation was inversely associated with mortality.⁶⁴ In an ecological study across 46 countries there appeared to be an association between vitamin D deficiency and an increased risk of infection by, and death from, COVID.⁶⁵ And while vitamin D appears to always accelerate the rate of clinical recovery, it has not been documented to eradicate an infection as a monotherapy, and it should not

...restoring and maintaining vitamin D to normal levels should be a part of ALL prevention and treatment protocols for COVID infections...

be relied upon to do so. But restoring and maintaining vitamin D to normal levels should be a part of ALL prevention and treatment protocols for COVID infections if not already part of a program for the maintenance of optimal health.

Vitamin D deficiency has also been found to contribute to the development of the acute respiratory distress syndrome (ARDS).⁶⁶ ARDS is a feature of advanced and often fatal COVID infection. Avoiding vitamin D deficiency appears to be a good way to help assure that even if COVID is contracted, death would be a very unlikely outcome, and any clinical course of the disease would be much less severe.⁶⁷⁻⁶⁹

Magnesium chloride (*prevents, improves, may cure*)

Magnesium, especially as magnesium chloride, has been shown to be a powerful anti-pathogen agent by itself. It has even been reported to quickly cure both acute and semi-acute poliovirus infections as a mono-therapy when ingested orally.^{70,71} Many other infectious diseases, in both humans and animals, have also been reported to respond very well to magnesium chloride.⁷²⁻⁷⁴

The chloride anion appears to be an important contributor to the anti-pathogen impact. One *in vitro* study showed that magnesium sulfate (or sodium sulfate) ***strongly protected*** the measles virus from heat-induced inactivation while magnesium chloride ***enhanced the inactivation*** of the virus at all temperatures tested.⁷⁵ Magnesium sulfate, as well as sodium sulfate, also enhanced viral replication in cultured cells.⁷⁶ The administration of intravenous magnesium sulfate in asthmatic infants and young children with virus-induced wheezing was ineffective in providing relief. One such study even suggested potential asthma aggravation.^{77,78} There is the possibility that the sulfate anion was supportive of the viral presence provoking the bronchospasm. However, the conclu-

sions are not clear-cut, as both zinc chloride and zinc sulfate markedly reduced viral titers in cultured cells.⁷⁹ Both anion and cation would appear to be important in determining the impact on viral replication, and it is not clear at this point how to reliably predict the anion impact from a given agent.

Many other studies on asthmatic adults and children are inconclusive on the effects of magnesium sulfate given by vein or by nebulization.⁸⁰ However, it seems reasonable that a better result would be seen with magnesium chloride, administered either by nebulization or by vein. Certainly, it is very clear that getting more magnesium inside the bronchial smooth muscle cells promotes bronchodilation and an easing of wheezing in asthmatics.⁸¹

For unclear reasons, the scientific literature contains very few studies that give magnesium intravenously in its chloride form rather than sulfate form in both clinical and animal trials, although the chloride form appears quite effective and safe for vasospasm and other known indications for magnesium therapy.⁸²⁻⁸⁵ No studies looking at the nebulization of magnesium chloride were found, although this may well be the superior route of administration and form of magnesium for this application. Certainly, for any infections being treated with magnesium, using the chloride form over the sulfate form appears to be the logical choice, regardless of the route of administration. Most supplement research is generally focused on the positively-charged cation, while virtually dismissing any possible relevance or unique physiological impact of the negatively-charged anion. Oftentimes, the only

significance given to the anion is whether it improves absorbability of the cation being ingested.

However, regardless of the anion involved in an administration of magnesium to a patient, the magnesium cation (Mg^{2+}) has its own powerful anti-infection impact due to its ability to help normalize increased IOS in infected cells throughout the body. Infections cannot be sustained when host cells or potential host cells maintain normal IOS levels.⁸⁶ No form of magnesium supplementation has ever shown that the accompanying anion could negate the positive impact of the magnesium. Nevertheless, it is advisable to try to use supplements that have both cations and anions with positive clinical impacts.

Zinc (prevents, improves)

Once coronavirus successfully infects a host cell, the growth, or replication, of that virus is dependent on an enzyme known as RNA polymerase.^{87,88} Zinc has been documented to inhibit this enzyme. It has been shown in cell culture that increasing intracellular zinc levels impairs viral replication.⁸⁹⁻⁹⁰ When the activity of this enzyme can be sufficiently inhibited, viral growth is effectively stopped, and natural immune mechanisms are then able to proceed with eliminating the infection.

Zinc deficiencies are extremely common, especially in the older population. When the intracellular levels of ionic zinc can be sufficiently raised, viruses either stop replicating or are unable to take hold in the first place, making zinc supplementation important for both preventing and resolving viral infections.^{91,92} Zinc has long been known to have significant antiviral proper-

ties.⁹³ It is a possibility worth considering that some of the younger individuals who had severe or even fatal COVID infections had significant zinc deficiencies increasing their susceptibility. Many individuals who are otherwise “healthy” have diets very limited in nutrition, particularly with regard to minerals and vitamins.

Due to their ionic nature, most forms of supplemental zinc have only a limited ability to reach the cytoplasm. Certain agents known as ionophores are able to complex with zinc and transport it across the cell membrane. Quercetin is one effective natural zinc ionophore, and both chloroquine and hydroxychloroquine are effective prescription ionophores.^{94,95} Quercetin has been documented to have significant antiviral effect against Ebola virus.⁹⁶ As an inexpensive and natural nutrient supplement, supplementation of quercetin along with zinc would be a good practice.

Prescription Agents and Interventions

There are a number of highly effective prescription drugs that offer a great deal of benefit in the prevention and treatment of COVID and other viral syndromes. These agents include chloroquine, hydroxychloroquine, and ivermectin. Antibody-based immunotherapy, which includes convalescent plasma transfusions and other forms of antibody/immunoglobulin administration have also shown significant benefit in viral infection resolution.⁹⁷

Chloroquine (CQ) and Hydroxychloroquine (HCQ) (*prevents, improves, may cure*)

CQ/HCQ are two old drugs that have been administered billions of times around the world, as they

have been used as anti-malarial agents since 1944. In fact, chloroquine is one of the most prescribed drugs in the entire world.⁹⁸ For their common indications of antimalarial prophylaxis as well as rheumatic disease treatment, side effects have been minimal, even when chronically administered over a period of several years. Appropriately monitored, CQ/HCQ result only in limited as well as easily preventable toxicity, and there should be no restraint in using them as preventive measures, much less in life-threatening situations.⁹⁹⁻¹⁰¹ For the entirety of 2020, there has existed a scientifically unfounded basis for excessive concern over the toxicity of these drugs, especially in the context of their potential role in slowing or even ending the pandemic. It would seem that the political side effects outweighed the medical side effects of CQ/HCQ.

CQ and HCQ have both long been documented to have clear-cut antiviral properties.^{102,103} The importance of intracellular zinc in stopping viral replication was mentioned above. CQ/HCQ are both zinc ionophores, and taking supplemental zinc with these drugs appears to improve the antiviral impact of these drugs in treating COVID.¹⁰⁴⁻¹⁰⁶ CQ/HCQ also appear to have broad spectrum anti-pathogen activity, including antibacterial and antifungal activity.^{107,108}

In two studies CQ administered with azithromycin has shown clear benefits in COVID patients. In addition to accelerated clinical resolution of the infections, a more rapid reduction in viral load was documented as well.^{109,110} Many other studies support the clinical benefit of CQ/HCQ therapy in COVID patients.¹¹¹⁻¹¹⁴ Yet, other published papers on CQ/HCQ with and without azithromycin have not been as

clear-cut in the benefits afforded.^{115,116} However, none of them have demonstrated any consistent harm to the patients.¹¹⁷ There should not be any opposition to using any agent that is inexpensive and of almost inconsequential toxicity when some clinicians are seeing the benefits of such a treatment.

This surprisingly vigorous opposition by many physicians and governmental authorities closely parallels the opposition to and even suppression of many powerful natural therapies, as noted above. CQ/HCQ are long past patent protection, and they remain very inexpensive. Also, as with many of the natural therapies already mentioned, there appears to be a strong opposition to finding anything that would definitively resolve the pandemic and negate the perceived “need” for vaccination of the entire world population.

A limited amount of clinical feedback given to the author of this book early in the course of the pandemic supports the conclusion that oral chloroquine phosphate is not only effective in the treatment of COVID, but profoundly so. In six patients with rapidly evolving symptoms of COVID the clinical impact of chloroquine was impressive. Ranging in age between 35 to 65 years, therapy was initiated in all of these individuals when breathing was already difficult and clearly worsening. Significant improvement was seen in all six within about four hours after the first dose of chloroquine, with nearly complete clinical recovery seen after about an average of three days. The oldest individual had a pulse oximetry reading of about 80 before the first dose of chloroquine, and the reading improved to 94 as the labored breathing eased over the next few hours.

The rapidity with which the shortness of breath improved and eventually resolved in all these individuals was completely consistent with evolving respiratory failure secondary to COVID-induced acute respiratory distress syndrome (ARDS). It is this ARDS

*Many other studies support
the clinical benefit of CQ/HCQ
therapy in COVID patients.*

that resulted in the need for mechanical ventilation that was very common early on in the pandemic. The chloroquine dosing was continued for several days after apparent complete clinical resolution to prevent potential relapses.

Of note as well, the numbers of COVID patients requiring mechanical ventilation dramatically declined as the pandemic continued. A much larger number of COVID patients and potential COVID patients were taking regular supplementation that included vitamin C, vitamin D, magnesium, and zinc several months into the pandemic. This could certainly account for the seemingly general decline in the clinical severity of COVID infections. Very many individuals toward the end of 2020 had positive COVID tests while having few symptoms or remaining symptom-free.

In the setting of high-risk COVID exposure, it is advisable to take zinc supplementation along with a regular dosing of CQ or HCQ. Azithromycin can be taken as well. However, unless you are convinced that only prescription drugs can offer you full protection,

regular HP nebulization along with the supplementation of vitamin C, vitamin D, magnesium chloride, and quercetin will further that protection. They only increase protection and treatment effectiveness, and they do not negate at all any of the benefits offered by the prescription agents. Additional therapy can always be taken at the first sign of any breakthrough symptoms of infection, or when a COVID test is positive.

It is also noteworthy that many front-line health-care workers, including physicians, are on preventive protocols that include CQ or HCQ. Yet many of these doctors resist using these same drugs on their patients. This is hard to reconcile with the assumption that the welfare of the patients is always their primary priority.

Ivermectin (*prevents, improves, may cure*)

Ivermectin was discovered in 1975 and was initially utilized as a broad-spectrum antiparasitic agent. As it turns out, however, it is also effective against a wide array of pathogens, including many viruses for which there are no accepted and effective treatments used by mainstream medicine. This includes prominent RNA viruses, like Zika, dengue, West Nile, Chikungunya, and the COVID virus.¹¹⁸ In the test tube, ivermectin has demonstrated the ability to inhibit the replication of SARS-CoV-2, the causative virus for COVID infections.¹¹⁹ It has also been shown to be effective against many DNA viruses.^{120,121} It appears to have multiple antiviral mechanisms.¹²²

There are countries that have been using ivermectin in a preventive role against parasitic infections. The routine mass administration of ivermectin as prophyl-

laxis against parasites appears to have resulted in a significantly reduced incidence of COVID in those countries.¹²³ India has been disseminating a combination of agents known as the “Ziverdo Kit” to much of its population as both prophylaxis against and treatment for COVID. It contains zinc acetate 50 gm (14 tablets), doxycycline 100 mg (10 capsules), and ivermectin 12 mg (3 tablets). The price per kit is less than three dollars USD. It is also advised that a vitamin D supplement be taken as well. In a systematic review and meta-analysis of ivermectin as an added therapy to other COVID treatment protocols, significantly improved clinical responses were seen.¹²⁴

While it is unclear how effective ivermectin would be as a monotherapy in moderate to advanced clinical cases of COVID, it is being increasingly incorporated into multi-agent protocols for treating this infection. In addition to the Ziverdo Kit mentioned above, many

The routine mass administration of ivermectin as prophylaxis against parasites appears to have resulted in a significantly reduced incidence of COVID in those countries.

researchers have been studying the antiviral impact of ivermectin in combination with hydroxychloroquine and azithromycin.¹²⁵ In an animal study, it has also been demonstrated that the nebulization of ivermectin is an effective way to readily achieve higher concentra-

tions in the lungs than is readily achieved with what are considered safe doses orally.¹²⁶

The Front Line COVID Critical Care Alliance (www.flcc.net) has issued their recommended prophylaxis and treatment protocols for COVID. As of December 9, 2020, the Alliance has included ivermectin in both preventing and treating COVID. Other agents in their protocols include vitamin C, vitamin D3, quercetin, and zinc.

Antibody-Based Immunotherapy *(improves, may cure)*

Antibodies, also known as immunoglobulins, are proteins produced by the immune system in order to protect the organism (body) from various foreign substances, including pathogens and toxins.¹²⁷ Serum therapies with an elevated content of antibodies to different infectious agents were first used for the treatment of infections caused by those agents in other individuals in the 1890s.^{128,129} At least three forms of antibody treatment have been utilized with good clinical effect on COVID patients. These forms are briefly discussed below.

Convalescent plasma

Convalescent plasma is collected from individuals who have mounted an immune defense and formed circulating antibodies as a result of recovery from an infectious disease. Depending on the severity of the COVID infection and the quality of the immune capacity in a given patient, the infusion of plasma harvested from recovered patients can be a highly effective treatment for other newly infected patients.

However, due to a number of factors, it cannot be assumed that all harvested plasma will be equally effective in the patients being treated. Nevertheless, when the viral load is sufficiently lowered, which is a common response, a clinical cure can sometimes be achieved. An improved survival rate has been documented in COVID patients who have received convalescent plasma therapy.^{130,131}

Monoclonal Antibodies

Along the same lines of reasoning as to why convalescent plasma is of benefit to COVID patients, the intravenous administration of monoclonal antibody preparations can be of substantial and prompt benefit as well. After their initial isolation from the blood of humans or animals that have mounted an immune response to a pathogen, special techniques allow for the production of concentrated amounts of these antibodies.¹³² Preparations of both a single monoclonal antibody or of two different combined monoclonal antibodies have significantly reduced COVID viral loads in patients.^{133,134} A similar result was seen in infected monkeys and hamsters.¹³⁵

Immunoglobulins

Intravenous immunoglobulins are harvested from a blood product pooled from many healthy donors. Generally speaking, these immunoglobulins represent the naturally circulating defenses of individuals who are not chronically ill and who have their own unique array of antibody and other immune responses to infections and conditions they have encountered

and overcome. Administration of these preparations have already proven to be of clear-cut benefit in COVID patients.^{136,137} Not surprisingly, the impact of such immunoglobulin therapy has much less to no significant impact when given too late in the course of COVID infection, as when the patient has already developed ARDS.

General Recommendations

Many different recommendations have been made with the intent of minimizing the chances of contracting COVID, as well as to minimize the clinical severity of the infection if a substantial enough virus exposure cannot be rebuffed. Based on the information already presented, it is important to make sure any supplementation regimen includes vitamin C, vitamin D, magnesium chloride, and zinc. Certainly, many other quality nutrient, vitamin, and mineral supplements can be taken that would offer even greater benefits for infection prevention/resolution. For many people, simple economics strongly limits what can be taken. These four foundational immune system-supporting supplements being recommended are also especially inexpensive.

Along with these four supplements, it cannot really overemphasized as to the contribution that nebulized HP can offer to prevent and/or cure not only COVID, but any respiratory viral syndrome, which includes all common colds and cases of influenza. Nebulizing for only a couple minutes per day can offer not only enormous protection against contracting respiratory viruses, it can also make an enormous contribution to normalizing the microbiome of the gut

and drastically reducing all variety of gastrointestinal disorders afflicting so much of the population. And it has also been shown how normalizing the gut microbiome positively impacts all chronic diseases.

When an infection has been contracted, it is always optimal to combine the HP nebulizations with IV vitamin C if possible, and/or ozone autohemotherapy and ultraviolet blood irradiation. All of these bio-oxidative therapies work very well together or as individual therapies. The HP nebulizations combined with IV vitamin C makes an especially beneficial partnering of therapies. The final “combination” of therapies for any given patient will ultimately depend on the knowledge and experience of the healthcare practitioner directing care, the availability of any given therapy, and sadly, whether a given therapy is even permitted in a certain hospital or clinical setting.

Recap

While an analysis of the politics of the COVID pandemic are beyond the scope of this book, it is nevertheless very important to realize that there are no valid medical reasons for not using any of the therapies discussed above for the prevention and/or treatment of COVID, or any other respiratory viral syndrome. None of these therapies interferes with the other, so there should be no hesitation to apply any combination of them that is readily available. Also remember that when a therapy is beneficial, or even just potentially beneficial, and it is both inexpensive and established to be safe, there is ***no reason or even justification*** for a physician to refuse or even actively block its administration. This is especially the case when a

COVID infection is progressing rapidly and has already compromised the ability to breathe.

To be as clear as possible, there is no good reason for anyone to die from or even suffer greatly from COVID if any of a variety of the treatment options discussed are properly applied. And with such options available as the HP nebulization, there is little reason to ever contract COVID or to allow it to get a foothold in the body.

HP Nebulization: Getting Started

This chapter provides a simple protocol for HP nebulization in the treatment of acute respiratory infections, the prevention of such infections, and a suggested maintenance protocol for keeping the aerodigestive tract free of CPC. There is also information on types of nebulizers and suggestions on getting started.

Nebulizer Types

There are two basic types of nebulizers: desktop jet nebulizers and small handheld mesh nebulizers. Both are effective for the delivery of HP but each type has its advantages. It is actually a good investment to have both units available for use.

The handheld version is very quiet and can deliver a finer, deeper-penetrating mist. This feature is especially important when dealing with established infections deep in the lungs. Cleaning a mesh nebulizer after misting with HP only requires a few moments of nebulizing with water after the session is complete, along with a gentle water rinse of both sides of the mesh. However, handheld mesh nebulizers are much

more prone to getting clogged when misting multiple dissolved agents and solutions with higher concentrations of those agents. When misting in such a manner, a prompt and very thorough cleaning is required.

Nebulizers are very common and inexpensive, usually under \$40, and a one-time purchase should be your only monetary outlay for the good health of your entire family.

Follow the instructions that come with the mesh nebulizer for a proper cleaning.

The jet nebulizer is better suited for aerosolization of various multi-solute and more concentrated solutions in the nebulization chamber that is connected to the air compressor with tubing. The nebulization chamber is separate from the jet nebulizing machine and is much easier to clear, using just requiring a hot water rinse.

Nebulizers are very common and inexpensive, usually under \$40, and a one-time purchase should be your only monetary outlay for the good health of your entire family. Various models are widely available on www.amazon.com, eBay, and elsewhere. The desktop jet nebulizers only run by plugging into a standard wall outlet. The handheld units offer a plug-in option or the use of non-rechargeable batteries. More expensive handheld versions can have a built-in rechargeable battery that can give as much as four hours of nebuliza-

tion, which can be a very convenient and useful feature, depending on your usage routine.

Generally, you will want to use the nebulizer face mask option rather than the mouthpiece option for your treatments. The face mask allows you to breath the mist into your nose, thereby accessing the sinuses and upper pharyngeal areas along with the throat and all of the respiratory tract. Even when you have “just” a sore throat, the face mask would still be preferable since the CPC will not just be limited to the throat area. However, the mouthpiece is of use when you have already done one or more full face mask nebulization, and you want to just target a symptomatic throat area with a higher concentration of HP or whatever solution you might be nebulizing. Generally, all of the nebulizers come with mask and mouthpiece attachments. However, when general family use is planned, it is a good idea to purchase additional attachments for everyone that might be using the nebulizer

Types of Hydrogen Peroxide

HP is available in varying strengths and quality grades. For most adults, regular OTC (over the counter) 3% HP is sufficient for nebulization therapy. Stronger concentrations than 3% are not recommended for nebulization. While there is no scientific evidence that purer grades of HP, such as food grade, are superior in safety or efficacy, it is perfectly acceptable and theoretically optimal to use such products. Food grade HP is free of any of the stabilizers found, although not routinely disclosed, on OTC preparations of HP. However, for those who wish to avoid any such additional agents, food grade HP is a good, albeit more

expensive, choice. However, for those individuals who have difficulty finding or affording the food grade forms of HP, the OTC form has not been shown to have any clinical toxicity, especially when viewed in the context of the conditions being treated or prevented, and the long-term benefits to general health seen with a regimen of nebulization.

Early Onset and Treatment of Virus

For most adults, regular OTC 3% concentration can be utilized in the nebulization chamber undiluted. This optimizes the degree and rapidity of antiviral and anti-pathogen effect. When a runny nose or slightly sore throat is already present, undergoing 10- to 15-minute nebulization sessions roughly four times daily or until a symptomatic relief is realized should be a good approach. Many individuals report significant improvement within a few hours (or less) after the first treatment. But it would be advisable to persist in these treatments several times daily for at least 24 to 48 hours after you feel everything is completely normal in your sinuses, nose, and throat. This helps to eliminate the possibility of a relapse of clinical infection and associated symptoms.

For some, the 3% concentration results in too much stinging/burning in the nose. Such individuals can dilute the HP with a normal saline solution—0.9% sodium chloride in water, or just water. However, the saline tends to have a more soothing effect on the throat and mucous membranes than the water. The goal is to utilize the highest easily tolerable HP concentration, while always staying below 3%. Nearly everybody can inhale a nebulization of a 50/50 combination

of the 3% HP and saline or water without difficulty. In fact, many people immediately feel a sense of relaxation and easier breathing when this is done. However, still lower concentrations can be utilized with clearly beneficial effect, although the benefits are realized more slowly.

Prevention/Maintenance

As it is a completely non-toxic therapy, nebulization can be administered as often as desired. If done on a daily basis at least once, a very positive impact on bowel and gut function will often be realized as killing the CPC present in most noses and throats stops the continual swallowing of these pathogens and their associated toxins. If daily prevention is not a desired or practical option, the effectiveness of this treatment is optimized when somebody sneezes in your face or you finally get off of the plane after a trans-Atlantic flight. Don't wait for initial symptoms. Just nebulize at your first opportunity. Dentists who face open mouths in close quarters for many hours at a time would be well-advised to take a brief (2 to 5 minutes) HP nebulization at the end of each work day. Any other profession or work environment that involves regular close contact with a large number of people would also be wise to take a brief nebulization at the end of the day.

For both the prevention of acute respiratory illness as well as for maintenance of a normal aerodigestive tract flora upon elimination of CPC, nebulization can be done as often as desired. Normally, nebulizing HP for 2 to 5 minutes once a week should be sufficient. However, any loosening of the stool during a time of maintenance without regular nebulizations is often

an indicator of a negative change in the microbiome. Whenever this is noticed, consideration should be given to resuming HP nebulization until normal bowel function is again seen.

HP Nebulization Protocol Chart

Feel free to copy this chart and place with your nebulizer. This is a rough guide only. Most individuals will eventually come to appreciate what routine or nebulization schedule best serves them.

Symptoms	Frequency	Duration
Early Onset of respiratory symptoms	3-4 times daily	10-15 minutes
Possible Exposure	Once (as soon as possible)	2-3 minutes
Loose stool after period of normal stool	3-4 times daily	10-15 minutes
Maintenance	Once per week	2-5 minutes

Tonsils: Focal Infection and CPC Heavyweights

Overview

Collectively, the tonsils are relatively large masses of secondary lymphoid tissue.¹ Some authors consider the tonsils to be variations of lymph nodes in terms of their physiological roles in the body. The “traditional” tonsils are known as the palatine tonsils, as they are adherent to the soft palate near the oropharynx and nasopharynx at either side of the back of the tongue in front of the throat. These tissue masses are the primary sites that get enlarged and/or chronically infected, mostly in children with severe and frequently recurrent tonsillitis, and that end up getting removed at tonsillectomy. Sometimes their removal is necessary to ease partially obstructed breathing as well.

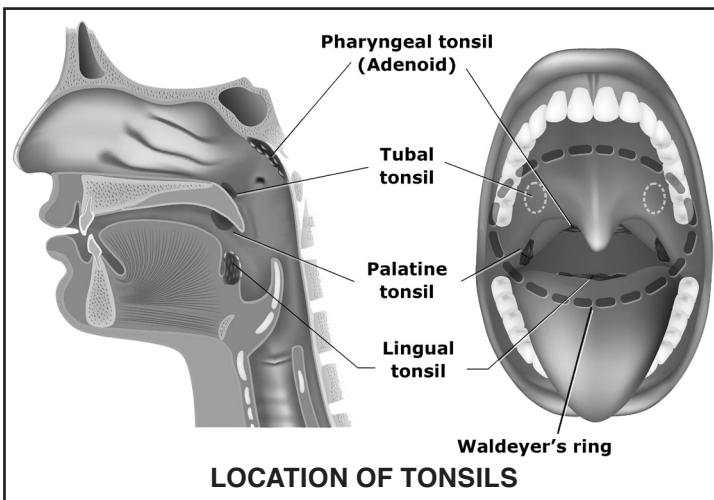
A smaller associated site of lymphoid tissue is on the back of the nasopharynx in the midline behind the nose (pharyngeal tonsil or adenoid). This tonsillar tissue is also commonly removed with the palatine

tonsils at the time of surgery (“tonsillectomy and adenoidectomy”). Another site of lymphoid tissue is the lingual tonsil, located far back on the base of the tongue, directly facing the back of the throat. A tonsillectomy can also involve the removal of this lingual tonsil, especially when it is partially obstructing free air flow.

Two less defined areas of tonsillar tissue are located on both sides of the mouth and extend between the palatine tonsils and the centrally located adenoid. Known as the tubal tonsils, they are embedded beneath the mucosa cells of the eustachian tubes as they approach the adenoid. Even though the tubal tonsils have a lessened density of tonsillar tissue relative to the palatine tonsils, they can also become inflamed and hypertrophied secondary to their proximity to inflamed and hypertrophied adenoid tonsillar tissue. The symptomatic manifestations of this hypertrophy can include obstructive air passage and sleep disorder, rhinosinusitis (runny nose and inflamed sinuses), and recurrent ear infections (otitis media). This tubal tonsillar hypertrophy is not uncommon, as it was found to be present in 10 of 24 patients evaluated for recurrent or persistent symptomatology after adenoidectomy.² There also exist even less concentrated areas of associated tonsillar/lymphoid tissue on the back of the throat (lateral pharyngeal bands and areas on the posterior pharyngeal wall).

When viewing the distribution of all these tonsillar structures together from an anatomical perspective, they effectively form a circle of lymphatic tissue that surrounds the passage of anything inhaled or ingested across the back of the mouth and into the throat. This

collection of tonsillar tissue is known as Waldeyer's ring. This ring configuration of the tonsillar tissue facilitates its role in minimizing the entry of pathogens, especially bacteria and viruses, into the body. There is also an encircling array of lymph nodes considered to be the outer ring of Waldeyer's. Nevertheless, this collection of lymphatic tissue in Waldeyer's ring and the associated lymph nodes are felt to be a relatively minor factors in the immune prevention of infection secondary to inhaled and ingested pathogens.³



Even today, many authors assert that the physiology of the tonsils, especially with relation to the immune system, remains poorly understood. A meta-analysis that reviewed 35 articles concluded that loss of the tonsils via tonsillectomy had no clinically significant negative effects on the immune system. However, the reviewed studies examined different combinations of immune parameters, and 4 of the

articles concluded that tonsillectomy actually did have some negative impact on the immune system.⁴

The presence of chronically infected teeth and/or gums eventually present the tonsils with more pathogens than can be completely processed, and chronically infected tonsils are the result. Once chronically infected and abscessed, the tonsils, especially the palatine tonsils, become an even ***more significant focal infection than the infections they drained***, even after those other foci of infections have been removed or resolved. Part of the tenacity and persistence of chronically infected tonsils is because biofilms protecting the acquired pathogens have been identified inside these tonsils. In one study on patients undergoing tonsillectomy, biofilms were found in 20 out of 20 tonsil specimens upon pathologic examination.⁵

This universal presence of biofilms in chronically infected tonsils helps explain why antibiotics never work with such infections, while ozone injections are highly effective.

Antibiotics are incapable of resolving biofilm-protected pathogens, while ozone is not only a premier anti-pathogen agent, it also readily destroys any biofilms as well.

The ozone treatment of infected tonsils is addressed in Chapter 14. A proper protocol of ozone tonsillar injections is so effective in resolving acute and chronic infections that the need for nearly all tonsillectomies would be virtually eliminated. Furthermore, any antibiotic-related side effects would be completely avoided as well, and the tonsils could once again resume their

protective immune-related role in dealing with inhaled and swallowed pathogens, however limited that might be. And, of course, the overwhelming negative clinical impact of the tonsils as a major focal infection seeding

*Once chronically infected and abscessed, the tonsils, especially the palatine tonsils, become an even **more significant focal infection than the infections they drained...***

pathogens and toxins throughout the body would be eliminated as well. Similar to HP nebulization, however, the time and money involved in providing ozone injections into the tonsils is inconsequential compared to the standard drugs and surgeries used by traditional medicine for dealing with acute and chronic tonsil infections.

It is important to appreciate that little to no pertinent research has been done on the normal-appearing but chronically infected tonsils found in so many people. The number of people around the world who have root canal-treated teeth, painless but chronically abscessed teeth, and also chronic periodontal disease is enormous. When any of these conditions have been present long enough, with the palatine tonsils (and other tonsillar tissue) draining the lymph and blood supply from the infected areas of the jawbone “24/7” the end-result of infected and chronically abscessed tonsils can simply be expected to develop nearly all of the time.

The best evidence indicating how common and how toxic chronically infected tonsils are comes from the work of the brilliant clinician, Josef Issels, MD.⁶ In 1951, Dr. Issels founded a hospital in Germany

As it turns out, the same conclusion can readily be drawn for the presence of such focal infections in nearly all heart attack patients with chronic coronary artery disease.

that specialized in the treatment of advanced cancer patients given up as incurable by traditional medicine after failing to respond to conventional cancer treatments. He ended up treating over 12,000 such patients over the course of several decades with his own unique and highly effective form of immunotherapy. He had a very high success rate with his overall treatment protocol, and he detailed many case reports in which patients with advanced metastatic cancers of various types were cured (or “put into remission” until death occurred from unrelated causes up to decades later).

Dr. Issels discussed the importance of bio-oxidative therapies, especially variations in the application of ozone, oxygen, and ultraviolet blood irradiation. He also fully realized the enormous negative health impact of focal infections, especially those situated in chronically infected teeth, including root canal-treated teeth. He also referred to such teeth as being dead or “devitalised.” When any tooth had what he termed

an “inflamed pulp” he advocated extraction “without delay.”

A survey of the adult cancer patients admitted to the Issels clinic found that 98% of them had between two and ten dead teeth. Considering the limited diagnostic capacities of dentistry many decades ago, the advent of 3D X-ray should readily demonstrate that this 98% figure is actually 100%. Conversely, such a statistic suggests that it is difficult to have advanced cancer without a mouthful of infected teeth.

It would appear, at the very least, that having a patient with advanced cancer with no oral cavity infections (including the tonsils) is extremely rare, from a statistical perspective. As it turns out, the same conclusion can readily be drawn for the presence of such focal infections in nearly all heart attack patients with chronic coronary artery disease. These relationships again underscore the fact that deep-seated infections are the strongest contributors to very high levels of oxidative stress throughout the body. And it is the chronic presence of such ***extremely elevated levels*** of oxidative stress that predispose to the development of cancer even more than the development of “regular” chronic disease, which reliably occurs at ***lower degrees of chronic elevation*** of oxidative stress in the body. All of the cancer patients that were put on the treatment protocol of Dr. Issels had all of their infected teeth removed, with a thorough cleaning and removal of any infection remaining at the extraction sites.

The presence and clinical significance of chronically infected tonsils in cancer patients first came to the attention of Dr. Issels in 1953. An advanced cancer patient at his clinic with severe rheumatic pains

Certainly, it can readily be asserted that addressing one and not the other [infected tonsils and infected teeth] will be a major shortcoming of any clinical protocol designed to alleviate and reverse any disease, including cancer.

and a long history of tonsillar disease had a tonsillectomy performed to help alleviate her chronic pain. The relation between infected tonsils and rheumatic pain had long been realized by the practitioners of integrative, non-traditional medicine. However, not only did her rheumatic pain disappear, but her “general toxic symptoms” promptly resolved as well. The cancer began to regress, and it ultimately resolved completely. This led Dr. Issels to have tonsillectomies performed on two more patients with chronic tonsillar disease and “therapy-resistant” cardiovascular disease. Prompt and dramatic clinical improvement was seen in both patients.

These experiences further led Dr. Issels to do tonsillectomies on a more frequent basis in his patients when tonsillar disease was suspected. He eventually made this surgery “virtually obligatory” in the treatment protocol of his patients. He noted that before this surgery became a routine part of his cancer protocol,

40% of the patients who died did so because of heart attacks. After the surgeries became routine, deaths due to heart attacks dropped to 5%.

In his discussion of the significance of tonsillar infections, Dr. Issels points out that chronically infected/inflamed tonsils can sometimes have greater negative clinical consequences than infected teeth. Certainly, it can readily be asserted that addressing one and not the other will be a major shortcoming of any clinical protocol designed to alleviate and reverse any disease, including cancer. Infected teeth and infected tonsils are always closely connected. Experiments have shown that an Indian ink injection into a sealed dental cavity promptly appears as spots on the tonsillar surface about 20 to 30 minutes later. The tonsils function to both detoxify and excrete pathogens with their related toxins from chronically infected teeth. However, when the pathogen exposure becomes too large and ongoing, the tonsils decompensate, become chronically infected, and the tonsil becomes its own focal infection.

Draining chronically infected teeth eventually transforms the tonsils from protectors to infectors. This means that the tonsils not only stop “detoxing” the infected teeth, they also become their own new and exceptionally efficient sites for the ongoing body-wide seeding of pathogens and toxins. This results in a doubly potent assault on the health of the body.

Chronically infected tonsils, such as were seen in the cancer patients of Dr. Issels, usually remain unad-

dressed by the physician or dentist. They not only appear healthy by external examination, they also often appear smaller than normal and **not** enlarged as might be expected with any ongoing state of chronic infection. A history of any symptoms typically associated with tonsillar infections is usually absent as well. It turns out that the chronic pathogen and toxin content in these tonsils ends up resulting in some degree of atrophy over a long enough period of time.

The tonsillectomies always followed the extraction of all infected, dead teeth in his patients. Dr. Issels asserted that **100%** of the extracted tonsils were found to have advanced pathological changes, including pus-filled abscesses and cysts, spongy and putrid-smelling tonsillar tissue, and an atrophy of lymphoid tissue. About 5% of the patients also had large peritonsillar or retrotonsillar abscesses that had been asymptomatic.

These normal-appearing but heavily infected tonsils should be regarded as:

The Tonsils of Chronic Disease

While these infected tonsils were always present in the cancer patients of Dr. Issels, it can logically be asserted that such infected tonsils will also be found in the oral cavity of all patients who have infected teeth or who have already had them extracted. As such, the elimination of such infective foci can be expected to improve the clinical status of any patient dealing with just about any chronic disease. And while tonsillectomy can always be expected to be of substantial clinical benefit to such a patient, ozone treatments of the tonsils are very reliable in eliminating or markedly

decreasing the pathogen and toxin load of such tonsils. Having a tonsillectomy as an adult is very traumatic, and it should be avoided if at all possible with a protocol of ozone injections. Furthermore, many

*...it is just as important to realize
that such infected tonsils that are
completely **normal in size and general
appearance** are very common.*

ozone-injected tonsils could end up healing enough to resuming an immunoprotective role against new pathogen exposure.

Discussed in more detail in Chapter 14 a protocol of direct tonsillar injections of ozone gas eliminates or substantially lessens the increased oxidative stress otherwise inflicted on the body by chronically infected tonsils. Such ozone injections, as they are technically simple, inexpensive, and requiring little time by the biological dentist, should be regularly be administered to patients at their routine dentist visits, especially when infected teeth are still present, and there is no plan to have them extracted. When done after infected teeth have been extracted and infected gums/periodontal tissue have been effectively treated, ozone injections need not be given so routinely. However, when there is any persistent evidence of increased oxidative stress throughout the body, several cycles of a more extensive ozone injection protocol into as much of Waldeyer's ring as possible should be done. There can be infected tonsillar tissue outside of the palatine

tonsils, and there can also be adjacent abscesses in the tissue spaces around the infected tonsils.

In addition to appreciating how consistently a good ozone injection protocol can normalize or near-normalize chronically infected tonsils, it is just as important to realize that such infected tonsils that are completely ***normal in size and general appearance*** are very common. Because infected tonsils often appear normal on examination, and the ozone injections are so simple and quick, it is a reasonable approach to have them periodically injected, probably at least once annually during a good oral health check or when tooth cleanings are scheduled, even when there is no longer any suspicion that they are chronically infected. For the biological dentist already using ozone treatments on a regular basis, the cost of the syringe and needle for injecting the ozone is more costly than the ozone itself.

It is also important to remember that infected tonsils can make a substantial contribution to the swallowed pathogens and toxins that promote an abnormal microbiome and leaky gut. CPC of the aerodigestive tract provides the greatest source of such pathogens and toxins, but infected tonsils do not just disseminate their “contents” only into draining blood and lymph circulations that reach the entire body. Rather, the tonsils can also be considered ***organs of excretion***, as pathogens and toxins are readily shed, or excreted, along with the debris and waste that forms and breaks off from any externalized areas of infection. This pathogen shedding would be more pronounced in the acute or semi-acutely infected tonsils rather than the small, normal-appearing but chronically infected

tonsils. Nevertheless, all infected tonsils can result in the swallowing of pathogens and toxins in addition to their dissemination internally via the blood and lymphatic circulations.

Recap

While there is substantial lack of agreement on how significant the palatine tonsils and the associated circle of tonsillar tissue (Waldeyer's ring) are in their ability to mount a significant immune defense against inhaled and ingested pathogens and toxins, they are very subject to becoming chronically infected and staying that way. And when they do become chronically infected, their impact on the development and evolution of most chronic diseases is often **profound** due to the continued release of pathogens and toxins into the blood and lymphatic circulations.

The treatment of any chronic disease should not only involve the removal of all infected dental foci, it must also involve a plan of treatment for chronically infected tonsils, even if they do not appear infected on physical examination. The effective treatment of such tonsils is achieved with a sequence of properly applied direct injections of ozone gas. Such tonsillar treatments should also be a routine part of any protocol for helping to achieve and maintain a normal microbiome, with the goal of eventual eradication of CPC in all areas and sites in the body.

Understanding the Cause of All Chronic Disease

Redox Biology and Oxidative Stress

Redox biology encompasses the interactions involved in the relay or transfer of electrons between molecules, particularly inside the cells.¹ Oxidation occurs when a biomolecule loses one or more electrons, and reduction occurs when a previously oxidized biomolecule regains its full complement of electrons. Biomolecules refer to the molecules that are produced by the body and intimately involved in the normal metabolic functions.² Such molecules include nucleic acids (RNA and DNA), lipids or fats, carbohydrates, proteins and enzymes, and structural molecules.

The physiological significance of the balance of oxidation and reduction in the body is profound, as an oxidized biomolecule loses all or most of its normal role, or biochemical function.³ It becomes a blank key, or a non-player. When a biomolecule is in its reduced form and is no longer electron-depleted, it fulfills its normal biochemical and physiological role. At a molecular level, then, the only “affliction” that any

biomolecule has in ANY medical condition or disease is whether it is oxidized or reduced. It does not possess any additional characteristics unique to the disease to which it is contributing.

But excess oxidation beyond that needed to signal or trigger normal metabolic pathways is also what actually defines the pathophysiology of any “diseased” cell or tissue.

Determining whether biomolecules, cells, tissues, or organs are healthy and not part of a disease is straightforward and simple: to what degree is oxidation present? Expanding on this, the nature and extent of a clinical disease depends on the following oxidation characteristics:

- ✓ **TYPE:** Which biomolecules are oxidized?
- ✓ **LOCATION:** Where are the oxidized biomolecules?
- ✓ **EXTENT:** To what degree is a certain class of biomolecules oxidized (minimal, moderate, advanced)?
- ✓ **TIME, CHRONICITY:** How long have the affected biomolecules been oxidized?

This leads to a simple but important distinction:

Disease is NOT some ill-defined state that is CAUSED by oxidative stress.

Rather, all disease IS the presence of the oxidized state, with no other abnormality or affliction being involved at the molecular level.

When the increased IOS (intracellular oxidative stress) is normalized in all of the affected cells, disease no longer exists.

The variations among different diseases are dictated entirely by the ***unique pattern or array of oxidized biomolecules***, as determined by the interplay and distribution of each of the four characteristics noted above.

Generally speaking, oxidative stress simply refers to an excessive presence of oxidized (electron-depleted) biomolecules relative to the presence of reduced (electron-filled) biomolecules.⁴ Since there is a continuous interplay between agents that oxidize and agents that reduce, there is always present some degree of oxidation in a normal cell or tissue, or in the extracellular space. And although all disease is a reflection of excess oxidative stress, oxidation is nevertheless essential for life, and a lower level of it works as a signaling factor to keep metabolic pathways functioning appropriately.^{5,6} This degree of oxidation in the cell is also referred to as a state of physiological oxidative stress, or “necessary” stress. But excess oxidation beyond that needed to signal or trigger normal metabolic pathways is also what actually defines the pathophysiology of any “diseased” cell or tissue. Optimal health requires an optimal balance between the opposing factors of oxidation and reduction.

Sources of Oxidative Stress

All oxidation is caused by pro-oxidant agents (toxins). These are molecules that cause, directly or indirectly, the loss of electrons from biomolecules. And once oxidized, these molecules can only be restored to their reduced, normal state by antioxidant agents. Antioxidants donate electrons to biomolecules that are depleted of electrons. To emphasize these points:

- ✓ **OXIDATION:** A pro-oxidant, or toxin, takes or causes to taken, electrons away from reduced (normal) biomolecules.
- ✓ **REDUCTION:** An antioxidant donates, or restores, electrons back to oxidized biomolecules. Vitamin C is the prototypical antioxidant in the body.
- ✓ **PRO-OXIDANTS** damage biomolecules.
- ✓ **ANTIOXIDANTS** repair biomolecules.

A lot of potentially confusing terminology is used in articles addressing redox biology. Quite simply, toxins, poisons, free radicals, and reactive oxygen species (ROS) all exert pro-oxidant effects on the biomolecules that they impact. All of these terms refer to agents that result in the oxidation of biomolecules.

Even though there is a tremendous variety in physical structure and chemical characteristics among all of the known toxins, they ALL SHARE the capacity to directly or indirectly oxidize biomolecules. In fact, if a molecule does not cause the loss of one or more electrons from another molecule, it IS NOT A TOXIN (or poison), and it does not have the ability to exert a toxic effect in the body.

An antioxidant, on the other hand, can also be referred to as a molecule with positive nutrient value. Good nutrition is ultimately defined by how effectively and completely anything that is eaten or other-

Optimal health requires an optimal balance between the opposing factors of oxidation and reduction.

wise assimilated into the body can be broken down to molecules with electron-donating capacity. At the molecular level, any molecule that depletes biomolecules of electrons is a toxin or poison, and any molecule that restores oxidized biomolecules back to a normal electron status is a nutrient.

Whether something is toxic or nutritious is determined SOLELY by its ultimate impact on the oxidation/reduction balance in the tissues. Keeping these concepts in mind is important in designing optimal treatment protocols for any medical condition.

The most clinically significant sources of toxins that promote disease states throughout the body are infections. Acute viral syndromes inflict toxicity throughout the body, and some acute hemorrhagic fevers, like Ebola, so completely consume the vitamin C stores in the body that a fatal body-wide scurvy can rapidly appear, causing the hemorrhage referred to in its designation.

However, the infections that play a prominent role in all chronic diseases are also chronic in nature, not acute. These are discussed at some length in Chapter

4. There is a documented presence of pathogens chronically colonizing the pertinent tissue and organs in some chronic diseases. Furthermore, the pathogens that have been isolated in these tissues are typically those found in oral infections/colonizations.

Many of these oral pathogens gain access to the rest of the body by direct expression into the venous blood and lymphatic circulation, as occurs when chewing with infected teeth. And many more gain access to the rest of the body as they are swallowed and then pass across the leaky gut that they help to create and sustain by the multiple oxidizing (toxic) agents associated with all pathogens. And this is further “multiplied” by the role that these swallowed pathogens play in causing an abnormal microbiome that produces its own pathogens

Additionally, these pathogens and toxins will feed any and all of the tissue sites of CPC that supply the ongoing new oxidative stress needed to sustain and worsen chronic diseases.

and toxins to sustain and worsen the leaky gut while gaining access across it to the rest of the body. As long as the CPC (chronic pathogen colonization) found in the aerodigestive tract of so many people is not eradicated, a huge “24/7” exposure to pathogens and toxins by the extensive interplay of factors addressed above will continue to disseminate body-wide.

Additionally, these pathogens and toxins will feed any and all of the tissue sites of CPC that supply the ongoing new oxidative stress needed to sustain and worsen chronic diseases. It is the ongoing, renewable supply of oxidative stress from CPC that causes so many diseases to largely remain unresponsive to treatment since new biomolecules continue to be oxidized and only rarely get returned to the healthy, reduced state.

If a medical condition was precipitated by a one-time, large toxin (pro-oxidant) exposure resulting in a relatively fixed amount of damage (oxidized biomolecules), a healthy diet with aggressive antioxidant and nutrient supplementation should be expected to steadily make that condition better, possibly to the point of complete resolution. However, such a complete resolution response to diet and supplementation is rarely seen. This is because all chronic diseases, even if not primarily precipitated by the seeding of pathogens and/or toxins in the tissues, will all be worsened and kept from complete clinical resolution by any steady pathogen/toxin exposure. This is why focal infection in the gums, teeth, and tonsils, along with chronic pathogen colonization in the aerodigestive tract and associated leaky gut with abnormal microbiome, must always be addressed. No disease can resolve with a continual shower of new pathogens and new toxins.

The basic foundations for the optimal therapy of any disease involves two basic approaches:

1. Preventing the exposure of diseased tissues to new toxins
2. Repairing the damage already inflicted by toxins

Traditional, “modern” medicine generally does not attempt to address either of these foundational approaches to the treatment of disease. There is little appreciation that toxins, mostly pathogen-generated, are continuing to cause the oxidation of more biomolecules in most diseases. And there is as little, or even less, appreciation that delivering targeted antioxidant therapies to diseased (oxidized) cells and tissues is the only way to ever achieve a true stabilization of a chronic disease, much less a reversal or resolution of it. Rather, the entirety of traditional medicine is mostly aimed at alleviating the symptoms associated with a disease, while leaving the continued progression of the disease itself unaddressed. Removing sites of chronic infection while optimizing the antioxidant status in the body simply does not generate the money needed to fuel the empire of modern medicine as it exists today.

Antioxidants are Antitoxins

A poison, any poison, is simply another toxin. And while the clinical manifestations of different poisonings can vary widely, the toxic identity of the offending poison means that all of the negative impact of the clinical poisoning is due to the degree and distribution of biomolecules that were poisoned (oxidized). Because of this, the optimal and most effective treatment of any poisoning is the prompt administration of very large amounts of antioxidants.

Vitamin C, as an effective antitoxin for almost every intoxication/poisoning imaginable, has been extensively documented in the medical and scientific literature, although you might never suspect it by the ways cases of clinical poisoning are treated in the emergency

room or the local poison control center.⁷ To date, no toxin or poison has been identified in the literature against which vitamin C has failed to have a substantial antitoxic impact. Examples of the extensive ability of vitamin C to neutralize, lessen, and even resolve the negative clinical and/or laboratory testing impact of diverse poisonings and intoxications include the following:

- ✓ Alcohol (ethanol)^{8,9}
- ✓ Barbiturates¹⁰⁻¹²
- ✓ Carbon monoxide¹¹⁻¹²
- ✓ Endotoxin¹³⁻¹⁵
- ✓ Cocaine¹⁶
- ✓ Acetaminophen¹⁷
- ✓ Acetaldehyde¹⁸
- ✓ Amphetamine¹⁹⁻²¹
- ✓ Benzene²²
- ✓ Carbon tetrachloride^{23,24}
- ✓ Cyanide^{25,26}
- ✓ Methanol²⁷
- ✓ Nicotine^{28,29}
- ✓ Paraquat^{30,31}
- ✓ Phencyclidine “PCP”³²
- ✓ Phenol^{33,34}
- ✓ Thallium³⁵
- ✓ Mushroom³⁶
- ✓ Pesticides^{37,38}
- ✓ Radiation^{39,40}
- ✓ Strychnine⁴¹
- ✓ Tetanus⁴²⁻⁴⁴
- ✓ Mercury^{45,46}
- ✓ Lead^{47,48}
- ✓ Chromium⁴⁹⁻⁵¹

- ✓ Arsenic⁵²
- ✓ Cadmium^{53,54}
- ✓ Nickel^{55,56}
- ✓ Aluminum^{57,58}
- ✓ Venoms [spider, snake]⁵⁹⁻⁶²

In the case of any acute poisoning, it is also extremely important to promptly administer magnesium. This is essential because an acutely prolonged QT interval (as documented on the electrocardiogram) can unpredictably proceed to a fatal arrhythmia at any time before the antioxidant capacity of vitamin C is able to neutralize the pro-oxidant stress causing and sustaining the prolongation. Such prolonged QT intervals can accompany the ingestion of a high enough quantity of **many different toxins**, including some prescription agents. Some of those agents will even affect the QT interval more promptly and specifically than others, as prolonged QT intervals may already be present in patients receiving therapeutic doses of psychotropic agents.^{63,64}

Magnesium has been shown to have the ability to promptly decrease and even normalize this abnormality, especially when precipitated by any acute toxin exposure.⁶⁵ And even if the QT interval is not prolonged, or an electrocardiogram cannot not be promptly obtained, the administration of magnesium along with vitamin C is still very synergistic in alleviating the potentially fatal increased oxidative stress induced by the poisoning.⁶⁶

Therefore, the first agents to be administered in **ANY** acute poisoning or toxin exposure, or with any overdose of a toxic agent are vitamin C and magnesium, intravenously if possible. Any other desired inter-

ventions can be done either at the same time or after the administration of vitamin C and magnesium. The important point is that vitamin C with magnesium

*Therefore, the first agents to be administered in **ANY** acute poisoning or toxin exposure, or with any overdose of a toxic agent are vitamin C and magnesium, intravenously if possible.*

is unquestionably the premier approach to neutralizing toxins, reversing their damage, and preventing the possibility of an early fatal arrhythmia. Additional measures should **NEVER** delay the administration of these two agents.

Vitamin C, Premier Antioxidant

Even though molecules with a full set of electrons and having the capability of donating them to oxidized biomolecules have antioxidant function by definition, all antioxidants are not remotely equivalent in their ability to reach and repair oxidized biomolecules. Antioxidant molecules have an almost endless variety of chemical characteristics. The same is true of the toxins that antioxidants neutralize. These characteristics, especially of toxins, include the following:

- ✓ Solubility (fat, water, both)
- ✓ Size (tiny to massive helps determine areas of physical access)

- ✓ Ionic charge versus electrically neutral (polar or nonpolar, often determines ease of access across cell membranes)
- ✓ Uniqueness of molecular structure
 1. Capability of substituting for another structural biomolecule
 2. Tendency to accumulate and physically impair the interaction of biomolecules
 3. Tendency to target specific biomolecules (oxidation of certain enzymes can have a great magnification of negative metabolic impact)
- ✓ Chemical stability (how readily electrons are acquired)
- ✓ Ease of excretion (how readily a molecule can be eliminated [chelation, sweating])

It is the combination of these and other characteristics that determine why one toxin oxidizes a certain array of biomolecules, and another toxin impacts a different array. This is why one toxin causes one disease and another toxin a different disease. If one toxin oxidizes an enzyme critical for metabolizing oxygen and producing ATP, it can kill in minutes (hydrogen cyanide inhalation). If it affects other less important biomolecules needed to immediately sustain life, it can accumulate and start producing a disease years later (mercury and multiple sclerosis). But the bottom line is that the only “toxicity” that is inflicted is due to the oxidation of biomolecules.

The basic redox nature of vitamin C and the pro-oxidant nature of all toxins concisely explains why vitamin C has been documented to be a nearly perfect

antitoxin against all toxins for which it has been tested, both *in vitro* and *in vivo*, and throughout all of nature. Plants, animals, and humans all require large amounts of vitamin C to neutralize the oxidative stress inflicted from any toxin as well as ongoing physiological oxidative stress. Highly-dosed vitamin C has been repeatedly documented to save an acutely poisoned or toxin-over-dosed patient, regardless of the toxic agent involved, including some prescription drugs.⁶⁷

Relative to other antioxidants, vitamin C is especially beneficial to the body. The main reasons for this include the following:

- ✓ It is a small molecule, similar to glucose in structure, and it will compete with glucose in utilizing the insulin-facilitated glucose transporters to reach the intracellular spaces. It can accumulate inside the cells of the body via both active and passive transport mechanisms. And due to this competition between glucose and vitamin C for intracellular access, it turns out that excess glucose presence (diabetes) results in especially low levels of vitamin C inside the cells of the body (intracellular scurvy).⁶⁸
- ✓ It can donate two electrons per molecule, whereas most antioxidants are limited to one electron donation from each molecule.
- ✓ It has a one-electron-depleted intermediate stage, ascorbyl radical, that is relatively stable and capable of subsequently promoting either oxidation or reduction,

depending on the demands of the microenvironment in which it is found.^{69,70}

- ✓ It is concentrated in very high amounts (up to 80-fold greater than the circulating blood) in monocytes and also substantially elevated in platelets and phagocytic cells.⁷¹⁻⁷³
- ✓ It is highly concentrated in the central nervous system (meaning the blood-brain barrier does not prevent access to the brain).^{74,75}
- ✓ Its sufficient presence inside the cells permits a rapid enough relay of electrons between molecules to help generate healthy microcurrents and transmembrane potentials (millivolts).
- ✓ All infections consume (oxidize) vitamin C, and the presence of any chronic infection assures some degree of vitamin C deficiency unless adequate supplementation is being taken. Other antioxidants can only partially compensate for this vitamin C deficiency, and only to a very limited degree. The very common presence of CPC throughout the body only further increases the need for vitamin C intake and not just antioxidants in general.

Intracellular Oxidative Stress (IOS)

While excess oxidative stress is always characteristic of some degree of pathology, it is increased intracellular oxidative stress (increased IOS) that is the truest tracker/marker of disease severity in a given condition. All chronic diseases feature increased IOS.

When increased IOS is brought back to a physiological amount of IOS (a normal redox balance), you once again have a normal cell. And when enough cells have normalized their redox balance, you have a normal tissue, or organ. And when all cells have normal levels of oxidation, you also secondarily correct any degrees of excess oxidation that might have been present in the extracellular spaces. Conceptually, the oxidative foundation of disease is straightforward. However, clinically resolving any area of excess oxidation is much more involved and complex than understanding its origin.

The primary markers/determinants of increased IOS are the intracellular levels of:

- ✓ Calcium
- ✓ Magnesium
- ✓ Vitamin C
- ✓ Glutathione

Calcium

The intracellular calcium concentration serves as both an indicator of increased IOS while also serving as the primary cause of increased IOS. The higher the calcium level, the greater the IOS. When the level of calcium can be brought back to normal, there is no longer excessive IOS, and the cell has been restored to a functionally normal status. Because of this,

Lowering elevated intracellular calcium levels is the most straightforward way to lower increased IOS and positively impact all disease processes.

It has been demonstrated in the literature that increased calcium intake by both supplemental and

dietary sources dramatically increases the chances of death from any disease (all-cause mortality).⁷⁶ Also, calcium supplementation alone has been shown to significantly increase the risk of heart attack.^{77,78} The body-wide toxicity of chronic excess calcium has been well-documented.⁷⁹

Magnesium

When intracellular calcium levels are high, intracellular magnesium levels are low. As more magnesium goes into the cell, less calcium is able to accumulate in the cells. Magnesium is both a natural calcium channel blocker and a general calcium metabolism antagonist. The ability of magnesium to block the calcium channels in the cell membranes and lessen calcium uptake into the cells is likely the single most important reason for the enormous positive health impact seen with magnesium supplementation. When intracellular magnesium levels are restored to normal, calcium levels have also been normalized, and there is no longer any increased IOS. This also means that such a cell is now physiologically normal, and when enough previously abnormal cells have normalized their previous oxidized state, there is no longer any disease present. And just as extra calcium increases all-cause mortality, more magnesium intake decreases all-cause mortality.⁸⁰⁻⁸²

Vitamin C

When intracellular calcium levels are high and intracellular magnesium levels are low, intracellular vitamin C levels will always be low as well. However, the more vitamin C that gets effectively delivered

inside cells, the easier it is for magnesium levels to rise while calcium levels drop. There is always an ongoing dynamic interplay between calcium, magnesium, and vitamin C as they jockey for a greater presence inside the cells. When the increase in IOS is severe, vitamin C levels inside the cells are markedly depressed, and a state of intracellular scurvy then exists.

Glutathione

Glutathione is the most concentrated and physiologically important of intracellular antioxidants. However, most of the glutathione is synthesized inside the cells rather than coming directly from food or supplements. And as long as there is a state of significantly increased IOS due to high calcium along with low magnesium and vitamin C inside the cells, the enzymes needed to synthesize glutathione will remain in an oxidized, inactive state, unable to keep glutathione levels normal. As such, normal glutathione metabolism inside the cells can never exist, regardless of diet or supplementation, as long as calcium and magnesium levels are unbalanced. Vitamin C supplementation can help a lot to increase glutathione in the cells, but it will always fall short until the primary insult of excess intracellular calcium is remedied.

Important factors that directly impact intracellular levels of calcium, magnesium, and vitamin C include hormones. Conceptually, all hormones share two basic purposes:

- ✓ They positively impact and accelerate normal metabolic pathways, making metabolic function much more efficient.
- ✓ They work to lessen increased IOS.

Even though different hormones have widely differing biochemical impact, they all share these two important functions. Hormones that are especially impactful on IOS include insulin, hydrocortisone, thyroid hormone, and the sex hormones. Any clinical intervention for any medical condition will be rendered less effective when there are deficiencies of these hormones. Keeping these hormones in balance also makes it much easier to optimize intracellular levels of vitamin C and magnesium. Vigorous supplementation with magnesium and vitamin C is always beneficial. However, their physiology is intimately intertwined with these hormones, and IOS will only optimize when all of these agents are in balance.

Estrogen

Estrogen serves as a calcium channel blocker.⁸³ It also serves to effectively raise intracellular magnesium levels⁸⁴ This is further supported by the fact that estrogen also serves to minimize magnesium wasting in the urine with increased renal tubular reabsorption.⁸⁵ Consistent with these positive impacts on intracellular calcium and magnesium levels, estrogen is also regarded as a powerful anti-inflammatory agent.⁸⁶ Resolving estrogen deficiencies dramatically decreases all-cause mortality, which more than justifies the administration of estrogen in virtually all deficient patients regardless of any other considerations.⁸⁷

Testosterone

Testosterone also serves as a calcium channel blocker, and it is also regarded as a powerful anti-in-

flammatiory agent.^{88,89} It has also been shown to lessen the laboratory abnormalities seen in metabolic syndrome. As with estrogen deficiencies, resolving testosterone deficiencies decrease all-cause mortality.⁹⁰ Testosterone lessens insulin resistance, likely due to facilitated cellular magnesium uptake.⁹¹

Insulin

Insulin has been clearly demonstrated to be a powerful healing agent in the scientific literature, even though nearly all the attention given it has been with regard to its vital impact on the metabolism of blood glucose.⁹²⁻⁹⁴ This healing effect of insulin is readily explained by its ability to minimize IOS, as it directly promotes the cellular uptake of both magnesium and vitamin C.^{95,96} The ability to increase needed magnesium uptake is especially important in light of the fact that the intracellular space always has a much higher concentration of magnesium than the extracellular space, a gradient that otherwise resists the uptake of more magnesium inside the cells. Insulin also serves to conserve magnesium by minimizing its excretion in the urine.⁹⁷ Of note, insulin resistance almost never sees a significant clinical improvement in the absence of good magnesium supplementation, demonstrating further the important interplay between these two agents.

Hydrocortisone

The properties of hydrocortisone also center around the normalization of increased IOS. Hydrocortisone serves as a calcium channel blocker, which is the most direct way to normalize IOS and

restore a cell to health.^{98,99} When cellular calcium levels drop, all of the other factors impacting IOS are much easier to manage. In addition to this direct effect on intracellular calcium, hydrocortisone also greatly enhances vitamin C uptake into the cell. Most likely, it is these two properties of hydrocortisone that are the primary reasons that hydrocortisone is such a potent anti-inflammatory agent. Inflammation effectively equals oxidative stress, and lowering calcium (which increases magnesium) while also increasing vitamin C inside the cells are the most direct ways to remediate it.

Thyroid Hormone

Of all the hormones, thyroid hormone is probably the most powerful at impacting IOS. Even seemingly minor thyroid hormone deficiencies (subclinical hypothyroidism) that are not apparent with traditional thyroid function blood testing have major impact on magnesium levels and IOS in all the cells of the body.¹⁰⁰⁻¹⁰²

In hypothyroid animals thyroid treatment has been shown to normalize intracellular magnesium levels. It is probably this effect of thyroid hormone on cellular magnesium levels that is the primary reason for its impact in minimizing IOS. Hyperthyroidism actually results in excess magnesium inside the cells.¹⁰³ Consistent with the reciprocal relationship between calcium and magnesium inside the cells, it has also been shown that intracellular calcium is increased in the presence of the hypothyroid state, and that is also correctable by the administration of thyroid hormone.^{104,105} Not surprisingly, even slightly decreased thyroid function is associated with increased all-cause

mortality.¹⁰⁶ Testing for and treating subclinical hypothyroidism is addressed elsewhere.¹⁰⁷

Because of the associated increased IOS, addressing subclinical hypothyroidism is also very important in order to lessen the susceptibility of the body to contracting infections. The seemingly minimal deficiency of thyroid hormone seen in this condition strongly increases the susceptibility to contracting infectious diseases. Also, this state greatly enhances the ability of focal infections, as seen in infected teeth, gums, and tonsils, to spread in a metastatic-like fashion throughout the body. Most noteworthy is the seeming ease of colonizing the coronary artery linings with oral pathogens and initiating atherosclerosis when thyroid function is depressed.^{108,109} And in an analogous fashion, this same state of thyroid deficiency strongly supports the metastatic spread of cancers that would otherwise stay put when thyroid hormone levels are normal. Put another way:

Increased IOS promotes and supports the evolution of all pathology, especially the seeding of infections. Optimal thyroid status is the most powerful way to normalize IOS and to prevent the dissemination of focal infections.

Primary Factors in Normalizing IOS

To recap, the normalization of IOS requires the elimination/minimization of new toxin exposure while repairing old toxin (pro-oxidant) damage. This is achieved primarily by:

- ✓ Optimizing thyroid function
- ✓ Optimizing sex hormone status
- ✓ Optimal supplementation
- ✓ Eliminating/minimizing focal infection and chronic pathogen colonization (CPC)
- ✓ Optimizing digestion

All of these factors must be addressed **in concert** to achieve the best clinical response and potential return to good health when disease is already present. Certainly, paying attention to these factors before the onset of disease will be even more effective when just looking to sustain good health.

After addressing any thyroid and/or sex hormone deficiencies and adhering to a regimen of optimal supplementation, the focal infection/CPC/digestion factors can only really be addressed together, since these factors are intimately intertwined. The following points highlight the interrelationship:

- ✓ Infected gums, teeth, and tonsils
 1. Release pathogens and toxins directly into the venous blood drainage.
 2. Release pathogens and toxins directly into the lymphatic drainage.
 3. Permits the continual swallowing of pathogens and toxins from these sources, which inflames the gut and causes leaky gut, infects and alters the microbiome, and directly initiates some cancers.
 4. Gives access to its swallowed pathogens and toxins into the blood and lymphatics surrounding the gut via

access across the leaky gut that they helped to cause.

✓ Chronic pathogen colonization (CPC) in the **aerodigestive tract**

1. Permits the continual swallowing of pathogens and toxins from these sources, which inflames the gut and causes leaky gut, infects and alters the microbiome, and directly initiates some cancers.
2. Gives access to its swallowed pathogens and toxins into the blood and lymphatics, surrounding the gut via access across the leaky gut that they helped to cause.
3. Lowers the natural immune defenses against potential new acute respiratory infections, keeping the mucosal and epithelial cells in a state of chronically increased IOS.

✓ Chronic pathogen colonization (CPC) **throughout the body** is initiated and sustained via leaky gut access and via blood and lymphatic access from oral cavity focal infections. Once seeded in various tissues throughout the body, these new areas of CPC will be self-sustaining unless and until the focal infections and the aerodigestive CPC sites are eradicated and both the leaky gut and microbiome are healed.

✓ A pathogen-overgrown microbiome, initiated and sustained by the above factors, produces its own “new” pathogens

and toxins that can cause all known digestive disorders and also cross the leaky gut into the rest of the body. This includes all forms of chronic inflammatory gastrointestinal disorders, including gluten sensitivity, celiac sprue, Crohn's disease, and chronic ulcerative colitis. Constipation, diarrhea, indigestion, and heartburn can also occur with a leaky gut and abnormal microbiome. There really is no symptom of digestive disorder that is not caused or worsened by the underlying problem of old and new pathogens and toxins, and the leaky gut that results.

- ✓ Poor digestion can both **cause or be caused by** a leaky gut and abnormal microbiome. As such, the presence of an abnormal gut and poor digestion always coexist. Incomplete digestion, fueled by poor food combinations, will severely slow gut transit time, resulting in constipation and food putrefaction, along with the proliferation of toxin-producing anaerobic bacteria such as *Clostridia* locally in the gut.

And even though all of these factors work together to sustain a given chronic disease, the eradication of oral focal infections and CPC in the aerodigestive tract is nevertheless the major intervention needed to improve or even normalize the clinical status of a patient. This again highlights the incredible clinical impact of periodic HP nebulizations. The protocol needed to address this is explained in Chapter 11.

Effective Treatment and Resolution of Chronic Diseases — Practical Bio-Oxidative Therapies

The main point of this book is to make the public aware, as rapidly as possible, that the COVID pandemic can literally be ended in one to two weeks with the widespread dissemination and application of the proper therapies. For reasons given earlier, HP nebulization is the single most important of these therapies, due to:

- ✓ insignificant expense,
- ✓ easy access and availability,
- ✓ lack of toxicity
- ✓ breakdown by-products (water, oxygen) that enhance healing,
- ✓ ease of administration,
- ✓ ability to be utilized without mandatory hospital, clinic, and/or physician intervention,

- ✓ an incredibly powerful positive clinical impact as a monotherapy, and
- ✓ clinical compatibility and synergy with other therapies

However, multiple clinical interventions for COVID and other respiratory viruses should always be implemented along with HP nebulization whenever available. In particular, the vigorous administration of vitamin C is ***always*** indicated for at least two important reasons. Vitamin C is strongly synergistic with the anti-pathogen impact of HP, helping sustain the Fenton reaction until the pathogen/host cell is destroyed. Also, rapidly evolving viral infections can quickly put the patient into an extreme degree of body-wide vitamin C deficiency, even to the point of acutely-induced scurvy. Such a state of vitamin C deficiency that is left unaddressed, or not addressed promptly enough, can end up:

- ✓ prolonging the time to clinical recovery from the infection
- ✓ increasing the suffering and morbidity of the infection greatly
- ✓ increasing the chances of death from the infection
- ✓ increasing the degree of residual oxidative tissue damage and long-term health impairment after the acute infection has been resolved, including susceptibility to new infections and new medical conditions, and a worsening of pre-existing medical conditions (for the pandemic, “long-haul COVID”)

Collectively, the most effective therapies against any infection, but especially against viral infections, are the bio-oxidative therapies. Discussed earlier, these

Also, rapidly evolving viral infections can quickly put the patient into an extreme degree of body-wide vitamin C deficiency, even to the point of acutely-induced scurvy.

therapies include HP, vitamin C, ozone, ultraviolet blood irradiation, and hyperbaric oxygen treatment. There are also a significant number of different ways in which these therapies can be administered for optimal clinical impact, especially with HP, vitamin C, and ozone. However, until a patient is clearly responding and resolving their viral infection, there should also be no hesitancy to utilize effective anti-COVID prescription agents, such as hydroxychloroquine, chloroquine, and ivermectin. One positive consequence of the pandemic is that it has brought to light for many people the fact that respiratory viruses can be readily prevented or cured, even if those remedies continue to be disparaged and ignored, or even suppressed.

The primary and secondary impact of chronic pathogen colonization (CPC), in tandem with chronic focal infections in the oral cavity and aero digestive tract, can cause and exacerbate many of the chronic diseases that are considered incurable and even unresponsive to traditional therapies. Recent studies support the position that HP nebulization as well as

other clinical treatments can quickly and completely end the pandemic.¹ Although that is the primary message of this book, many of these treatments can also lessen and **even eliminate** some chronic diseases due to CPC and focal infections.

HP nebulization is the **most important** of all the therapies required to eliminate CPC and to lessen CPC impact on the gut's microbiome and the body-wide seeding of pathogens and toxins via a leaky gut. That means that while HP nebulization is resolving a cold, flu, or COVID, it is simultaneously providing the conditions that will allow the gut flora as well as gut function to return to normal, leading to the healing of a leaky gut. As the leaky gut is resolved, easy dissemination of pathogens and toxins to the rest of the body is prevented which results in an improvement, and sometimes the disappearance of the chronic diseases these pathogens and toxins cause and worsen.

General Approach for the Improvement of Chronic Disease

The premises of this book center on the fact that every chronic disease results from the existence of a unique array of oxidized biomolecules in the affected tissues or organs. No excess oxidation, no disease. As all toxins exert their toxicity through oxidation, and nearly all **clinically significant** toxin sources originate from sites of pathogen growth, it follows that any protocol for the effective reversal or control of chronic diseases must involve two basic treatment approaches:

1. New toxin exposure and new biomolecule oxidation must be stopped or curtailed.

2. Oxidized biomolecules must be reduced and returned to normal physiological function as completely as possible with increased antioxidant presence.

Stopping the production of new toxins

In order to substantially slow or stop the production of new toxins, focal sites of concentrated infection (infected gums, teeth, tongue, and tonsils) must be addressed, and larger areas of less concentrated chronic pathogen colonization (CPC) must also be resolved or minimized. The CPC in the oral cavity, throat, and upper respiratory tract is most readily addressed. Depending on the degree of positive clinical response, consideration should also be given to a more extensive treatment protocol that can help eradicate or reduce the pathogen burden now known to have been seeded from these infections in many of the affected tissues of different diseases.

Not everybody will want to or be able to follow all of these recommendations for stopping new toxin exposure. Nevertheless, just following some of the suggestions will be vastly better for general health than ignoring them all out of the frustration that doing everything seems impossible.

Many people will feel noticeably better in general with regard to the frequency and degree of bothersome symptoms associated with different medical conditions. Also, the monitoring of serial blood testing can be relied upon to see if less pathogens and toxins are being released into the body. In particular, a decline or normalization of a pre-existing and chronically elevated C-reactive protein level is an excellent indi-

cator that the measures taken are having a significant positive impact on the entire body. The improvement of any other previously abnormal blood test results also supports this conclusion. Similarly, improvement in any other diagnostic testing (scans, X-rays, etc.) shows that the right approach is being taken, offering important psychological support to the patient that the recommendations are worth following for life.

In order to stop the production of new toxin exposure coming from the body, as many of the following conditions must be resolved as possible:

- ✓ Focal infection resolution
- ✓ Acute respiratory tract infection resolution
- ✓ Chronic pathogen colonization (CPC) resolution in the aerodigestive tract
- ✓ Normalization of an abnormal gut microbiome with healing of a leaky gut
- ✓ Resolution of CPC in the affected tissues of various diseases

General Approach for Discovering and Treating Focal Infections

Addressed at great length elsewhere, the diagnosis of focal infection requires substantial effort, as such areas of infection nearly always consist of **pain-free** chronic abscesses/infections in root canal-treated teeth, in untreated but structurally damaged teeth, in the sinuses, in the tonsils and associated tonsillar tissue, and in the gums.²⁻⁴ Some CPC is also addressed in this context, since even focal infections with chronic abscesses also have the biofilm seen in areas of CPC that have lower concentrations of pathogens and toxins.

Gums:

The entirety of the tissue surrounding and supporting the teeth is the periodontium, and when it is chronically inflamed and infected, oral pathogens and their associated toxins readily disseminate

Antibodies to many of the common periodontal pathogens have been detected in blood of patients with many of these medical conditions as well.

throughout the body. Technically the gums are the most superficial aspect of the periodontium, but they always appear clearly diseased when the underlying periodontal tissue is infected as well. Known as periodontitis when infected, the extensive documentation establishing the connection of this condition with a large number of different medical conditions is found in Chapter 6. Antibodies to many of the common periodontal pathogens have been detected in blood of patients with many of these medical conditions as well, and a number of these same pathogens have also been detected in the affected tissues of chronic diseases (see Chapter 4).

While it is perfectly fine for someone to seek professional periodontal care, it should only occur after taking some simple, highly effective measures to treat the gums/periodontal tissue first. These measures can often resolve periodontal disease completely, even when already advanced in degree. Regular irrigation of

the gums under pressure, as with a Waterpik® machine, is very effective in not only flushing out the impacted and residual food particles from recent meals, but also in cleaning out areas of infection that go deeper below the gum line.

Three percent (3%) HP, 15 to 30 ml added to 100 to 200 ml of water in the holding tank, is an excellent solution to use for regular irrigation of the gums. The warmer the solution, the better. A splash of a favorite mouthwash for improving the taste of the solution is perfectly acceptable as well. Early on, significantly diseased gums will readily bleed at even the lowest of pressure settings. This is to be expected, and most individuals who irrigate at least a couple of times a day will quickly tolerate higher pressure settings. For many people, the gums recover so quickly that maximal pressures can be readily tolerated without bleeding after only a few weeks of irrigation sessions. However, as long as bleeding is easily induced, there should be no urgency to go to higher pressures until the gums clearly start to heal.

Of note, regular flossing is NOT being recommended. Not only do many people damage their gums in a recurrent manner by utilizing a poor flossing technique, water irrigation routinely flushes out all impacted food particles much more thoroughly than can be eliminated with flossing. Just as with the body in general, it is much more natural and effective to “wash” soiled areas rather than pick at them in a mechanical fashion, as can be analogized with irrigation versus flossing. For the skeptic: irrigate and then floss. On another occasion: floss and then irrigate. It will quickly be realized that flossing does not remove

hidden debris that has not already been removed by irrigation, whereas irrigation leaves behind nothing for the flossing to remove.

Of note: smokers **ALWAYS** have chronically diseased gums and variable degrees of periodontitis. The protocols being discussed will help the gums of the smoker, but they will never resolve the underlying infection completely as long as smoking continues to any degree. But certainly, having three cigarettes daily will have a far less negative gum impact than three packs a day. Remember that the reason smoking has a strong relationship to heart disease is that it **ALWAYS** causes and sustains the periodontitis that continually seeds the coronary artery linings with oral pathogens and their associated antioxidant-consuming toxins.

Chronically Abscessed Teeth:

3D cone beam imaging, also commonly known as 3D X-ray, readily diagnoses virtually all abscesses present at the root tips of the teeth when it is properly performed and interpreted. Such abscesses are extremely harmful to general health, and the increased body-wide inflammation resulting from their presence precipitates many diseases and worsens all diseases. In particular, they result in a great deal of heart disease and cancer, especially breast cancer. But all diseases are negatively impacted. It is just that their role in causing heart disease and breast cancer should be especially appreciated, as these two conditions account for over half of the deaths on the planet annually.⁴

In order to optimize the chances of having good long-term health or at least minimally problematic medical conditions, all infected teeth must be extracted

by an experienced biological dentist. This **includes root canal-treated teeth**, which all always infected with necrotic (dead) pulps. Specific expertise is required to see that the remaining infected tissue in and around the tooth sockets are properly cleaned

Two to five grams of liposome-encapsulated vitamin C, taken daily for several days before, the day of, and several days after the extraction of the infected tooth is a good approach.

of residual infection, along with the removal of any portions of the periodontal ligament remaining in place after the teeth are removed. The presence of residual tissue infection and periodontal ligament remnants pretty much assures the sockets will remain chronically infected, making incomplete healing and cavitation formation very likely. Optimal biological dentistry also utilizes platelet-rich plasma and multiple applications of ozone to optimize rapid healing and minimize any chances of long-term chronic infection.

While not commonly available even among biological dentists, the administration of intravenous vitamin C further assures an optimal long-term post-extraction outcome. However, taking oral vitamin C, especially in a quality liposome-encapsulated form, is also very effective in achieving the optimal post-surgical healing of the extraction site. Two to five grams of liposome-encapsulated vitamin C, taken daily for several days before, the day of, and

several days after the extraction of the infected tooth is a good approach. More vitamin C can be taken for a longer period of time if desired, as vitamin C is very difficult to overdo in the context of supporting good general health. Some degree of daily vitamin C supplementation should be done for life.

Tongue:

Tongue cleaning is simple but important. Discussed earlier, the surface of the tongue is a major site of CPC in very many people, sometimes to the point of development of tongue-related medical conditions, such as geographic tongue and various manifestations of tongue inflammation and infection. Tongue cleaning, therefore, is serving the health of the tongue as well as the rest of the body. And while cleaning the tongue occasionally is better than no cleaning at all, the optimal benefits of this procedure are only realized with at least daily cleaning/scraping. A quick scraping before each tooth brushing is optimal, but if it is to be performed once daily, it is best to do it directly before going to bed at night.

While it is always of benefit to brush the surface of the tongue as part of a tooth brushing routine, it should not displace a concerted scraping of the tongue with a tongue scraper. While either order is acceptable, it is probably optimal to complete the scraping of the tongue first (taking 10 to 15 seconds) and then proceeding with tooth brushing followed by a quick tongue brushing (another 10 to 15 seconds). Any of a number of antiseptic mouth rinsing/gargling agents can then complete this session of oral cavity hygiene (which can also include HP or agents with HP). Much

of what goes on with CPC in the tongue feeds chronic periodontal disease. Addressing this hygiene in this manner can provide for a much more complete resolution of periodontitis as well, since fewer pathogens in the mouth lessen the chances of re-infection or support of ongoing infection in the gum tissue.

Tonsils:

As discussed in Chapter 12, the tonsils are almost always chronically infected with multiple internal pockets of pus after being in an oral cavity containing chronically infected teeth and gums for a long enough period of time. Furthermore, the fact that such infected tonsils rarely have an abnormal exterior appearance upon oral examination makes their contribution to chronic diseases especially insidious and still largely unappreciated. Such tonsillar infection is a factor that very frequently is playing a leading role in all chronic disease, but especially in heart disease. Also, the “diabolical” nature of this hidden infection is that these seemingly-normal tonsils appear virtually ***incapable of healing themselves*** once chronically abscessed, even after appropriate extractions of infected teeth and healing of infected gums have taken place.

Throughout the history of medicine, the only approach to chronically infected tonsils was surgical removal. This occurred especially in the context of young children and even early teens who maintained large, grossly infected tonsils that only resolved minimally between bouts of acute tonsillitis. Oftentimes the indication was to help breathing since sufficient tonsillar enlargement would impair ease of respiration.

The injection of ozone gas directly into the tonsils is a very effective way of healing or at least debulking the infection load in both acutely infected tonsils as well as in the normal-appearing but chronically

*...the “diabolical” nature of this hidden infection is that these seemingly-normal tonsils appear virtually **incapable of healing themselves** once chronically abscessed...*

infected tonsils just described. It is a simple procedure and takes only a few minutes per tonsil in an ozone-equipped biological dentist office.

An unpublished clinical study in Cuba conducted over an 8-year period enrolled 2,300 patients with a diagnosis of tonsillitis. Injections containing 5 cc of ozone at a concentration of 50 gamma were given in these tonsils. This was repeated for a total of 4 injection sessions. A number of different antioxidant and pro-oxidant markers were measured in all patients, and all of these markers dramatically normalized. No clinical relapses or recurrent tonsillar infections were seen over a follow-up period of a year. The author concluded that outstanding results were seen in 97% of the patients. Such a therapy can virtually eliminate the surgical indications for tonsillectomy.

Anecdotally, a 69-year male came down with an acute tonsillitis, with massive swelling of both tonsils and the classical external appearance of foci of pus over the surface of the tonsils. One of the tonsils enlarged to

the point of partially obstructing the throat and inhibiting the normal ability to breathe easily. A biological dentist injected multiple sites in the tonsils with a 50 gamma concentration of ozone. The clinical impact was so dramatic that the patient noted increased ease of respiration by the time he left the dental office. Several more injection sessions over the next few weeks facilitated a complete healing of the tonsils, with a normal size and appearance, along with a return to a normal CRP blood test from a high level recorded before the tonsillar injections.

The International Scientific Committee of Ozone Therapy has published a recommended protocol for the injection of infected tonsils with ozone gas (<https://isco3.org/>). This protocol should be primarily regarded as a general guide, as dentists experienced in the use and injection of ozone can vary the ozone dose and other aspects of this protocol. The local treatment consists of the following:

1. Use a needle of 30 gauge 1 and 1/2 inches in length
2. Ozone concentrations between 10 to 20 gamma with a volume of 2.5 ml per injection point
3. Inject at the anterior and rear pillars of both tonsils

Depending on the preferences of the dentist and the clinical/tissue response seen, more injections can be given in each tonsil, especially if substantial enlargement is present. Concentrations of ozone can be up to about 50 gamma, as was noted to have been done in the large Cuba study just described above.

For the normal-appearing yet chronically infected tonsils, consideration should be given to weekly injections for about a month. While not always present, the lowering of an elevated CRP level is a strong indicator

The clinical impact was so dramatic that the patient noted increased ease of respiration by the time he left the dental office.

that chronic infection in the tonsils is being alleviated or resolved. If there is suspicion of residual infection or a CRP test does not normalize completely, consideration can be given to slightly larger volume injections with higher ozone concentrations, while injecting in different areas of the tonsils at each treatment session. As noted directly above with “regular” tonsillitis, intratonsillar ozone injections, especially when periodically repeated, can be expected to resolve many such chronic tonsillar infections. Furthermore, the resolution of such advanced infection and abscesses can also be expected to return to a normal status much of the protective immune function provided by the tonsils.

These injection protocols can be utilized while infected teeth and gums are still present in the mouth, with the intention of some positive health benefit still accruing. However, an infection-free tonsil in the long-term should never be assumed until there has been administered a series of ozone injections **after** all oral infections have been removed or effectively treated

(as with the gums), and the CRP remains in the lower range of normal.

When CRP levels remain high or a clinical response is felt to be suboptimal after tonsillar ozone injections, consideration can be given to a similar series of injections to as much of the tonsillar tissue in Waldeyer's ring as can be reached, including the lingual tonsil, the palatine tonsils (or the healed tissue where they had been surgically removed), the tubal tonsils, and the adenoids. At the time of the writing of this book, there are only a few practitioners who have given this extensive an ozone treatment of all the tonsillar tissue, but that number is continuing to grow. Experienced ozone practitioners are not generally reluctant to inject new tissue areas as long as there is evidence or suspicion of chronic infection there. And other than the discomfort of the injection, ozone does no harm to normal tissue if no infection is actually present.

General Approach for Treating Chronic Pathogen Colonization

Elimination of the pathogens present in focal infections and aerodigestive sites of CPC is a giant step toward the resolution of CPC residing in other chronic disease-associated tissues throughout the body. As discussed previously, the majority of CPC in the body exists in the aerodigestive tract (sinuses, nose, mouth, throat, airways, and esophagus). It is these oral focal infections and areas of aerodigestive CPC that perpetually "seed" CPC in the other areas of the body, primarily through a leaky gut. Therefore, it is essential to address these infections and the sites of aerodigestive CPC in order to eliminate this continual flow of pathogens and

toxins to other tissues of the body as a primary step toward resolving the CPC in those tissues. As these secondary CPC sites start to resolve, those individuals with mild presentations of a chronic disease could see

As these secondary CPC sites start to resolve, those individuals with mild presentations of a chronic disease could see a complete resolution of their medical conditions.

a complete resolution of their medical conditions. Of course, the input of an integrative physician and/or a biological dentist is required to prescribe and execute an optimal treatment protocol and long-term health maintenance plan.

Probably the primary way that CPC is initiated in the aerodigestive tract is from residual pathogen colonization following the clinical resolution of an acute cold or bout of flu. Residual colonizations from colds and flus almost always occur as a rule. Areas of CPC do not resolve themselves so they should be assumed to be present in all or part of the aerodigestive tract after a cold or flu has clinically resolved, no matter how much time has elapsed since the initial infection. For that reason, pre-emptive steps for preventing a cold or flu from taking hold or for prompt treatment after any characteristic symptoms occur is ***always indicated*** to minimize the chances of sites of CPC getting started. Any of the focal infections discussed can also seed and start areas of CPC in the aerodigestive tract.

Anti-pathogen nebulization for pre-existing CPC is the most important part of any chronic disease resolution protocol, in addition to being essential for the prevention and/or resolution of any acute respiratory infection.

The single best monotherapy for a cold, flu, or COVID infection is HP nebulization. As an added benefit, individuals who use it are simultaneously breaking up pre-existing CPC in the aerodigestive tract. Those individuals treating acute respiratory infections with properly-administered HP nebulization often experience a sudden surge of well-being — and even a sense of what optimal health feels like — as the biofilm-protected pathogens that have been present for years are eradicated along with the new pathogens responsible for their acute respiratory infections.

Besides HP, several other non-toxic, anti-pathogenic agents can be nebulized. Some of these include:

- ✓ DMSO
- ✓ Vitamin C
- ✓ Magnesium chloride
- ✓ Zinc chloride
- ✓ Nascent iodine
- ✓ Sodium bicarbonate
- ✓ N-acetylcysteine
- ✓ Normal saline and hypertonic saline

If an increase in the intracellular uptake of vitamin C and/or magnesium is desired, insulin and/or hydrocortisone may be added to the nebulized solution as well. Modern medicine has long focused on the oral or parenteral delivery of drugs with the hope that

sufficient doses will eventually arrive at the target tissues. Nebulization provides the ability to deliver higher concentrations of therapeutic agents and nutrients directly to diseased or infected lung tissues than

Those individuals treating acute respiratory infections with properly-administered HP nebulization often experience a sudden surge of well-being...

can often be achieved with traditional administration. In some situations, nebulization also allows the therapeutic levels of inhaled agents to diffuse throughout the body while avoiding potentially toxic doses that might be required with other means of intake.

Although HP is a powerful monotherapy for the treatment/resolution of acute respiratory infections or CPC in the aerodigestive tract, the agents listed above can also be added to HP depending on the therapeutic needs and objectives. While exceptionally nontoxic, overexposure to HP can nevertheless manifest pro-oxidant effects such as nasal burning, irritation, runny nose, sneezing, a minimally sore throat, or a tendency for a non-productive cough. If an HP nebulization is overly aggressive and continues long enough beyond pathogen resolution, combinations of these other agents may also be nebulized separately as a means of helping to heal the tissue after pathogen elimination and/or to soothe any possible irritation. Lower concentrations of HP in nebulization solutions will almost completely avoid such side effects, but a 3% concentra-

tion will produce a much more rapid pathogen kill and lead to a quicker clinical recovery.

Although it is never recommended that one nebulize with a HP concentration greater than 3%, a

Often, many acutely ill and heavily-infected individuals experience no side effects when nebulizing with a 3% HP concentration and report that it is extremely easy to inhale and even very calming.

weaker concentration is fine and may be created by diluting with water or saline. Saline is preferable as a rule, since sodium chloride has its own anti-pathogen impact, and the nebulization of water tends to provoke coughing. The greater strength solutions will work more quickly, but weaker solutions may be used to avoid the potential effects noted above. Often, many acutely ill and heavily-infected individuals experience no side effects when nebulizing with a 3% HP concentration and report that it is extremely easy to inhale and even very calming. The higher concentration is also able to improve blood oxygenation more quickly than lower ones.

Although both the desktop jet nebulizer or a small handheld mesh nebulizer are effective, the handheld version is very quiet and delivers a finer, deeper-penetrating mist. The finer mist is especially important for dealing with infections which are already established

deep in the lungs. Chapter 11 has more information on the purchase and maintenance of a nebulizer.

The following HP nebulization protocol is a simple, inexpensive approach for the prevention and treatment of all respiratory tract infections. When used less frequently, the same protocol serves as a reliable and easy way to normalize and maintain the microbial flora in the aerodigestive tract.

Hydrogen Peroxide Nebulization Protocol

Regular OTC (over the counter) 3% HP can be utilized, especially for acute infections. If expense and availability present no difficulties, food-grade or other purer forms of HP can be used. However, there is no evidence of any harm in using the OTC HP for nebulization. Nor is there any scientific reason to believe that any potential harm will eventually be seen. Nevertheless, if someone plans on adopting intermittent HP nebulization as a procedure to be applied for life, HP of optimal purity is certainly a reasonable option. However, only having the availability of OTC HP should not deter anyone from its regular use. Arguably, normal respiration in many environments presents as much, or more, potential “toxicity” as nebulized OTC HP.

As noted above, extremely dilute solutions of nebulized HP have been found to be very effective in dealing with respiratory pathogens. However, to optimize the speed and degree of effectiveness, 3% is still the best concentration to use if well-tolerated. But there should be no hesitation to use a lower, even much lower, concentration to find a HP nebulization that is not

only tolerated, but pleasant and supportive of easy breathing.

When a runny nose or slightly sore throat is already present, a good approach would be to nebulize as many as four times during the day for 10 to 15 minutes at a time. There is nothing wrong with doing the nebulization more often than this if desired. Generally, significant symptomatic relief will be realized by the completion of the second nebulization, although many report near-immediate improvement in symptoms and an improved sense of well-being after the first nebulization.

As with all therapeutic anti-pathogen measures, it is always advisable to continue the nebulization to some degree for at least 24 to 48 hours beyond complete symptom resolution and a return to feeling normal in order to minimize the chances of clinical relapse and renewed infective symptoms.

For both the prevention of acute respiratory illness as well as for maintenance of a normal aerodigestive tract flora upon elimination of CPC, nebulization can be done as often as desired. For some professions that involve close contact with many people, such as the dentist who is only a few feet away from multiple open mouths for many hours, a brief nebulization at the end of the workday is an excellent idea. This can be done for 2 to 3 minutes at a time — a session of a 10- to 15-minute duration would not be needed.

For someone feeling well who experienced a positive impact on bowel and gut function after initial CPC eradication with HP nebulizations, a 2- to 5-minute HP nebulization once weekly would be a good maintenance approach. However, the normalcy

of the microbiome can deteriorate much more rapidly than is commonly appreciated, and any loosening of bowel function after an earlier return to more formed bowel movements should signal the need for imme-

When a runny nose or slightly sore throat is already present, a good approach would be to nebulize as many as four times during the day for 10 to 15 minutes at a time.

diate daily HP nebulization until a return to normal bowel function is seen.

Secondary measures and considerations that can be applied in the support of the recovering abnormal microbiome and associated leaky gut after the aerodigestive CPC has been largely eradicated include the following:

- ✓ All of the measures applying to focal infection discussed above can contribute to lessening the swallowing of pathogens and toxins, and to minimizing the recurrence of CPC in the aerodigestive tract, especially with good tongue and gum care. The maintenance of a healthy tongue and healthy gums is substantially more important to keeping a normal flora in the aerodigestive tract than by the elimination of infected teeth and tonsils. This is due to the route by which most of the involved pathogens and toxins are released into the

body. The infected teeth and tonsils have much a greater direct negative impact on body-wide increased oxidative stress, as they release most of their pathogens and toxins directly into the draining blood and lymphatic circulations, and not into the throat to be swallowed into the lower digestive tract.

- ✓ Probiotics can be used early on when a program of HP nebulization is being initiated. While not necessary for most individuals once the measures above have been implemented, they can certainly help some individuals “kickstart” the recolonization of their microbiome with good gut bugs. And this is not to say that anyone who feels that probiotics are helping their gut function and digestion significantly should feel compelled to stop taking them. However, most people who only had minimal digestive/bowel problems prior to HP nebulization simply do not need the “support” of probiotics indefinitely to restore and maintain a normal microbiome. Stopping the continual swallowing of pathogens from the CPC in the aerodigestive tract usually improves gut function very rapidly.
- ✓ Also discussed earlier, it is important to avoid dietary iron sources as much as possible, as is seen especially with foods that are labeled as being enriched or fortified. Extra ingested iron beyond what is needed to maintain normal hemoglobin

synthesis is never desirable. This includes supplemental or prescribed iron. This extra iron results in increased oxidative stress in the gut, directly promoting inflammation causing the barrier function of the intestinal cells to become degraded, resulting in a leaky gut. And even when the microbiome has not yet become overpopulated with pathogens, this leaky gut can permit the swallowed pathogens and toxins from unresolved CPC in the aerodigestive tract to be readily assimilated into the blood and lymphatic circulations and disseminate throughout the body.

- ✓ Regardless of any other factors that are involved in improving gut function, optimal function will never be realized if no attention is paid to the established principles of proper food combining. The wrong combinations **always** slow gut motility and bowel transit times, often profoundly. Food which should be retained in the stomach for 1 to 2 hours might stay there for 8 to 9 hours. This results in incomplete digestion and increased putrefaction of the food. The abnormal gut bugs, like many *Clostridia* species that are always present and waiting for a favorable sluggish bowel environment, quickly proliferate and produce potent pathogen-related toxins directly in the gut. Of course, this just further sustains any abnormal gut microbiome and leaky gut that the

CPC eradication protocol is attempting to correct.

CPC in Diseased Tissues:

The chronic colonization of pathogens, usually seeded from oral origins, has been documented to be present in the affected tissues of a number of different chronic diseases (see Chapter 4). It is likely that such CPC would also be found in the diseased tissues of nearly all chronic medical conditions, since it is only the presence of significant sources of new oxidative stress that prevent damaged (oxidized) tissue from getting repaired and showing clinical improvement. In other words, a redox (reduction/oxidation) state in the affected tissues must chronically favor reduction (repair) over oxidation (damage) in order for true healing to take place. And this almost never occurs when the CPC in a diseased tissue is permitted to continue to persist and chronically produce new pathogen-related toxins to keep the focal redox balance in that tissue skewed toward oxidation.

So far, however, only a relatively small number of diseases have been investigated specifically for the presence of such oral pathogens. However, the documentation that a chronic dental infection such as periodontitis has a probable cause-and-effect relationship to an enormous number of different diseases and conditions is a strong indicator that CPC will be found in a vast majority of the tissues affected by any chronic disease if sensitive enough testing is utilized. This is especially the case for any disease that shows little positive response to quality antioxidant therapy or any tendency toward actual disease reversal. However,

when these areas of CPC can be eliminated or substantially minimized in their presence in the tissues, a great deal of pathology reversal can be anticipated, and some early chronic diseases can be expected to resolve completely.

In order to resolve or minimize any CPC in diseased tissues throughout the body, several important interventions need to take place:

- ✓ Stop the seeding of new pathogens. This is achieved by the measures already discussed above, which include
 - eradicating oral cavity focal infections,
 - eradicating aerodigestive sites of CPC that permit pathogen access to the body through the leaky gut that the CPC helps to cause and sustain,
 - following the measures needed to heal the leaky gut and restore a normal gut microbiome, and
 - optimizing the levels of the most critical oxidative stress-modulating hormones in the body (estrogen, testosterone, thyroid).
- ✓ Eradicate the areas of CPC already in the tissues as directly as possible with the most potent anti-pathogen agents available (bio-oxidative therapies, usually in combination).
- ✓ Help infected and previously infected cells regain optimal health by the restoration of normal intracellular levels of magnesium, vitamin C, and glutathione.

As can be readily appreciated at this point in addressing chronic infections in the body, there are always complex interrelationships among all the factors involving pathogens and their related toxins, along with their chronic dissemination and seeding throughout the body.

The measures that will now be discussed have the potential to cause substantial healing and resolution of chronic disease by themselves. However, an optimal clinical response, and sometimes just any positive clinical response at all, ***must first involve the resolution of the factors continuing to freshly seed the CPC in the tissues of the body.***

In order to eradicate CPC in the tissues of the body outside of the aerodigestive tract, optimal therapeutic agents should have the collective ability to:

- ✓ Kill/inactivate and metabolically degrade any infectious agents, including bacteria, viruses, protozoa, and fungi [bio-oxidative therapies]
- ✓ Hydrate and oxygenate the previously infected tissues [bio-oxidative therapies].
- ✓ Restore normal levels of antioxidants, vitamins, minerals, and other nutrients to the tissues (especially vitamin C and magnesium) [proper supplementation]
- ✓ Restore normal levels of thyroid hormone and the appropriate sex hormone [management and prescription by a qualified integrative health care practitioner]

Not surprisingly, it is a combination of bio-oxidative therapies (HP, vitamin C, ozone, ultraviolet

blood irradiation, and hyperbaric oxygen therapy) that is needed to achieve the goal of CPC eradication in the tissues. That is not to say that all five of them would always be required. Clinical oversight

However, a multi-pronged bio-oxidative approach is the best way to significantly improve chronic diseases in the most patients.

from an experienced integrative physician or other healthcare provider can help optimize the eradication protocol. Clinical oversight will be needed for a program of optimal supplementation along with the treatment of hormone deficiencies and serial laboratory test monitoring.

Unlike the treatment and prevention of acute respiratory infections, the effective treatment of areas of CPC throughout the body must involve a licensed healthcare practitioner who understands the treatment protocol and wants to apply it as completely as possible. The number of such practitioners is small currently, but continuing to increase in number. Also, some such open-minded practitioners might only have the ability and resources to offer a part of the suggested protocol. For example, intravenous vitamin C might be available but not ozone blood treatment. For some individuals, just one arm of the protocol could suffice for CPC resolution.

However, a multi-pronged bio-oxidative approach is the best way to significantly improve chronic diseases

in the most patients. Furthermore, the patient should know that eradicating entrenched CPC throughout the body will not be achieved with the same level of success as the eradication of oral cavity focal infections and aerodigestive tract CPC. But that should not deter the clinician from giving each patient their best chance at their own optimal degree of health.

Also, while many patients will have already experienced significant improvement of their general health with focal infection/aerodigestive CPC eradication, there are still not many patients who have had the entirety of the protocol(s) that are being recommended applied. Nevertheless, there is every reason to believe that aggressive anti-pathogen measures applied *after* the eradication of aerodigestive pathogens and oral focal infections will produce excellent clinical results. However, the normalization of the gut microbiome and the healing of any associated leaky gut needs to occur first in order to bring the health of the patient to a level never achieved with the mainstream medical therapies that simply ignore the presence of chronic pathogen production and ingestion.

The primary bio-oxidative therapies for eliminating diseased tissue CPC are:

- ✓ Intravenous HP infusions.
- ✓ Intravenous vitamin C and magnesium chloride infusions (also oral liposome-encapsulated forms of vitamin C and magnesium). The magnesium administration is not primarily bio-oxidative by itself, but highly supportive of the clinical impact of the bio-oxidative agents.

- ✓ Ozone autohemotherapy treatments (extract blood, expose to ozone, re-infuse).
- ✓ Ultraviolet blood irradiation treatments (extract blood, expose to ultraviolet light, re-infuse).

Hyperbaric oxygen treatment (HBOT) would probably be an excellent adjunct based on its ability to resolve otherwise non-responding infections like large bedsores or chronic osteomyelitis, which are very analogous to “deep-seated” CPC in a diseased tissue. However, combining HBOT with these other therapies has not yet been done to a great enough degree to just be included whenever possible in an uncontrolled manner. Intravenous vitamin C has been successfully infused through special chamber adaptors during HBOT treatments, and this is a good simultaneous combination. HBOT should prove to be an excellent bio-oxidative therapy to add in any of a number of combinations with the other therapies, but at this point in time it is recommended that it should be done before or after the other therapies, not simultaneous with them.

The Riordan Clinic in Wichita, Kansas has developed an “Ascorbazone Protocol,” which integrates the administrations of intravenous vitamin C (ascorbate), ozone autohemotherapy, and ultraviolet blood irradiation, with good clinical results in a number of clinical conditions, including some cases of advanced Lyme disease. Combined with the intravenous administrations, an oral “Multi-C Protocol” seeks to achieve as complete as possible a vitamin C saturation in both the intracellular and extracellular spaces. Additional options at the discretion of the treating clinician

include small doses of insulin and/or hydrocortisone, which can substantially enhance the uptake of vitamin C into the cells.⁵⁻⁷ While not part of the Ascorbazone Protocol, the addition of periodic intravenous applications of HP would be expected to result in an even better clinical outcome.

A suggested oral dosing schedule in seeking to reach a state of vitamin C saturation includes the following:

- ✓ 2 packets of liposome-encapsulated vitamin C, taken 3 times daily (6 packets total)
- ✓ 1 packet of liposome-encapsulated glutathione, taken 3 times daily (3 packets total)
- ✓ 1 packet of liposome-encapsulated magnesium, taken 2 times daily (2 packets total)
- ✓ 1 level teaspoon of sodium ascorbate powder in water or juice, taken 3 times daily (roughly 10 grams total)
 1. If the patient is unacceptably bothered with an ascorbate-induced bowel-flushing effect, subsequent days of the protocol will be ½ level teaspoon of sodium ascorbate powder 3 times daily (about 5 grams total)
 2. If no bowel effect occurs, subsequent days of the protocol will be 1 and ½ level teaspoons of powder 3 times daily (about 15 grams total)
 3. If this increase produces no bowel effect, then the dose will be increased to 2 level teaspoons

3 times daily and maintained at that amount for the duration of the protocol (about 20 grams total)

- ✓ 1 gram of ascorbyl palmitate as 500 mg capsules, 2 taken 3 times daily (3 grams total)

An optimal regimen of intravenous administrations should include vitamin C, ozone- and/or ultraviolet light-treated blood, and HP (all given separately, NOT in the same IV administration). An intravenous infusion of vitamin C, eventually at a dose of 50 grams or more for the average-sized adult, can be given first and then followed by an appropriately administered infusion of appropriately dilute HP. Many thousands of IVC infusions have been given at the Riordan Clinic, and the basic protocol used there can readily be applied as part of the approach for eradicating CPC described here.⁸

Infusions of dilute HP are exceptionally effective anti-pathogen measures. It is well-documented that they can readily cure respiratory viral syndromes in an appropriate clinical protocol. However, there is no need to go straight to the intravenous administration of HP when nebulized HP, along with high doses of oral or intravenous vitamin C can be given and relied upon to do the job most of the time. However, for the purposes of deep tissue penetration throughout the body to eliminate as much CPC as possible throughout the body, the administration of intravenous HP is very important, and likely essential. As mentioned earlier, the HP and the vitamin C are very synergistic agents that are effective in the elimination of pathogens and their protective biofilms wherever they are encountered.

The administration of HP intravenously requires much more attention to detail than is needed for IVC and other infusions. Most infusions can have widely varying concentrations, infusion rates, potential combinations of different solutes (therapeutic agents), and solvents (water, saline, etc.). HP infusions must adhere to the established protocols. Given with proper attention to detail, it is highly effective clinically and completely safe. One concern is the uncommon occurring irritation or inflammation of the vein being accessed for infusion. If a large enough vein or HP concentration cannot be found for an infusion without pain or burning, a central line should be started or HP infusion efforts for that patient should be abandoned, as prolonged irritation/inflammation of a vein reliably results in its thrombosis and loss as an access point for any IV administration. Having a central vein access, however, is a very good idea if possible for any plan for an extended series of IV infusions, including HP. However, most experienced practitioners of intravenous HP therapies never experience this complication or the need for a central line.

One HP infusion protocol was given to a series of 56 patients with persistent joint pains after the resolution of acute Chikungunya viral infection syndromes. They were given 100 cc infusions of normal saline with 3 cc of a 3% solution of HP added. The infusions also had 500 mg of magnesium chloride and 1,000 micrograms of methylcobalamin added to them. These infusions were followed with a 500 cc infusion of sterile water or lactated Ringer's solution with 20 to 50 grams of ascorbic acid and a B-vitamin Complex. All of this was slowly infused over a 2- to 4-hour period. Note that HP

must be given with saline, lactated Ringer's solution, or a 5% dextrose solution. If just given in sterile water, the final solution has no significant osmolarity and can cause hemolysis, just as if straight water was given intravenously. This sequence of HP infusion followed by vitamin C infusion was very well-tolerated.⁹

Basically, then, one possible protocol of intravenous infusions to eliminate CPC in different diseased tissues is:

- ✓ Daily (or 5x per week) infusions of 50 to 75 grams of vitamin C with 500 mg of magnesium chloride added to each infusion
- ✓ Daily (or 5x per week) ozone autohemotherapy blood treatments and/or ultraviolet blood irradiation treatments
- ✓ Daily (or 5x per week) of HP infusions as described above

Individual patient responses will vary widely, and a complete CPC eradication cannot always be achieved, even if significant clinical improvement is realized as it is lessened in the degree of its presence in the tissues. However, depending on the patience and financial resources of the patient, the chances of an optimal clinical outcome can be steadily improved the longer such a regimen can be continued. Anecdotally, in a series of 12 patients with advanced Lyme disease, the infusion of 50 grams or more of vitamin C daily (5x to 6x per week), resulted in a complete clinical cure after the 20th infusion in each patient. However, little improvement was seen until the last few infusions.

While this highlights the therapeutic potential of extended and highly-dosed vitamin C infusion proto-

cols, many clinicians, and justifiably so, will not be keen on having any patient failing to show significant improvement after such an extensive and expensive (no insurance coverage) protocol of infusions. Nevertheless, it is anticipated that the inclusion of the ozone and/or ultraviolet treatments and the HP infusions will significantly increase the chances of a substantial and permanent positive clinical response, especially after focal infections and aerodigestive tract CPC sites have been eliminated, and the gut has been able to heal or substantially improve. Achieving such a positive clinical response further underscores the need for a maintenance program to prevent recurrent aerodigestive CPC and the eventual reseeding of pathogens again throughout the body.

For those seeking some guidance and structure in a program of regular long-term oral supplementation for the support of optimal health, this information can be found elsewhere.¹⁰ Periodic 3D X-ray of the oral cavity is advisable to detect new but pain-free tooth infections, especially when new disease symptoms occur, or old disease symptoms recur.¹¹ Once a chronic disease has shown a clearly positive response to the treatment approaches discussed in this chapter, there must be continued vigilance to prevent/detect any recurrence of focal infections, CPC, and abnormal gut function in order to preserve clear-cut gains in overall health.

Recap

The improvement and maintenance of good, or even optimal, health depends on keeping the redox balance throughout the body shifted toward the side of reduction. Optimal health depends on only the

presence of the minimal amounts of oxidative stress generated by normal metabolism that is needed to support normal signaling functions in the metabolic pathways in the cells.

Rapid Virus Recovery (RVR) leads directly to Rapid Gut Recovery (RGR). Even though this book aims to let as many people around the world know that they need never suffer again from ANY acute respiratory viral syndrome, it is just as important for the individual to realize how directly the RVR measures taken can result in RGR.

The measures taken to achieve RVR are the first and major steps to RGR. When an acute respiratory virus is obliterated, the microbe colonization of the aerodigestive tract is reset to normal, even when it was not normal before the respiratory virus exposure took place. In individuals with less gut problems, this intervention alone can result in a seemingly complete normalization of gut function as the swallowing of pathogens and their toxins largely stops. Other measures to more completely stop this pathogen/toxin swallowing involve addressing infections or pathogen colonizations of the tongue, the gums, the tonsils, and the teeth.

It needs to be re-emphasized that a comprehensive approach to the recovery and maintenance of good health has been offered in this chapter. For many reasons, everything that is recommended often cannot be accomplished by a given patient. Nevertheless, as many of the measures described as possible should be undertaken. Even the intermittent long-term nebulization of HP after RVR can improve general health to a level never previously attained by many patients.

In a nutshell, then:

- ✓ Resolve all pathogen colonizations and infections in the aerodigestive tract.
- ✓ Eat well and digest well.
- ✓ Balance the microbiome and heal the leaky gut.
- ✓ Normalize critical hormone levels.
- ✓ Supplement optimally.
- ✓ And then eradicate CPC throughout the body.

And finally, stop being afraid and start working on **improving** your health, not just avoiding the pitfalls that worsen your health.

Further Reading and Other Resources

For general inquiries (not including medical consultations or direct advice regarding the evaluation or treatment of a specific patient):

televymd@yahoo.com

Books by Dr. Levy:

Uninformed Consent: The Hidden Dangers in Dental Care (with

Hal A. Huggins, DDS, MS), Charlottesville, VA: Hampton Roads Publishing Company, Inc., 1999.

Optimal Nutrition for Optimal Health, New York, NY: McGraw-Hill (Keats Publishing), 2001.

The Roots of Disease: Connecting Dentistry and Medicine

(with Robert Kulacz, DDS), Philadelphia, PA: Xlibris Corporation, 2002.

Curing the Incurable: Vitamin C, Infectious Diseases, and

Toxins, Henderson, NV: MedFox Publishing, 2002.

Stop America's #1 Killer, Henderson, NV: MedFox Publishing,

2006.

GSH: Master Defender Against Disease, Toxins, and Aging,

Henderson, NV: MedFox Publishing, 2008.

Living in Your Right Mind, Henderson, NV: MedFox Publishing,

2010

Primal Panacea, Henderson, NV: MedFox Publishing, 2011.

Death by Calcium: Proof of the Toxic Effects of Dairy and Calcium Supplements, Henderson, NV: MedFox Publishing, 2013.

The Toxic Tooth: How a Root Canal Could Be Making You Sick

(with Robert Kulacz, DDS), Henderson, NV: MedFox Publishing, 2014.

Hidden Epidemic: Silent Oral Infections Cause Most Heart

Attacks and Breast Cancers, Henderson, NV: MedFox Publishing, 2017.

Magnesium: Reversing Disease, Henderson, NV: MedFox

Publishing, 2019.

Books available at these websites and elsewhere:

<https://www.medfoxpath.com/>

<https://www.amazon.com/>

Articles by Dr. Levy:

<https://www.peakenergy.com/>

To help find a healthcare practitioner who might be open to some or all of the treatment options discussed in this book:

<https://www.acam.org/page/Searches>

<http://orthomolecular.org/resources/pract.shtml>

<https://www.a4m.com/find-a-doctor.html>

<https://icimed.com/icim-member-search/>

<https://www.acimconnect.com/Resources/>

Find-a-Health-Professional

<https://riordanclinic.org/>

To help find a biological dentist who might be open to some or all of the treatment options discussed in this book:

<https://iaomt.org/for-patients/search/>

<https://hugginsappliedhealing.com/find-a-dentist/>

<https://iabdm.org/location/>

To help find quality supplements and other products as discussed in this book:

Liposome-encapsulated vitamins, minerals, and nutrients:

<https://www.livonlabs.com/>

<https://www.altrient.com/>

Supplements in general:

<https://www.lifeextension.com/>

<https://www.swansonvitamins.com/>

Injectables:

<https://www.meritpharm.com/>

<https://www.mcguffpharmaceuticals.com/>

<https://torrancecompany.com/>

Additional Information Resources:

<http://orthomolecular.org/resources/omns/index.shtml>

<https://www.mercola.com/>

<http://www.doctoryourself.com/>

<https://riordanclinic.org/>

<https://www.naturalhealth365.com/>

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References

Chapter One

1. Dockrell H, Playfair J (1983) Killing of blood-stage murine malaria parasites by hydrogen peroxide. *Infection and Immunity* 39:456-459. PMID: 6822428
2. Heckert R, Best M, Jordan L et al., (1997) Efficacy of vaporized hydrogen peroxide against exotic animal viruses. *Applied and Environmental Microbiology* 63:3916-3918. PMID: 9327555
3. Berrie E, Andrews L, Yezli S, Otter J (2011) Hydrogen peroxide vapour (HPV) inactivation of adenovirus. *Letters in Applied Microbiology* 52:555-558. PMID: 21418259
4. Goyal S, Chander Y, Yezli S, Otter J (2014) Evaluating the virucidal efficacy of hydrogen peroxide vapour. *The Journal of Hospital Infection* 86:255-259. PMID: 24656442
5. Halliwell B, Clement M, Long L (2000) Hydrogen peroxide in the human body. *FEBS Letters* 486:10-13. PMID: 11108833
6. Caffarelli C, Calcinai E, Rinaldi L et al. (2012) Hydrogen peroxide in exhaled breath condensate in asthmatic children curing acute exacerbation and after treatment. *Respiration* 84:291-298. PMID: 23018317
7. Klebanoff S, Locksley R, Jong E, Rosen H (1983) Oxidative response of phagocytes to parasite invasion. *Ciba Foundation Symposium* 99:92-112. PMID: 6315321
8. Thomas D (2017) The phagocyte respiratory burst: historical perspectives and recent advances. *Immunology Letters* 192:88-96. PMID: 28864335
9. Nagaraja C, Shashibhushan B, Sagar et al. (2012) Hydrogen peroxide in exhaled breath condensate: a clinical study. *Lung India* 29:123-127. PMID: 22628925
10. Rysz J, Stolarek R, Luczynski R et al. (2007) Increased hydrogen peroxide concentration in the exhaled breath condensate of stable COPD patients after nebulized N-acetylcysteine. *Pulmonary Pharmacology & Therapeutics* 20:281-289. PMID: 16753318
11. Reth M (2002) Hydrogen peroxide as second messenger in lymphocyte activation. *Nature Immunology* 3:1129-1134. PMID: 12447370
12. Nelson D, Murray D (1987) Dexamethasone inhibition of hydrogen peroxide-stimulated glucose transport. *Endocrinology* 120:156-159. PMID: 3780558
13. Pillai K, Akhter J, Chua T, Morris D (2012) Mucolysis by ascorbic acid and hydrogen peroxide on compact mucin secreted in pseudomyxoma peritonei. *The Journal of Surgical Research* 174:e69-e73. PMID: 22261589
14. Boveris A, Chance B (1973) The mitochondrial generation of hydrogen peroxide. General properties and effect of hyperbaric oxygen. *The Biochemical Journal* 134:707-716. PMID: 4749271

15. Bao L, Avshalumov M, Patel J et al. (2009) Mitochondria are the source of hydrogen peroxide for dynamic brain-cell signaling. *The Journal of Neuroscience* 29:9002-9010. PMID: 19605638
16. Rice M (2011) H₂O₂: a dynamic modulator. *Neuroscientist* 17:389-406. PMID: 21666063
17. Arnaiz S, Coronel M, Boveris A (1999) Nitric oxide, superoxide, and hydrogen peroxide production in brain mitochondria after haloperidol treatment. *Nitric Oxide: Biology and Chemistry* 3:235-243. PMID: 10442855
18. Rowen R (2019) Ozone and oxidation therapies as a solution to the emerging crisis in infectious disease management: a review of current knowledge and experience. *Medical Gas Research* 9:232-237. PMID: 31898609
19. Maddalena L, Selim S, Fonseca J et al. (2017) Hydrogen peroxide production is affected by oxygen levels in mammalian cell culture. *Biochemical and Biophysical Research Communications* 493:246-251. PMID: 28899780
20. Munns S, Lui J, Arthur P (2005) Mitochondrial hydrogen peroxide production alters oxygen consumption in an oxygen-concentration-dependent manner. *Free Radical Biology & Medicine* 38:1594-1603. PMID: 15917188
21. Hidvegi M (2020) Inhaled nebulized sodium pyruvate use in COVID-19 patients. *The Israel Medical Association Journal* 22:278. PMID: 32378817
22. Jobsis Q, Raatgeep H, Schellekens S et al. (1998) Hydrogen peroxide in exhaled air of healthy children: reference values. *The European Respiratory Journal* 12:483-485. PMID: 9727806
23. Varma S, Devamanoharan P (1990) Excretion of hydrogen peroxide in human urine. *Free Radical Research Communications* 8:73-78. PMID: 2318421
24. Gough D, Cotter T (2011) Hydrogen peroxide: a Jekyll and Hyde signalling molecule. *Cell Death & Disease* 2:e213. PMID: 21975295
25. Lee J, Walker K, Han H (2019) Spontaneous generation of hydrogen peroxide from aqueous microdroplets. *Proceedings of the National Academy of Sciences of the United States of America* 116:19294-19298. PMID: 31451646
26. Zhu C, Francisco J (2020) Production of hydrogen peroxide enabled by microdroplets. *Proceedings of the National Academy of Sciences of the United States of America* 116:19222-19224. PMID: 31484759
27. Lazarou J, Pomeranz B, Corey P (1998) Incidence of adverse drug reactions in hospitalized patients: a meta-analysis of prospective studies. *JAMA* 279:1200-1205. PMID: 9555760
28. Watt B, Proudfoot A, Vale J (2004) Hydrogen peroxide poisoning. *Toxicological Reviews* 23:51-57. PMID: 15298493

29. Akuji M, Chambers D (2017) Hydrogen peroxide: more harm than good? *British Journal of Anaesthesia* 118:958-959. PMID: 28575345

Chapter Two

1. Winterbourn C (1995) Toxicity of iron and hydrogen peroxide: the Fenton reaction. *Toxicology Letters* 82-83:969-974. PMID: 8597169
2. Aruoma O, Halliwell B (1988) The iron-binding and hydroxyl radical scavenging action of anti-inflammatory drugs. *Xenobiotica* 18:459-470. PMID: 3135672
3. Halliwell B, Clement M, Ramalingham J, Long L (2000) Hydrogen peroxide. Ubiquitous in cell culture and *in vivo*? *IUBMB Life* 50: 251-257. PMID: 11327318
4. Root R, Metcalf J, Oshino N, Chance B (1975) H₂O₂ release from human granulocytes during phagocytosis. I. Documentation, quantitation, and some regulating factors. *The Journal of Clinical Investigation* 55:945-955. PMID: 1123431
5. Root R, Metcalf J (1977) H₂O₂ release from human granulocytes during phagocytosis. Relationship to superoxide anion formation and cellular catabolism of H₂O₂: studies with normal and cytochalasin B-treated cells. *The Journal of Clinical Investigation* 60:1266-1279. PMID: 199619
6. Levine M, Padayatty S, Espey M (2011) Vitamin C: a concentration-function approach yields pharmacology and therapeutic discoveries. *Advances in Nutrition* 2:78-88. PMID: 22332036
7. Pei Z, Wu K, Li Z et al. (2019) Pharmacologic ascorbate as a pro-drug for hydrogen peroxide release to kill mycobacteria. *Biomedicine & Pharmacotherapy* 109:2119-2127. PMID: 30551469
8. Fenton H (1894) Oxidation of tartaric acid in the presence of iron. *J Chem Soc Trans* 65:899-910.
9. Babuponnusami A, Muthukumar K (2014) A review on Fenton and improvements to the Fenton process for wastewater treatment. *Journal of Environmental Chemical Engineering* 2:557-572.
10. Valko M, Jomova K, Rhodes C et al. (2016) Redox- and non-redox-metal-induced formation of free radicals and their role in human disease. *Archives of Toxicology* 90:1-37. PMID: 26343967

Chapter Three

1. Martin A, Finlay W (2015) Nebulizers for drug delivery to the lungs. *Expert Opinion on Drug Delivery* 12:889-900. PMID: 25534396
2. Stein S, Thiel C (2017) The history of therapeutic aerosols: a chronological review. *Journal of Aerosol Medicine and Pulmonary Drug Delivery* 30:20-41. PMID: 27748638

3. Muers M (1997) Overview of nebulizer treatment. *Thorax* 52 Suppl 2:S25-S30. PMID: 9155848
4. Aziz S, Scherliess, Steckel H (2020) Development of high dose oseltamivir phosphate dry powder for inhalation therapy in viral pneumonia. *Pharmaceutics* 12:1154. PMID: 33261071
5. Shirk M, Donahue K, Shirvani J (2006) Unlabeled uses of nebulized medications. *American Journal of Health-System Pharmacy* 63:1704-1716. PMID: 16960254
6. Martin A, Finlay W (2015) Nebulizers for drug delivery to the lungs. *Expert Opinion on Drug Delivery* 12:889-900. PMID: 25534396
7. Lavorini F, Buttini F, Usmani O (2019) 100 years of drug delivery to the lungs. *Handbook of Experimental Pharmacology* 260:143-159. PMID: 31792683
8. Fontana G, Lavorini F, Pistolesi M (2002) Water aerosols and cough. *Pulmonary Pharmacology & Therapeutics* 15:205-211. PMID: 12099765
9. Karimpour H, Hematpour B, Mohammadi S et al. (2020) Effect of nebulized eucalyptus for preventing ventilator-associated pneumonia in patients under mechanical ventilation: a randomized double blind clinical trial. *Alternative Therapies in Health and Medicine* Feb 21. Online ahead of print. PMID: 32088670
10. McCarthy S, Gonzalez H, Higgins B (2020) Future trends in nebulized therapies for pulmonary disease. *Journal of Personalized Medicine* 10:E37. PMID: 32397615
11. Morais C, Nascimento J, Ribeiro A et al. (2020) Nebulization of vancomycin provides higher lung tissue concentrations than intravenous administration in ventilated female piglets with healthy lungs. *Anesthesiology* 132:1516-1527. PMID: 32053565
12. Reifen R, Berkovich Z, Mandelberg A (2015) Vitamin A supplementation via aerosol spray in asthmatic children. *Pediatric Allergy and Immunology* 26:578-579. PMID: 26173085
13. Gelfand C, Sakurai R, Wang Y et al. (2020) Inhaled vitamin A is more effective than intramuscular dosing in mitigating hyperoxia-induced lung injury in a neonatal rat model of bronchopulmonary dysplasia. *American Journal of Physiology. Lung Cellular and Molecular Physiology* 319:L576-L584. PMID: 32755324
14. Taylor S, Sakurai R, Sakurai T, Rehan V (2016) Inhaled vitamin D: a novel strategy to enhance neonatal lung maturation. *Lung* 194:931-943. PMID: 27614961
15. Kalmarzi R, Ahmadi S, Rahehagh R et al. (2020) The effect of vitamin D supplementation on clinical outcomes of asthmatic children with vitamin D insufficiency. *Endocrine, Metabolic & Immune Disorders Drug Targets* 20:149-155. PMID: 31942850

16. Yamamoto Y, Enkhbaatar P, Sousse L et al. (2012) Nebulization with α -tocopherol ameliorates acute lung injury after burn and smoke inhalation in the ovine model. *Shock* 37:408-414. PMID: 22266978
17. Laouini A, Andrieu V, Vecellio L et al. (2014) Characterization of different vitamin E carriers intended for pulmonary drug delivery. *International Journal of Pharmaceutics* 471:385-390. PMID: 24939617
18. Levin E, Behm F, Carnahan E et al. (1993) Clinical trials using ascorbic acid aerosol to aid smoking cessation. *Drug and Alcohol Dependence* 33:211-223. PMID: 8261886
19. Antolick A, Ouellette L, Judge B et al. (2020) Accidental chlorine gas exposure in a pediatric patient: a case report. *Clinical Practice and Cases in Emergency Medicine* 4:205-207. PMID: 32426673
20. Bosse G (1994) Nebulized sodium bicarbonate in the treatment of chlorine gas inhalation. *Journal of Toxicology. Clinical Toxicology* 32:233-241. PMID: 8007031
21. Vinsel P (1990) Treatment of acute chlorine gas inhalation with nebulized sodium bicarbonate. *The Journal of Emergency Medicine* 8:327-329. PMID: 2165079
22. Ahmed T, Iskandrani A, Uddin M (2000) Sodium bicarbonate solution nebulization in the treatment of acute severe asthma. *American Journal of Therapeutics* 7:325-327. PMID: 11317181
23. Devadason S (2006) Recent advances in aerosol therapy for children with asthma. *Journal of Aerosol Medicine* 19:61-66. PMID: 16551216
24. Murphy K, Hong J, Wandalsen G et al. (2020) Nebulized inhaled corticosteroids in asthma treatment in children 5 years or younger: a systematic review and global expert analysis. *The Journal of Allergy and Clinical Immunology. In Practice* Jan 30. Online ahead of print. PMID: 32006721
25. Rossi I, Sonvico F, McConville J et al. (2018) Nebulized coenzyme Q10 nanosuspensions: a versatile approach for pulmonary anti-oxidant therapy. *European Journal of Pharmaceutical Sciences* 113:159-170. PMID: 29066385
26. Adewale A, Libby E, Fu L et al. (2020) Novel therapy of bicarbonate, glutathione, and ascorbic acid improves cystic fibrosis mucus transport. *American Journal of Respiratory Cell and Molecular Biology* 63:362-373. PMID: 32374624
27. Wu L, Yu Y, Li L et al. (2020) [Clinical effect of nebulized acetyl-cysteine inhalation combined with bronchoscopy in the treatment of elderly patients with severe ventilator-associated pneumonia]. Article in Chinese. 36:267-272. PMID: 32340416
28. Fox L, Foushee J, Jackson D, Watson H (2011) Visual compatibility of common nebulizer medications with 7% sodium chloride solution. *American Journal of Health-System Pharmacy* 68:1032-1035. PMID: 21593232

29. Zhong L, Xiong Y, Zheng Z et al. (2020) Effect of short-term inhalation of warm saline atomised gas on patients with non-cystic fibrosis bronchiectasis. *ERJ Open Research* 6:00130-2019. PMID: 32055629
30. Cho J, Kim H, Yang H et al. (2020) Pilot study of aerosolized plus intravenous vancomycin in mechanically ventilated patients with methicillin-resistant *Staphylococcus aureus* pneumonia. *Journal of Clinical Medicine* 9:476. PMID: 32050447
31. Akkerman-Nijland A, Yousofi M, Rottier B et al. (2020) Eradication of *Pseudomonas aeruginosa* in cystic fibrosis patients with inhalation of dry powder tobramycin. *Therapeutic Advances in Respiratory Disease* 14:1753466620905279. PMID: 32046620
32. Prazak J, Valente L, Iten M et al. (2020) Nebulized bacteriophages for prophylaxis of experimental ventilator-associated pneumonia due to methicillin-resistant *Staphylococcus aureus*. *Critical Care Medicine* Apr 16. Online ahead of print. PMID: 32304419
33. Chen L, Shi M, Deng Q et al. (2020) A multi-center randomized prospective study on the treatment of infant bronchiolitis with interferon α1b nebulization. *PLoS One* 15:e0228391. PMID: 32084142
34. Brodski N (2020) Add-on or alone? Inhaled nebulized immunoglobulin reduces upper airway infections: 24 months of real-life experience. *Immunotherapy* 12:389-394. PMID: 32308072
35. Kamel A, Amin O (2020) Analgo-sedative effects of oral or nebulized ketamine in preschoolers undergoing elective surgery: a comparative, randomized, double-blind study. *Pain Physician* 23:E195-E202. PMID: 32214298
36. Nguyen A, Denault A, Theoret Y et al. (2020) Inhaled milrinone in cardiac surgical patients: a pilot randomized controlled trial of jet vs. mesh nebulization. *Scientific Reports* 10:2069. PMID: 32034202
37. Klimke A, Hefner G, Will B, Voss U (2020) Hydroxychloroquine as an aerosol might markedly reduce and even prevent severe clinical symptoms after SARS-CoV-2 infection. *Medical Hypotheses* 142:109783. PMID: 32402766
38. Wan G, Tsai Y, Wu Y, Tsao K (2004) A large-volume nebulizer would not be an infectious source for severe acute respiratory syndrome. *Infection Control and Hospital Epidemiology* 25:1113-1115. PMID: 15636302
39. Amirav I, Newhouse M (2020) Transmission of coronavirus by nebulizer: a serious, underappreciated risk. *CMAJ* 192:E346. PMID: 32392488
40. Benge C, Barwise J (2020) Aerosolization of COVID-19 and contamination risks during respiratory treatments. *Federal Practitioner* 37:160-163. PMID: 32322146
41. Choi K (2012) Viral polymerases. *Advances in Experimental Medicine and Biology* 726:267-304. PMID: 22297518

42. Yin W, Mao C, Luan X et al. (2020) Structural basis for inhibition of the RNA-dependent RNA polymerase from SARS-CoV-2 by remdesivir. *Science* 368:1499-1504. PMID: 32358203
43. Kaushik N, Subramani C, Anang S et al. (2017) Zinc salts block hepatitis E virus replication by inhibiting the activity of viral RNA-dependent RNA polymerase. *Journal of Virology* 91:e00754-17. PMID: 28814517
44. Bohlmann L, De Oliveira D, El-Deeb I et al. (2018) Chemical synergy between ionophore PBT2 and zinc reverses antibiotic resistance. *mBio* 9:e02391-18. PMID: 30538186
45. Justiniano R, Perer J, Hua A et al. (2017) A topical zinc ionophore blocks tumorigenic progression in UV-exposed SKH-1 high-risk mouse skin. *Photochemistry and Photobiology* 93:1472-1482. PMID: 28503778
46. Vaden R, Guillen K, Salvant J et al. (2019) A cancer-selective zinc ionophore inspired by the natural product naamidine A. *ACS Chemical Biology* 14:106-117. PMID: 30571086
47. Xue J, Moyer A, Peng B et al. (2014) Chloroquine is a zinc ionophore. *PLoS One* 9:e109180. PMID: 25271834
48. Carlucci P, Ahuja T, Petrilli C et al. (2020) Zinc sulfate in combination with a zinc ionophore may improve outcomes in hospitalized COVID-19 patients. *Journal of Medical Microbiology* 69:1228-1234. PMID: 32930657
49. Dabbagh-Bazarbachi H, Clergeaud G, Quesada I et al. (2014) Zinc ionophore activity of quercetin and epigallocatechin-gallate: from Hepa 1-6 cells to a liposome model. *Journal of Agricultural and Food Chemistry* 62:8085-8093. PMID: 25050823
50. Levy T (2002) *Curing the Incurable: Vitamin C, Infectious Diseases, and Toxins*. Henderson, NV: MedFox Publishing
51. Levy T (2011) *Primal Panacea*. Henderson, NV: MedFox Publishing
52. Gonzales M, Berdiel M, Duconge J et al. (2018) High dose intravenous vitamin C and influenza: a case report. *Journal of Orthomolecular Medicine* Volume 33 pp. 1-3.
53. Gonzales M, Berdiel M, Miranda-Massari J et al. (2016) High dose intravenous vitamin C treatment for Zika fever. *Journal of Orthomolecular Medicine* Volume 31, pp. 19-23.
54. Gonzales M, Miranda-Massari J, Berdiel M et al. (2014) High dose intravenous vitamin C and Chikungunya fever: a case report. *Journal of Orthomolecular Medicine* 29:154-156. PMID: 25705076
55. Rowen R, Robins H, Carew K et al. (2016) Rapid resolution of hemorrhagic fever (Ebola) in Sierra Leone with ozone therapy. *African Journal of Infectious Diseases* 10:49-54.
56. Al-Ani F, Chehade S, Lazo-Langner A (2020) Thrombosis risk associated with COVID-19 infection. A scoping review. *Thrombosis Research* 192:152-160. PMID: 32485418

57. Al-Samkari H, Leaf R, Dzik W et al. (2020) COVID-19 and coagulation: bleeding and thrombotic manifestations of SARS-CoV-2 infection. *Blood* 136:489-500. PMID: 32492712
58. Colmenero I, Santonja C, Alonso-Riano M et al. (2020) SARS-CoV-2 endothelial infection causes COVID-19 chilblains: histopathological, immunohistochemical and ultrastructural study of seven paediatric cases. *The British Journal of Dermatology* 183:729-737. PMID: 32562567
59. Connors J, Levy J (2020) COVID-19 and its implications for thrombosis and anticoagulation. *Blood* 135:2033-2040. PMID: 32339221
60. Levy T (2019) *Magnesium: Reversing Disease* Henderson, NV: MedFox Publishing

Chapter 4

1. Del Pozo J (2018) Biofilm-related disease. *Expert Review of Anti-Infective Therapy* 16:51-65. PMID: 29235402
2. Kuang X, Chen V, Xu X (2018) Novel approaches to the control of oral microbial biofilms. *BioMed Research International* 2018:6498932. PMID: 30687755
3. Roy R, Tiwari M, Donelli G, Tiwari V (2018) Strategies for combating bacterial biofilms: a focus on anti-biofilm agents and their mechanisms of action. *Virulence* 9:522-554. PMID: 28362216
4. Mirzaei R, Mohammadzadeh R, Alikhani M et al. (2020) The biofilm-associated bacterial infections unrelated to indwelling devices. *IUBMB Life* 72:1271-1285. PMID: 32150327
5. Sato Y, Motoyama S, Takano H et al. (2016) Esophageal cancer patients have a high incidence of severe periodontitis and preoperative dental care reduces the likelihood of severe pneumonia after esophagectomy. *Digestive Surgery* 33:495-502. PMID: 27287475
6. Surlin P, Mirela-Nicolae F, Marin-Surlin V et al. (2020) Could periodontal disease through periopathogen *Fusobacterium nucleatum* be an aggravating factor for gastric cancer? *Journal of Clinical Medicine* 9:E3885. PMID: 33260439
7. Castellarin M, Warren R, Freeman J et al. (2012) *Fusobacterium nucleatum* infection is prevalent in human colorectal carcinoma. *Genome Research* 22:299-306. PMID: 22009989
8. Kim M, Lee S, Choi S et al. (2020) *Fusobacterium nucleatum* in biopsied tissues from colorectal cancer patients and alcohol consumption in Korea. *Scientific Reports* 10:19915. PMID: 33199749
9. Vacante M, Ciuni R, Basile F, Biondi A (2020) Gut microbiota and colorectal cancer development: a closer look to the adenoma-carcinoma sequence. *Biomedicines* 8:489. PMID: 33182693

10. Karpinski T (2019) The microbiota and pancreatic cancer. *Gastroenterology Clinics of North America* 48:447-464. PMID: 31383281
11. Zeng X, Xia L, Zhang Y et al. (2016) Periodontal disease and incident lung cancer risk: a meta-analysis of cohort studies. *Journal of Periodontology* 87:1158-1164. PMID: 27294431
12. Liu Y, Yuan X, Chen K et al. (2020) Clinical significance and prognostic value of *Porphyromonas gingivalis* infection in lung cancer. *Translational Oncology* 14:100972
13. Tezal M, Sullivan M, Reid M et al. (2007) Chronic periodontitis and the risk of tongue cancer. *Archives of Otolaryngology—Head & Neck Surgery* 133:450-454. PMID: 17515503
14. Mattila K, Puusinen P, Paju S (2005) Dental infections and cardiovascular diseases: a review. *Journal of Periodontology* 76:2085-2088. PMID: 16277580
15. Ott S, El Mokhtari N, Musfeldt M et al. (2006) Detection of diverse bacterial signatures in atherosclerotic lesions of patients with coronary heart disease. *Circulation* 113:929-937. PMID: 16490835
16. Zaremba M, Gorska R, Suwalski P, Kowalski J (2007) Evaluation of the incidence of periodontitis-associated bacteria in the atherosclerotic plaque of coronary blood vessels. *Journal of Periodontology* 78:322-327. PMID: 17274722
17. Mahendra J, Mahendra L, Kurian V et al. (2010) 16S rRNA-based detection of oral pathogens in coronary atherosclerotic plaque. *Indian Journal of Dental Research* 21:248-252. PMID: 20657096
18. Haraszthy V, Zambon J, Trevisan M et al. (2000) Identification of periodontal pathogens in atheromatous plaques. *Journal of Periodontology* 71:1554-1560. PMID: 11063387
19. Pessi T, Karhunen V, Karjalainen P et al. (2013) Bacterial signatures in thrombus aspirates of patients with myocardial infarction. *Circulation* 127:1219-1228. PMID: 23418311
20. Pinon-Estabán P, Nunez L, Moure R et al. (2020) Presence of bacterial DNA in thrombotic material of patients with myocardial infarction. *Scientific Reports* 10:16299. PMID: 33004892
21. Vakhitov D, Tuomisto S, Martiskainen M et al. (2018) Bacterial signatures in thrombus aspirates of patients with lower limb arterial and venous thrombosis. *Journal of Vascular Surgery* 67:1902-1907. PMID: 28847664
22. Louhelainen A, Aho J, Tuomisto S et al. (2014) Oral bacterial DNA findings in pericardial fluid. *Journal of Oral Microbiology* 6:25835. PMID: 25412607
23. Pyysalo M, Pyysalo L, Pessi T et al. (2016) Bacterial DNA findings in ruptured and unruptured intracranial aneurysms. *Acta Odontologica Scandinavica* 74:315-320. PMID: 26777430

24. Haheim L, Schwarze P, Thelle D et al. (2020) Low levels of antibodies for the oral bacterium *Tannerella forsythia* predict cardiovascular disease mortality in men with myocardial infarction: a prospective cohort study. *Medical Hypotheses* 138:109575. PMID: 32088522
25. Dominy S, Lynch C, Ermini F et al. (2019) *Porphyromonas gingivalis* in Alzheimer's disease brains: evidence for disease causation and treatment with small-molecule inhibitors. *Science Advances* 5:easu3333. PMID: 30746447
26. Diaz-Zuniga J, More J, Melgar-Rodriguez S et al. (2020) Alzheimer's disease-like pathology triggered by *Porphyromonas gingivalis* in wild type rats is serotype dependent. *Frontiers in Immunology* 11:588036. PMID: 33240277
27. Dioguardi M, Crincoli V, Laino L et al. (2020) The role of periodontitis and periodontal bacteria in the onset and progression of Alzheimer's disease: a systematic review. *Journal of Clinical Medicine* 9:495. PMID: 32054121
28. Ogendrik M (2013) Rheumatoid arthritis is an autoimmune disease caused by periodontal pathogens. *International Journal of General Medicine* 6:383-386. PMID: 23737674
29. Reichert S, Haffner M, Keysser G et al. (2013) Detection of oral bacterial DNA in synovial fluid. *Journal of Clinical Periodontology* 40:591-598. PMID: 23534379
30. Totaro M, Cattani P, Ria F et al. (2013) *Porphyromonas gingivalis* and the pathogenesis of rheumatoid arthritis: analysis of various compartments including the synovial tissue. *Arthritis Research & Therapy* 15:R66. PMID: 23777892
31. Flak M, Colas R, Munoz-Atienza E et al. (2019) Inflammatory arthritis disrupts gut resolution mechanisms, promoting barrier breakdown by *Porphyromonas gingivalis*. *JCI Insight* 4:e125191. PMID: 31292292
32. Du Q, Ma X (2020) [Research progress of correlation between periodontal pathogens and systemic diseases]. Article in Chinese. *Journal of Southern Medical University* 40:759-764. PMID: 32897213
33. Ye C, Katagiri S, Miyasaka N et al. (2020) The periodontopathic bacteria in placenta, saliva and subgingival plaque of threatened preterm labor and preterm low birth weight cases: a longitudinal study in Japanese pregnant women. *Clinical Oral Investigations* 24:4261-4270. PMID: 32333174
34. Fiorillo L, Cervino G, Laino L et al. (2019) *Porphyromonas gingivalis*, periodontal and systemic implications: a systematic review. *Dentistry Journal* 7:114. PMID: 31835888
35. Zhou Y, Luo G (2019) *Porphyromonas gingivalis* and digestive system cancers. *World Journal of Clinical Cases* 7:819-829. PMID: 31024953

36. Mei F, Xie M, Huang X et al. (2020) *Porphyromonas gingivalis* and its systemic impact: current status. *Pathogens* 9:944. PMID: 33202751
37. Laugisch O, Johnen A, Maldonado A et al. (2018) Periodontal pathogens and associated intrathecal antibodies in early stages of Alzheimer's disease. *Journal of Alzheimer's Disease* 66:105-114. PMID: 3022339
38. Adams B, Nunes J, Page M et al. (2019) Parkinson's disease: a systemic inflammatory disease accompanied by bacterial inflam-magens. *Frontiers in Aging Neuroscience* 11:210. PMID: 31507404
39. Figueiredo C, Sete M, Carlos J et al. (2018) Presence of anti-*Porphyromonas gingivalis*-peptidylarginine deiminase antibodies in serum from juvenile systemic lupus erythematosus patients. *Acta Reumatologica Portuguesa* 43:239-240. PMID: 30414375
40. Bagavant H, Dunkleberger M, Wolska N et al. (2019) Antibodies to periodontogenic bacteria are associated with higher disease activity in lupus patients. *Clinical and Experimental Rheumatology* 37:106-111. PMID: 29998833
41. Hieken T, Chen J, Hoskin T et al. (2016) The microbiome of asep-tically collected human breast tissue in benign and malignant disease. *Scientific Reports* 6:30751. PMID: 27485780
42. Park D, Woo B, Lee B et al. (2019) Serum levels of interleukin-6 and titers of antibodies against *Porphyromonas gingivalis* could be potential biomarkers for the diagnosis of oral squamous cell carci-noma. *International Journal of Molecular Sciences* 20:2749. PMID: 31167516
43. Fischer L, Demerath E, Bittner-Eddy P, Costalonga M (2019) Placental colonization with periodontal pathogens: the poten-tial missing link. *American Journal of Obstetrics and Gynecology* 221:383-392. PMID: 31051120
44. Pillai R, Iyer K, Spin-Neto R et al. (2018) Oral health and brain injury: causal or casual relation? *Cerebrovascular Diseases Extra* 8:1-15. PMID: 29402871
45. Aoki S, Hosomi N, Nishi H et al. (2020) Serum IgG titers to peri-odontal pathogens predict 3-month outcome in ischemic stroke patients. *PLoS One* 15:e0237185. PMID: 32760103
46. Kudo C, Shin W, Sasaki N et al. (2018) Effects of periodontal treatment on carotid intima-media thickness in patients with life-style-related diseases: Japanese prospective multicentre obser-vational study. *Odontology* 106:316-327. PMID: 29330707
47. Giles J, Reinholdt J, Andrade F, Konig M (2020) Associations of antibodies targeting periodontal pathogens with subclinical coronary, carotid, and peripheral arterial atherosclerosis in rheu-matoid arthritis. *Arthritis & Rheumatology* Nov 17. Online ahead of print. PMID: 33205531

48. Bracci P (2017) Oral health and the oral microbiome in pancreatic cancer: an overview of epidemiological studies. *Cancer Journal* 23:310-314. PMID: 29189325
49. Aoyama N, Kure K, Minabe M, Izumi Y (2019) Increased heart failure prevalence in patients with a high antibody level against periodontal pathogen. *International Heart Journal* 60:1142-1146. PMID: 31447467
50. Takamisawa K, Sugita N, Komatsu S et al. (2020) Association between serum IgG antibody titers against *Porphyromonas gingivalis* and liver enzyme levels: a cross-sectional study in Sado Island. *Heliyon* 6:e05531. PMID: 33294679
51. Nakamori M, Hosomi N, Nishi H et al. (2020) Serum IgG titers against periodontal pathogens are associated with cerebral hemorrhage growth and 3-month outcome. *PLoS One* 15:e0241205. PMID: 33112888
52. Tuominen H, Taina M, Puranen M et al. (2020) Serum high-sensitive C-reactive protein may reflect periodontitis in patients with stroke. *In Vivo* 34:2829-2835. PMID: 32871821
53. Zhang Y, Leveille S, Edward J (2020) Wisdom teeth, periodontal disease, and C-reactive protein in US adults. *Public Health* 187:97-102. PMID: 32942171

Chapter 5

1. Donlan R and Costerton J (2002) Biofilms: survival mechanisms of clinically relevant microorganisms. *Clinical Microbiology Reviews* 15:167-193. PMID: 11932229
2. Roy R, Tiwari M, Donelli G, Tiwari V (2018) Strategies for combating bacterial biofilms: a focus on anti-biofilm agents and their mechanisms of action. *Virulence* 9:522-554. PMID: 28362216
3. Das T, Sehar S, Manefield M (2013) The roles of extracellular DNA in the structural integrity of extracellular polymeric substance and bacterial biofilm development. *Environmental Microbiology Reports* 5:778-786. PMID: 24249286
4. Chiba A, Sugimoto S, Sato F et al. (2015) A refined technique for extraction of extracellular matrices from bacterial biofilms and its applicability. *Microbial Biotechnology* 8:392-403. PMID: 25154775
5. Gilbert P, Das J, Foley I (1997) Biofilm susceptibility to antimicrobials. *Advances in Dental Research* 11:160-167. PMID: 9524452
6. Hall-Stoodley L, Costerton J, Stoodley P (2004) Bacterial biofilms: from the natural environment to infectious diseases. *Nature Reviews. Microbiology* 2:95-108. PMID: 15040259
7. Parsek M, Singh P (2003) Bacterial biofilms: an emerging link to disease pathogenesis. *Annual Review of Microbiology* 57:677-701. PMID: 14527295
8. Fanning S, Mitchell A (2012) Fungal biofilms. *PLoS Pathogens* 8:e1002585. PMID: 22496639

9. Pais-Correia A, Sachse M, Guadagnini S et al. (2010) Biofilm-like extracellular viral assemblies mediate HTLV-1 cell-to-cell transmission at virological synapses. *Nature Medicine* 16:83-89. PMID: 20023636
10. Thoulouze M, Alcover A (2011) Can viruses form biofilms? *Trends in Microbiology* 19:257-262. PMID: 21458997
11. Kiedrowski M, Gaston J, Kocak B et al. (2018) *Staphylococcus aureus* biofilm growth on cystic fibrosis airway epithelial cells is enhanced during respiratory syncytial virus coinfection. *mSphere* 3:e00341-18. PMID: 30111629
12. Ascione C, Sala A, Mazaheri-Tehrani E et al. (2017) *Herpes simplex* virus-1 entrapped in *Candida albicans* biofilms displays decreased sensitivity to antivirals and UVA1 laser treatment. *Annals of Clinical Microbiology and Antimicrobials* 16:72. PMID: 29137671
13. Callow J, Callow M (2006) Biofilms. *Progress in Molecular and Subcellular Biology* 42:141-169. PMID: 16805442
14. Pinto G, Silva M, Peddey M et al. (2016) The role of bacteriophages in periodontal health and disease. *Future Microbiology* 11:1359-1369. PMID: 27633580
15. Bjarnsholt T (2013) The role of bacterial biofilms in chronic infections. *APMIS. Supplementum* 136:1-51. PMID: 23635385
16. Donlan R (2001) Biofilm formation: a clinically relevant microbiological process. *Clinical Infectious Diseases* 33:1387-1392. PMID: 11565080
17. Hoyle B, Costerton J (1991) Bacterial resistance to antibiotics: the role of biofilms. *Progress in Drug Research* 37:91-105. PMID: 1763187
18. Rabin N, Zheng Y, Opoku-Temeng C et al. (2015) Biofilm formation mechanisms and targets for developing antibiofilm agents. *Future Medicinal Chemistry* 7:493-512. PMID: 25875875
19. Hoyle B, Wong C, Costerton J (1992) Disparate efficacy of tobramycin on Ca(2+)-, Mg(2+)-, and HEPES-treated *Pseudomonas aeruginosa* biofilms. *Canadian Journal of Microbiology* 38:1214-1218. PMID: 1477794
20. Thurlow L, Hanke M, Fritz T et al. (2011) *Staphylococcus aureus* biofilms prevent macrophage phagocytosis and attenuate inflammation *in vivo*. *Journal of Immunology* 186:6585-6596. PMID: 21525381
21. Williams D, Kawaguchi B, Taylor N et al. (2020) *In vivo* efficacy of a unique first-in-class antibiofilm antibiotic for biofilm-related wound infections caused by *Acinetobacter baumannii*. *Biofilm* 2:100032. PMID: 33447817
22. Seneviratne C, Zhang C, Samaranayake L (2011) Dental plaque biofilm in oral health and disease. *The Chinese Journal of Dental Research* 14:87-94. PMID: 22319749

23. Hall-Stoodley L, Hu F, Gieseke A et al. (2006) Direct detection of bacterial biofilms on the middle-ear mucosa of children with chronic otitis media. *JAMA* 296:202-211. PMID: 16835426
24. Sochocka M, Zwolinska K, Leszek J (2017) The infectious etiology of Alzheimer's disease. *Current Neuropharmacology* 15:996-1009. PMID: 28294067
25. Laugisch O, Johnen A, Maldonado A et al. (2018) Periodontal pathogens and associated intrathecal antibodies in early stages of Alzheimer's disease. *Journal of Alzheimer's Disease* 66:105-114. PMID: 30223397
26. Matsushita K, Yamada-Furukawa M, Kurosawa M, Shikama Y (2020) Periodontal disease and periodontal disease-related bacteria involved in the pathogenesis of Alzheimer's disease. *Journal of Inflammation Research* 13:275-283. PMID: 32636667
27. Vigasova D, Nemergut M, Liskova B, Damborsky J (2021) Multi-pathogen infections and Alzheimer's disease. *Microbial Cell Factories* 20:25. PMID: 33509204
28. James G, Swogger E, Wolcott R et al. (2008) Biofilms in chronic wounds. *Wound Repair and Regeneration* 16:37-44. PMID: 18086294
29. Kania R, Lamers G, Vonk M et al. (2008) Characterization of mucosal biofilms on human adenoid tissues. *The Laryngoscope* 118:128-134. PMID: 17975509
30. Nistico L, Kreft R, Gieseke A et al. (2011) Adenoid reservoir for pathogenic biofilm bacteria. *Journal of Clinical Microbiology* 49:1411-1420. PMID: 21307211
31. Vestby L, Gronseth T, Simm R, Nesse L (2020) Bacterial biofilm and its role in the pathogenesis of disease. *Antibiotics* 9:59. PMID: 32028684
32. Lanter B, Sauer K, Davies D (2014) Bacteria present in carotid arterial plaques are found as biofilm deposits which may contribute to enhanced risk of plaque rupture. *mBio* 5:e01206-14. PMID: 24917599
33. Snow D, Everett J, Mayer G et al. (2016) The presence of biofilm structures in atherosclerotic plaques of arteries from legs amputated as a complication of diabetic foot ulcers. *Journal of Wound Care* 25:S16-S22. PMID: 26878370
34. Taymoortash A, Wollstein A, Lippert B et al. (2002) Bacteria and pathogenesis of human salivary calculus. *Acta Oto-Laryngologica* 122:210-214. PMID: 11936916
35. Schroder S, Eickhardt S, Bjarnsholt T et al. (2018) Morphological evidence of biofilm in chronic obstructive sialadenitis. *The Journal of Laryngology and Otology* 132:611-614. PMID: 29986787
36. Perez-Tanoira R, Aarnisalo A, Haapaniemi A et al. (2019) Bacterial biofilm in salivary stones. *European Archives of Oto-Rhino-Laryngology* 276:1815-1822. PMID: 31028534

37. Kao W, Chole R, Ogden M (2020) Evidence of a microbial etiology for sialoliths. *The Laryngoscope* 130:69-74. PMID: 30861582
38. Crawford R, Rosales-Reyes R, Ramirez-Aguilar M et al. (2010) Gallstones play a significant role in *Salmonella* spp. Gallbladder colonization and carriage. *Proceedings of the National Academy of Sciences of the United States of America* 107:4353-4358. PMID: 20176950
39. Prouty A, Schwesinger W, Gunn J (2002) Biofilm formation and interaction with the surfaces of gallstones by *Salmonella* spp. *Infection and Immunity* 70:2640-2649. PMID: 11953406
40. Marshall J, Flechtner A, La Perle K, Gunn J (2014) Visualization of extracellular matrix components within sectioned *Salmonella* biofilms on the surface of human gallstones. *PLoS One* 9:e89243. PMID: 24551241
41. Swidsinski A, Weber J, Loening-Baucke V et al. (2005) Spatial organization and composition of the mucosal flora in patients with inflammatory bowel disease. *Journal of Clinical Microbiology* 43:3380-3389. PMID: 16000463
42. Swidsinski A, Loening-Baucke V, Herber A (2009) Mucosal flora in Crohn's disease and ulcerative colitis—an overview. *Journal of Physiology and Pharmacology* 60 Suppl 6:61-71. PMID: 20224153
43. Motta J, Wallace J, Buret A et al. (2021) Gastrointestinal biofilms in health and disease. *Nature Reviews. Gastroenterology & Hepatology* Jan 28. Online ahead of print. PMID: 33510461
44. Li S, Konstantinov S, Smits R, Peppelenbosch M (2017) Bacterial biofilms in colorectal initiation and progression. *Trends in Molecular Medicine* 23:18-30. PMID: 27986421
45. Machado D, Castro J, Palmeira-de-Oliveira A et al. (2016) Bacterial vaginosis biofilms: challenges to current therapies and emerging solutions. *Frontiers in Microbiology* 6:1528. PMID: 26834706
46. Castro J, Machado D, Cerca N (2019) Unveiling the role of *Gardnerella vaginalis* in polymicrobial bacterial vaginosis biofilms: the impact of other vaginal pathogens living as neighbors. *The ISME Journal* 13:1306-1317. PMID: 30670827
47. Swidsinski A, Verstraeten H, Loening-Baucke V et al. (2013) Presence of a polymicrobial endometrial biofilm in patients with bacterial vaginosis. *PLoS One* 8:e53997. PMID: 23320114
48. Ramakrishnan Y, Shields R, Elbadawey M, Wilson J (2015) Biofilms in chronic rhinosinusitis: what is new and where next? *The Journal of Laryngology and Otology* 129:744-751. PMID: 26120023
49. Manciula L, Jeican I, Tudoran L, Albu S (2020) Biofilms and inflammation in patients with chronic rhinosinusitis. *Medicine and Pharmacy Reports* 93:374-383. PMID: 33225263

50. Kinnari T, Lampikoski H, Hyyrynen T, Aarnisalo A (2012) Bacterial biofilm associated with chronic laryngitis. *Archives of Otolaryngology—Head & Neck Surgery* 138:467-470. PMID: 22652944
51. Kinnari T (2015) The role of biofilm in chronic laryngitis and in head and neck cancer. *Current Opinion in Otolaryngology & Head and Neck Surgery* 23:448-453. PMID: 26371604
52. Singh P, Schaefer A, Parsek M et al. (2000) Quorum-sensing signals indicate that cystic fibrosis lungs are infected with bacterial biofilms. *Nature* 407:762-764. PMID: 11048725
53. Starner T, Zhang N, Kim G et al. (2006) *Haemophilus influenzae* forms biofilms on airway epithelia: implications in cystic fibrosis. *American Journal of Respiratory and Critical Care Medicine* 174:213-220. PMID: 16675778
54. Hoiby N, Ciofu O, Bjarnsholt T (2010) *Pseudomonas aeruginosa* biofilms in cystic fibrosis. *Future Microbiology* 5:1663-1674. PMID: 21133688
55. Hector A, Frey N, Hartl D (2016) Update on host-pathogen interactions in cystic fibrosis lung disease. *Molecular and Cellular Pediatrics* 3:12. PMID: 26905568
56. Scott V, Haake D, Churchill B et al. (2015) Intracellular bacterial communities: a potential etiology for chronic lower urinary tract symptoms. *Urology* 86:425-431. PMID: 26189137
57. Kanamura S, Kurazono H, Terai A et al. (2006) Increased biofilm formation in *Escherichia coli* isolated from acute prostatitis. *International Journal of Antimicrobial Agents* 28 Suppl 1:S21-S25. PMID: 16828264
58. Mazzoli S (2010) Biofilms in chronic bacterial prostatitis (NIH-II) and in prostatic calcifications. *FEMS Immunology and Medical Microbiology* 59:337-344. PMID: 20298500
59. Bartoletti R, Cai T, Nesi G et al. (2014) The impact of biofilm-producing bacteria on chronic bacterial prostatitis treatment: results from a longitudinal cohort study. *World Journal of Urology* 32:737-742. PMID: 23918259
60. Cai T, Tessarolo F, Caola I et al. (2018) Prostate calcifications: a case series supporting the microbial biofilm theory. *Investigative and Clinical Urology* 59:187-193. PMID: 29744476
61. Davis S, Ricotti C, Cazzaniga A et al. (2008) Microscopic and physiologic evidence for biofilm-associated wound colonization *in vivo*. *Wound Repair and Regeneration* 16:23-29. PMID: 18211576
62. Barnes B, Galton L (1976) *Hypothyroidism: The Unsuspected Illness*. New York, NY: Harper & Row
63. Glynn A, O'Donnell S, Molony D et al. (2009) Hydrogen peroxide induced repression of icaADBC transcription and biofilm development in *Staphylococcus epidermidis*. *Journal of Orthopaedic Research* 27:627-630. PMID: 18942741

64. Wu Y, Zhu H, Willcox M, Stapleton F (2011) Impact of cleaning regimens in silver-impregnated and hydrogen peroxide lens cases. *Eye & Contact Lens* 37:365-369. PMID: 21983551
65. Lineback C, Nkemngong C, Wu S et al. (2018) Hydrogen peroxide and sodium hypochlorite disinfectants are more effective against *Staphylococcus aureus* and *Pseudomonas aeruginosa* biofilms than quaternary ammonium compounds. *Antimicrobial Resistance and Infection Control* 7:154. PMID: 30568790
66. Watson F, Keevil C, Wilks S, Chewins J (2018) Modelling vapourised hydrogen peroxide efficacy against mono-species biofilms. *Scientific Reports* 8:12257. PMID: 30115938
67. Clifford D, Repine J (1982) Hydrogen peroxide mediated killing of bacteria. *Molecular and Cellular Biochemistry* 49:143-149. PMID: 6298593
68. Zhu G, Wang Q, Lu S, Niu Y (2017) Hydrogen peroxide: a potential wound therapeutic target? *Medical Principles and Practice* 26:301-308. PMID: 28384636
69. Knight G, McIntyre J, Craig G et al. (2008) The inability of *Streptococcus mutans* and *Lactobacillus acidophilus* to form a biofilm *in vitro* on dentine pretreated with ozone. *Australian Dental Journal* 53:349-353. PMID: 19133951
70. Suh Y, Patel S, Kaitlyn R et al. (2019) Clinical utility of ozone therapy in dental and oral medicine. *Medical Gas Research* 9:163-167. PMID: 31552882
71. Tonon C, Panariello B, Spolidorio D et al. (2020) Anti-biofilm effect of ozonized physiological saline solution on peri-implant-related biofilm. *Journal of Periodontology* Nov 24. Online ahead of print. PMID: 33231303
72. Rowen R (2019) Ozone and oxidation therapies as a solution to the emerging crisis in infectious disease management: a review of current knowledge and experience. *Medical Gas Research* 9:232-237. PMID: 31898609
73. Eydou Z, Jad B, Elsayed Z et al. (2020) Investigation on the effect of vitamin C on growth & biofilm-forming potential of *Streptococcus mutans* isolated from patients with dental caries. *BMC Microbiology* 20:231. PMID: 32731889
74. Kirmusaoglu S, Kasikci H (2020) Identification of *ica*-dependent biofilm production by *Staphylococcus aureus* clinical isolates and antibiofilm effects of ascorbic acid against biofilm production. *Journal of Clinical Pathology* 73:261-266. PMID: 32213553
75. Silva H, de Souza G, Fernandes J et al. (2020) Unravelling the effects of the food components ascorbic acid and capsaicin as a novel anti-biofilm agent against *Escherichia coli*. *Journal of Food Science and Technology* 57:1013-1020. PMID: 32123422

76. Syal K, Chatterji D (2018) Vitamin C: a natural inhibitor of cell wall functions and stress response in *Mycobacteria*. *Advances in Experimental Medicine and Biology* 1112:321-332. PMID: 30637707
77. Majtan J, Sojka M, Palenikova H et al. (2020) Vitamin C enhances the antibacterial activity of honey against planktonic and biofilm-embedded bacteria. *Molecules* 25:992. PMID: 32102181
78. Pei Z, Wu K, Li Z et al. (2019) Pharmacologic ascorbate as a pro-drug for hydrogen peroxide release to kill mycobacteria. *Biomedicine & Pharmacotherapy* 109:2119-2127. PMID: 30551469
79. Oknin H, Steinberg D, Shemesh M (2015) Magnesium ions mitigate biofilm formation of *Bacillus* species via downregulation of matrix genes expression. *Frontiers in Microbiology* 6:907. PMID: 26441856
80. Guo Q, Wu Q, Bai D et al. (2016) Potential use of dimethyl sulfoxide in treatment of infections caused by *Pseudomonas aeruginosa*. *Antimicrobial Agents and Chemotherapy* 60:7159-7169. PMID: 27645245
81. Yahya M, Alias Z, Karsani S (2018) Antibiofilm activity and mode of action of DMSO alone and its combination with afatinib against Gram-negative pathogens. *Folia Microbiologica* 63:23-30. PMID: 28540585
82. Jacob S, Herschler R (1986) Pharmacology of DMSO. *Cryobiology* 23:14-27. PMID: 3007027
83. Guo Q, Wu Q, Bai D et al. (2016) Potential use of dimethyl sulfoxide in treatment of infections caused by *Pseudomonas aeruginosa*. *Antimicrobial Agents and Chemotherapy* 60:7159-7169. PMID: 27645245
84. Mogosanu G, Grumezescu M, Huang K et al. (2015) Prevention of microbial communities: novel approaches based natural products. *Current Pharmaceutical Biotechnology* 16:94-111. PMID: 25594287
85. Francolini I, Piozzi A (2020) Role of antioxidant molecules and polymers in prevention of bacterial growth and biofilm formation. *Current Medicinal Chemistry* 27:4882-4904. PMID: 30963965
86. Ong K, Mawang C, Daniel-Jambun et al. (2018) Current anti-biofilm strategies and potential of antioxidants in biofilm control. *Expert Review of Anti-Infective Therapy* 16:855-864. PMID: 30308132
87. Wu C, Labrie J, Tremblay Y et al. (2013) Zinc as an agent for the prevention of biofilm formation by pathogenic bacteria. *Journal of Applied Microbiology* 115:30-40. PMID: 23509865
88. Bhattacharyya P, Agarwal B, Goswami M et al. (2018) Zinc oxide nanoparticle inhibits the biofilm formation of *Streptococcus pneumoniae*. *Antonie van Leeuwenhoek* 111:89-99. PMID: 28889242

89. Huang Z, Wu L, Li X et al. (2020) Zn(II) suppresses biofilm formation in *Bacillus amyloliquefaciens* by inactivation of the Mn(II) uptake. *Environmental Microbiology* 22:1547-1558. PMID: 31715659
90. Vergalito F, Pietrangelo L, Petronio G et al. (2019) Vitamin E for prevention of biofilm-caused healthcare-associated infections. *Open Medicine* 15:14-21. PMID: 31922015
91. Bigliardi P, Alsagoff S, El-Kafrawi H et al. (2017) Povidone iodine in wound healing: a review of current concepts and practices. *International Journal of Surgery* 44:260-268. PMID: 28648795

Chapter 6

1. Zhou Y, Luo G (2019) *Porphyromonas gingivalis* and digestive system cancers. *World Journal of Clinical Cases* 7:819-829. PMID: 31024953
2. Mei F, Xie M, Huang X et al. (2020) *Porphyromonas gingivalis* and its systemic impact: current status. *Pathogens* 9:944. PMID: 33202751
3. Fiorillo L, Cervino G, Laino L et al. (2019) *Porphyromonas gingivalis*, periodontal and systemic implications: a systematic review. *Dentistry Journal* 7:114. PMID: 31835888
4. Janakiram C, Mehta A, Venkitachalam R (2020) Prevalence of periodontal disease among adults in India: a systematic review and meta-analysis. *Journal of Oral Biology and Craniofacial Research* 10:800-806. PMID: 33204609
5. Nozawa A, Oshima H, Togawa N et al. (2020) Development of Oral Care Chip, a novel device for quantitative detection of the oral microbiota associated with periodontal disease. *PLoS One* 15:e0229485. PMID: 32109938
6. Romandini M, Baima G, Antonoglou G et al. (2020) Periodontitis, edentulism, and risk of mortality: a systematic review with meta-analysis. *Journal of Dental Research* Aug 31. Online ahead of print. PMID: 32866427
7. Larvin H, Kang J, Aggarwal V et al. (2020) Risk of incident cardiovascular disease in people with periodontal disease: a systematic review and meta-analysis. *Clinical and Experimental Dental Research* Oct 30. Online ahead of print. PMID: 33124761
8. Franek E, Napora M, Blach A et al. (2010) Blood pressure and left ventricular mass in subjects with type 2 diabetes and gingivitis or chronic periodontitis. *Journal of Clinical Periodontology* 37:875-880. PMID: 20796107
9. Foratori-Junior G, Mascoli L, Marchese C et al. (2020) Association between arterial hypertension and periodontal status in morbidly obese patients who are candidates for bariatric surgery. *International Dental Journal* Oct 10. Online ahead of print. PMID: 33040359

10. Pietropaoli D, Monaco A, D'Aiuto F et al. (2020) Acute gingival inflammation is linked to hypertension. *Journal of Hypertension* 38:2018-2027. PMID: 32890278
11. Beck J, Philips K, Moss K et al. (2020) Periodontal disease classifications and incident coronary heart disease in the Atherosclerosis Risk in Communities study. *Journal of Periodontology* 91:1409-1418. PMID: 32449797
12. Byon M, Kim S, Kim J et al. (2020) Association of periodontitis with atherosclerotic cardiovascular diseases: a nationwide population-based retrospective matched cohort study. *International Journal of Environmental Research and Public Health* 17:7261. PMID: 33020434
13. Nikolaeva E, Tsarev V, Tsareva T et al. (2019) Interrelation of cardiovascular diseases with anaerobic bacteria of subgingival biofilm. *Contemporary Clinical Dentistry* 10:637-642. PMID: 32792823
14. Aoyama N, Kure K, Minabe M, Izumi Y (2019) Increased heart failure prevalence in patients with a high antibody level against periodontal pathogen. *International Heart Journal* 60:1142-1146. PMID: 31447467
15. Sandi R, Pol K, Basavaraj P et al. (2014) Association of serum cholesterol, triglyceride, high and low density lipoprotein (HDL and LDL) levels in chronic periodontitis subjects with risk for cardiovascular disease (CVD): a cross sectional study. *Journal of Clinical and Diagnostic Research* 8:214-216. PMID: 24596778
16. Gomes-Filho I, Balinha I, da Cruz S et al. (2020) Moderate and severe periodontitis are positively associated with metabolic syndrome. *Clinical Oral Investigations* Nov 23. Online ahead of print. PMID: 33226499
17. Dewake N, Ishioka Y, Uchida K et al. (2020) Association between carotid artery calcification and periodontal disease progression in Japanese men and women: a cross-sectional study. *Journal of Clinical Medicine* 9:3365. PMID: 33092208
18. Jimenez M, Krall E, Garcia R et al. (2009) Periodontitis and incidence of cerebrovascular disease in men. *Annals of Neurology* 66:505-512. PMID: 19847898
19. Slowik J, Wnuk M, Grzech K et al. (2010) Periodontitis affects neurological deficit in acute stroke. *Journal of the Neurological Sciences* 297:82-84. PMID: 20723913
20. Lafon A, Pereira B, Dufour T et al. (2014) Periodontal disease and stroke: a meta-analysis of cohort studies. *European Journal of Neurology* 21:1155-1161. PMID: 24712659
21. Palm F, Lahdentauta L, Sorsa T et al. (2014) Biomarkers of periodontitis and inflammation in ischemic stroke: a case-control study. *Innate Immunity* 20:511-518. PMID: 24045341

22. Lin H, Chen C, Yeh Y et al. (2019) Dental treatment procedures for periodontal disease and the subsequent risk of ischaemic stroke: a retrospective population-based cohort study. *Journal of Clinical Periodontology* 46:642-649. PMID: 30989681
23. Sen S, Mascari R (2020) Exploring the periodontal disease–ischemic stroke link. *Journal of Periodontology* 91 Suppl 1:S35-S39. PMID: 32592499
24. Patel U, Malik P, Kodumuri et al. (2020) Chronic periodontitis is associated with cerebral atherosclerosis—a nationwide study. *Cureus* 12:e11373. PMID: 33304705
25. Pyysalo M, Pyysalo L, Hiltunen J et al. (2018) The dental infections in patients undergoing preoperative dental examination before surgical treatment of saccular intracranial aneurysm. *BMC Research Notes* 11:600. PMID: 30126459
26. Hallikainen J, Kieranen S, Savolainen J et al. (2020) Role of oral pathogens in the pathogenesis of intracranial aneurysm: review of existing evidence and potential mechanisms. *Neurosurgical Review* 44:239-247. PMID: 32034564
27. Takahashi M, Nakanishi Y, Hamada Y et al. (2020) A case of brain abscess caused by *Actinomyces cardifensis* and *Parvimonas micra*. *The Tokai Journal of Experimental and Clinical Medicine* 45:189-194. PMID: 33300589
28. da Silva R, Caugant D, Eribé E et al. (2006) Bacterial diversity in aortic aneurysms determined by 16S ribosomal RNA gene analysis. *Journal of Vascular Surgery* 44:1055-1060. PMID: 17098542
29. Iwai T (2009) Periodontal bacteremia and various vascular diseases. *Journal of Periodontal Research* 44:689-694. PMID: 19874452
30. Heji E, Bukhari A, Bahammam M et al. (2020) Periodontal disease as a predictor of undiagnosed diabetes or prediabetes in dental patients. *European Journal of Dentistry* Dec 7. Online ahead of print. PMID: 33285572
31. Quadri M, Fageeh H, Ibraheem W, Jessani A (2020) A case-control study of type 2 diabetes mellitus and periodontitis in Saudi Arabian adults. *Journal of Multidisciplinary Healthcare* 13:1741-1748. PMID: 33273822
32. Gulati N, Masamatti S, Chopra P (2020) Association between obesity and its determinants with chronic periodontitis: a cross-sectional study. *Journal of Indian Society of Periodontology* 24:167-172. PMID: 32189846
33. Khan M, Alasqah M, Alammar L, Alkhaibari Y (2020) Obesity and periodontal disease: a review. *Journal of Family Medicine and Primary Care* 9:2650-2653. PMID: 32984101
34. Dev Y, Goyal O (2013) Recurrent lung infection due to chronic periodontitis. *Journal of the Indian Medical Association* 111:127. PMID: 24003573

35. Gomes-Filho I, de Oliveira T, da Cruz S et al. (2014) Influence of periodontitis in the development of nosocomial pneumonia: a case control study. *Journal of Periodontology* 85:e82-e90. PMID: 24171504
36. Gomes-Filho I, Soledade-Marques K, da Cruz S et al. (2014) Does periodontal infection have an effect on severe asthma in adults? *Journal of Periodontology* 85:e179-e187. PMID: 24224961
37. Zhou X, Han J, Liu Z et al. (2014) Effects of periodontal treatment on lung function and exacerbation frequency in patients with chronic obstructive pulmonary disease and chronic periodontitis: a 2-year pilot randomized controlled trial. *Journal of Clinical Periodontology* 41:564-572. PMID: 24593836
38. Brasil-Oliveira R, Cruz A, Souza-Machado A et al. (2020) Oral health-related quality of life in individuals with severe asthma. *Jornal Brasileiro de Pneumologia* 47:e20200117. PMID: 33174972
39. Wee J, Yoo D, Byun S et al. (2020) Subjective oral health status in an adult Korean population with asthma or allergic rhinitis. *Medicine* 99:e22967. PMID: 33120860
40. She Y, Kong X, Ge Y et al. (2020) Periodontitis and inflammatory bowel disease: a meta-analysis. *BMC Oral Health* 20:67. PMID: 32164696
41. Leira Y, Ameijeira P, Dominguez C et al. (2020) Severe periodontitis is linked with increased peripheral levels of sTWEAK and PTX3 in chronic migraineurs. *Clinical Oral Investigations* 24:597-606. PMID: 31111284
42. Stein P, Steffen M, Smith C et al. (2012) Serum antibodies to periodontal pathogens are a risk factor for Alzheimer's disease. *Alzheimer's & Dementia* 8:196-203. PMID: 22546352
43. Sochocka M, Zwolinska K, Leszek J (2017) The infectious etiology of Alzheimer's disease. *Current Neuropharmacology* 15:996-1009. PMID: 28294067
44. Ding Y, Ren J, Yu H et al. (2018) *Porphyromonas gingivalis*, a periodontitis causing bacterium, induces memory impairment and age-dependent neuroinflammation in mice. *Immunity & Ageing* 15:6. PMID: 29422938
45. Botelho J, Mascarenhas P, Mendes J, Machado V (2020) Network protein interaction in Parkinson's disease and periodontitis interplay: a preliminary bioinformatic analysis. *Genes* 11:1385. PMID: 33238395
46. Costa A, Yasuda C, Shibasaki W et al. (2014) The association between periodontal disease and seizure severity in refractory epilepsy patients. *Seizure* 23:227-230. PMID: 24456623
47. Aldosari M, Helmi M, Kennedy E et al. (2020) Depression, periodontitis, caries and missing teeth in the USA, NHANES 2009-2014. *Family Medicine and Community Health* 8:e000583. PMID: 33303491

48. Chang K, Hsu Y, Chiu I et al. (2020) Association between periodontitis and bipolar disorder: a nationwide cohort study. *Medicine* 99:e21423. PMID: 32756145
49. Tzeng N, Chung C, Yeh C et al. (2016) Are chronic periodontitis and gingivitis associated with dementia? A nationwide, retrospective, matched-cohort study in Taiwan. *Neuroepidemiology* 47:82-93. PMID: 27618156
50. Demmer R, Norby F, Lakshminarayan K et al. (2020) Periodontal disease and incident dementia: The Atherosclerosis Risk in Communities Study (ARIC). *Neurology* 95:e1660-e1671. PMID: 32727837
51. Ogendrik M (2013) Rheumatoid arthritis is an autoimmune disease caused by periodontal pathogens. *International Journal of General Medicine* 6:383-386. PMID: 23737674
52. Gomez-Banuelos E, Mukherjee A, Darrah E, Andrade F (2019) Rheumatoid arthritis-associated mechanisms of *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans*. *Journal of Clinical Medicine* 8:1309. PMID: 31454946
53. Pandey A, Rajak R, Pandey M (2020) Periodontal diseases and its association with disease activity in ankylosing spondylitis/SpA: a systematic review. *European Journal of Rheumatology* Dec 1. Online ahead of print. PMID: 33284102
54. Skeie M, Gil E, Cetrella L et al. (2019) Oral health in children and adolescents with juvenile idiopathic arthritis—a systematic review and meta-analysis. *BMC Oral Health* 19:285. PMID: 31856793
55. Disale P, Zope S, Suragimath G et al. (2020) Prevalence and severity of periodontitis in patients with established rheumatoid arthritis and osteoarthritis. *Journal of Family Medicine and Primary Care* 9:2919-2925. PMID: 32984149
56. Katz J, Rotstein I (2020) Prevalence of periapical lesions in patients with osteoporosis. *Journal of Endodontics* Oct 29. Online ahead of print. PMID: 33130060
57. Xu S, Zhang G, Guo J, Tan Y (2020) Associations between osteoporosis and risk of periodontitis: a pooled analysis of observational studies. *Oral Diseases* Jul 2. Online ahead of print. PMID: 32615008
58. Acharya C, Sahingur S, Bajaj J (2017) Microbiota, cirrhosis, and the emerging oral-gut-liver axis. *JCI Insight* 2:e94416. PMID: 28978799
59. Mohammed H, Varoni E, Cochis A et al. (2018) Oral dysbiosis in pancreatic cancer and liver cirrhosis: a review of the literature. *Biomedicines* 6:115. PMID: 30544974
60. Gerlovin H, Michaud D, Cozier Y, Palmer J (2019) Oral health in relation to pancreatic cancer risk in African American women. *Cancer Epidemiology, Biomarkers, & Prevention* 28:675-679. PMID: 30923045

61. Wang L, Yang X, Zou X et al. (2020) Relationship between periodontal disease and lung cancer: a systematic review and meta-analysis. *Journal of Periodontal Research* 55:581-593. PMID: 32583879
62. Chen Y, Yang Y, Zhu B et al. (2020) Association between periodontal disease, tooth loss and liver diseases risk. *Journal of Clinical Periodontology* 47:1053-1063. PMID: 32621350
63. Di Spirito F, Toti P, Pilone V et al. (2020) The association between periodontitis and human colorectal cancer: genetic and pathogenic linkage. *Life* 10:211. PMID: 32962181
64. Xuen K, Jha A, Zhao T et al. (2020) Is periodontal disease associated with increased risk of colorectal cancer? A meta-analysis. *International Journal of Dental Hygiene* Dec 2. Online ahead of print. PMID: 33269543
65. Kawasaki M, Ikeda Y, Ikeda E et al. (2020) Oral infectious bacteria in dental plaque and saliva as risk factors in patients with esophageal cancer. *Cancer* Nov 6. Online ahead of print. PMID: 33156979
66. Velly A, Franco E, Schlecht N et al. (1998) Relationship between dental factors and risk of upper aerodigestive tract cancer. *Oral Oncology* 34:284-291. PMID: 9813724
67. Kageyama S, Takeshita T, Takeuchi K et al. (2019) Characteristics of the salivary microbiota in patients with various digestive tract cancers. *Frontiers in Microbiology* 10:1780. PMID: 31428073
68. Mathur R, Singhavi H, Malik A et al. (2019) Role of poor oral hygiene in causation of oral cancer—a review of literature. *Indian Journal of Surgical Oncology* 10:184-195. PMID: 30948897
69. Gopinath D, Menon R, Veettil S et al. (2020) Periodontal diseases as putative risk factors for head and neck cancer: systematic review and meta-analysis. *Cancers* 12:1893. PMID: 32674369
70. Payao S, Rasmussen L (2016) *Helicobacter pylori* and its reservoirs: a correlation with the gastric infection. *World Journal of Gastrointestinal Pharmacology and Therapeutics* 7:126-132. PMID: 26855818
71. Ma H, Zheng J, Li X (2020) Potential risk of certain cancers among patients with periodontitis: a supplementary meta-analysis of a large-scale population. *International Journal of Medical Sciences* 17:2531-2543. PMID: 33029095
72. Sun J, Tan Q, Yu S et al. (2020) Role of the oral microbiota in cancer evolution and progression. *Cancer Medicine* 9:6306-6321. PMID: 32638533
73. Figuero E, Han Y, Furuichi Y (2020) Periodontal diseases and adverse pregnancy outcomes: mechanisms. *African Health Sciences* 83:175-188. PMID: 32385886

74. Heo J, Ahn K, Park J (2020) Radiological screening of maternal periodontitis for predicting adverse pregnancy and neonatal outcomes. *Scientific Reports* 10:21266. PMID: 33277556
75. Kothiwale S, Desai B, Kothiwale V et al. (2014) Periodontal disease as a potential risk factor for low birth weight and reduced maternal haemoglobin levels. *Oral Health & Preventive Dentistry* 12:83-90. PMID: 24619787
76. Wu D, Lin Z, Zhang S et al. (2020) Decreased hemoglobin concentration and iron metabolism disorder in periodontitis: systematic review and meta-analysis. *Frontiers in Physiology* 10:1620. PMID: 32082180
77. Adulaijan H, Cohen R, Stellrecht E et al. (2020) Relationship between hypothyroidism and periodontitis: a scoping review. *Clinical and Experimental Dental Research* 6:147-157. PMID: 32067402
78. Chau S, Lee C, Huang J et al. (2020) The existence of periodontal disease and subsequent ocular diseases: a population-based cohort study. *Medicina* 56:621. PMID: 33218003
79. Sun K, Shen T, Chen S et al. (2020) Periodontitis and the subsequent risk of glaucoma: results from the real-world practice. *Scientific Reports* 10:17568. PMID: 33067540
80. Wu C, Yang T, Lin H et al. (2013) Sudden sensorineural hearing loss associated with chronic periodontitis: a population-based study. *Otology & Neurotology* 34:1380-1384. PMID: 24026022
81. Costa A, Cota L, Mendes V et al. (2020) Periodontitis and the impact of oral health on the quality of life of psoriatic individuals: a case-control study. *Clinical Oral Investigations* Sep 21. Online ahead of print. PMID: 32955692
82. Young H, Ward W (2020) The relationship between polycystic ovarian syndrome, periodontal disease, and osteoporosis. *Reproductive Sciences* Sep 10. Online ahead of print. PMID: 32914348
83. Lee J, Jeong S (2020) A population-based study on the association between periodontal disease and major lifestyle-related comorbidities in South Korea: an elderly cohort study from 2002-2015. *Medicina* 56:575. PMID: 33138320
84. Machado V, Lopes J, Patrao M et al. (2020) Validity of the association between periodontitis and female infertility conditions: a concise review. *Reproduction* 160:R41-R54. PMID: 32716008
85. Hickey N, Shalamanova L, Whitehead K et al. (2020) Exploring the putative interactions between chronic kidney disease and chronic periodontitis. *Critical Reviews in Microbiology* 46:61-77. PMID: 32046541
86. Schutz J, Azambuja C, Cunha G et al. (2020) Association between severe periodontitis and chronic kidney disease severity in prediabetic patients: a cross-sectional study. *Oral Diseases* 26:447-456. PMID: 31742816

87. Yue H, Xu X, Liu Q et al. (2020) Effects of non-surgical periodontal therapy on systemic inflammation and metabolic markers in patients undergoing haemodialysis and/or peritoneal dialysis: a systematic review and meta-analysis. *BMC Oral Health* 20:18. PMID: 31969148
88. Degasperi G, Ossick M, Pinheiro S, Etchegaray A (2020) Autoimmunity and periodontal disease: arguing a possible correlation. *Indian Journal of Dental Research* 31:615-620. PMID: 33107465
89. Benli M, Batool F, Stutz C et al. (2019) Orofacial manifestations and dental management of systemic lupus erythematosus: a review. *Oral Diseases* Dec 30. Online ahead of print. PMID: 31886584
90. Pessoa L, Aleti G, Choudhury S et al. (2019) Host-microbial interactions in systemic lupus erythematosus and periodontitis. *Frontiers in Immunology* 10:2602. PMID: 31781106
91. Manchery N, Henry J, Nangle M (2020) A systematic review of oral health in people with multiple sclerosis. *Community Dentistry and Oral Epidemiology* 48:89-100. PMID: 31815299
92. Costa F, Esteves-Lima R, Cortelli S et al. (2020) Effect of compliance during periodontal maintenance therapy on C-reactive protein levels: a 6-year follow-up. *Journal of Clinical Periodontology* Dec 1. Online ahead of print. PMID: 33259118
93. Esteves-Lima R, Reis C, Santirocchi-Junior F et al. (2020) Association between periodontitis and serum C-reactive protein levels. *Journal of Clinical and Experimental Dentistry* 12:e838-e843. PMID: 32994872
94. Gupta S, Suri P, Patil P et al. (2020) Comparative evaluation of role of hs-C-reactive protein as a diagnostic marker in chronic periodontitis patients. *Journal of Family Medicine and Primary Care* 9:1340-1347. PMID: 32509613
95. Isola G, Alibrandi A, Rapisarda E et al. (2020) Association of vitamin D in patients with periodontitis: a cross-sectional study. *Journal of Periodontal Research* 55:602-612. PMID: 32173876
96. Munday M, Rodricks R, Fitzpatrick M et al. (2020) A pilot study examining vitamin C levels in periodontal patients. *Nutrients* 12:2255. PMID: 32731485
97. Richenbacher O, Filippi C, Zurcher A, Filippi A (2019) Acceptance of a tongue vacuum cleaner among children and evaluation of tongue cleaning at home. *Swiss Dental Journal* 129:102-107. PMID: 30647023
98. Kulacz R, Levy T (2014) *The Toxic Tooth: How a root canal could be making you sick*, Henderson, NV: MedFox Publishing
99. Levy T (2017) *Hidden Epidemic: Silent oral infections cause most heart attacks and breast cancers*, Henderson, NV: MedFox Publishing

100. Gao L, Liu P, Song J (2010) [Relationship between tongue presentations and serum level of C-reactive protein in patients with acute cerebral infarction]. Article in Chinese. *Chinese Journal of Integrated Traditional and Western Medicine* 30:1146-1148. PMID: 21275163
101. Pedrazzi V, Sato S, de Mattos M et al. (2004) Tongue-cleaning methods: a comparative clinical trial employing a toothbrush and a tongue scraper. *Journal of Periodontology* 75:1009-1012. PMID: 15341360
102. Outhouse T, Fedorowicz Z, Keenan J, Al-Alawi R (2006) A Cochrane systematic review finds tongue scrapers have short-term efficacy in controlling halitosis. *General Dentistry* 54:352-360. PMID: 17004573
103. Danser M, Gomez S, Van der Weijden G (2003) Tongue coating and tongue brushing: a literature review. *International Journal of Dental Hygiene* 1:151-158. PMID: 16451515
104. Amou T, Hinode D, Yoshioka M, Grenier D (2014) Relationship between halitosis and periodontal disease—associated oral bacteria in tongue coatings. *International Journal of Dental Hygiene* 12:145-151. PMID: 23890391
105. Cherel F, Mobilia A, Lundgren T et al. (2008) Rate of reformation of tongue coatings in young adults. *International Journal of Dental Hygiene* 6:371-375. PMID: 19138189

Chapter 8

1. Lee S (2015) Intestinal permeability regulation by tight junction: implication on inflammatory bowel diseases. *Intestinal Research* 13:11-18. PMID: 25691839
2. Kelly C, Bai J, Liu E, Leffler D (2015) Advances in diagnosis and management of celiac disease. *Gastroenterology* 148:1175-1186. PMID: 25662623
3. Ye B, McGovern D (2016) Genetic variation in IBD: progress, clues to pathogenesis and possible clinical utility. *Expert Review of Clinical Immunology* 12:1091-1107. PMID: 27156530
4. Bhattacharai Y, Pedrogo D, Kashyap P (2017) Irritable bowel syndrome: a gut microbiota-related disorder? *American Journal of Physiology. Gastrointestinal and Liver Physiology* 312:G52-G62. PMID: 27881403
5. Rogler G, Hausmann M (2019) The long and winding road: from genetic risk factors to the understanding of disease-pathogenesis in Crohn's disease. *Genes and Immunity* 20:607-608. PMID: 30773533

Chapter 9

1. Schooley R (2018) The human microbiome: implications for health and disease, including HIV infection. *Topics in Antiviral Medicine* 26:75-78. PMID: 3038432
2. Lezutekong J, Nikhanj A, Oudit G (2018) Imbalance of gut microbiome and intestinal epithelial barrier dysfunction in cardiovascular disease. *Clinical Science* 132:901-904. PMID: 29712884
3. Obrenovich M (2018) Leaky gut, leaky brain? *Microorganisms* 6:107. PMID: 30340384
4. Belkaid Y, Harrison O (2017) Homeostatic immunity and the microbiota. *Immunity* 46:562-576. PMID: 28423337
5. Barko P, McMichael M, Swanson K, Williams D (2018) The gastrointestinal microbiome: a review. *Journal of Veterinary Internal Medicine* 32:9-25. PMID: 29171095
6. Maruvada P, Leone V, Kaplan L, Chang E (2017) The human microbiome and obesity: moving beyond associations. *Cell Host & Microbe* 22:589-599. PMID: 29120742
7. Pushalkar S, Hundeyin M, Daley D et al. (2018) The pancreatic cancer microbiome promotes oncogenesis by induction of innate and adaptive immune suppression. *Cancer Discovery* 8:403-416. PMID: 29567829
8. Aykut B, Pushalkar S, Chen R et al. (2019) The fungal microbiome promotes pancreatic oncogenesis via activation of MBL. *Nature* 574:264-267. PMID: 31578522
9. Mendoza L (2019) Potential effect of probiotics in the treatment of breast cancer. *Oncology Reviews* 13:422. PMID: 31583054
10. Song M, Chan A, Sun J (2020) Influence of the gut microbiome, diet, and environment on risk of colorectal cancer. *Gastroenterology* 158:322-340. PMID: 31586566
11. Kitai T, Kir sop J, Tang W (2016) Exploring the microbiome in heart failure. *Current Heart Failure Reports* 13:103-109. PMID: 26886380
12. Fandriks L (2017) Roles of the gut in the metabolic syndrome: an overview. *Journal of Internal Medicine* 281:319-336. PMID: 27991713
13. Barna I, Nyul D, Szentes T, Schwab R (2018) [Review of the relation between gut microbiome, metabolic disease and hypertension]. Article in Hungarian. *Orvosi Hetilap* 159:346-351. PMID: 29480046
14. Neuman H, Koren O (2017) The pregnancy microbiome. *Nestle Nutrition Institute Workshop Series* 88:1-9. PMID: 28346919
15. Zhang J, Ma S, Wu S et al. (2019) Effects of probiotic supplement in pregnant women with gestational diabetes mellitus: a systematic review and meta-analysis of randomized controlled trials. *Journal of Diabetes Research* 2019:5364730. PMID: 31583250

16. Ipci K, Altintoprak N, Muluk N et al. (2017) The possible mechanisms of the human microbiome in allergic diseases. *European Archives of Oto-Rhino-Laryngology* 274:617-626. PMID: 27115907
17. Santos S, Konstantyner T, Cocco R (2020) Effects of probiotics in the treatment of food hypersensitivity in children: a systematic review. *Allergologia et Immunopathologia* 48:95-104. PMID: 31477401
18. Kohling H, Plummer S, Marchesi J et al. (2017) The microbiota and autoimmunity: their role in thyroid autoimmune diseases. *Clinical Immunity* 183:63-74. PMID: 28689782
19. Yurtdas G, Akdevelioglu Y (2019) A new approach to polycystic ovary syndrome: the gut microbiota. *Journal of the American College of Nutrition* 12:1-12. PMID: 31513473
20. Gareau M (2016) Cognitive function and the microbiome. *International Review of Neurobiology* 131:227-246. PMID: 27793221
21. Winek K, Dirnagl U, Meisel A (2016) The gut microbiome as therapeutic target in central nervous system diseases: implications for stroke. *Neurotherapeutics* 13:762-774. PMID: 27714645\
22. Zheng P, Zeng B, Zhou C et al. (2016) Gut microbiome remodeling induces depressive-like behaviors through a pathway mediated by the host's metabolism. *Molecular Psychiatry* 21:786-796. PMID: 27067014
23. Quigley E (2017) Microbiota-brain-gut axis and neurodegenerative diseases. *Current Neurology and Neuroscience Reports* 17:94. PMID: 29039142
24. Naghibi M, Day R, Stone S, Harper A (2019) Probiotics for the prophylaxis of migraine: a systematic review of randomized placebo controlled trials. *Journal of Clinical Medicine* 8:1441. PMID: 31514352
25. Simkin D (2019) Microbiome and mental health, specifically as it relates to adolescents. *Current Psychiatry Reports* 21:93. PMID: 31478105
26. Srikantha P, Mohajeri M (2019) The possible role of the microbiota-gut-brain-axis in autism spectrum disorder. *International Journal of Molecular Sciences* 20:2115. PMID: 31035684
27. Ding F, Karkhaneh M, Zorzela L et al. (2019) Probiotics for paediatric functional abdominal pain disorders: a rapid review. *Paediatrics & Child Health* 24:383-394. PMID: 31528110
28. Glassner K, Abraham B, Quigley E (2020) The microbiome and inflammatory bowel disease. *The Journal of Allergy and Clinical Immunology* 145:16-27. PMID: 31910984
29. Tuomisto S, Pessi T, Collin P et al. (2014) Changes in gut bacterial populations and their translocation into liver and ascites in alcoholic liver cirrhosis. *BMC Gastroenterology* 14:40. PMID: 24564202

30. Victor D, Quigley E (2016) The microbiome and the liver: the basics. *Seminars in Liver Disease* 36:299-305. PMID: 27997968
31. Joyce S, Gahan C (2017) Disease-associated changes in bile acid profiles and links to altered gut microbiota. *Digestive Diseases* 35:169-177. PMID: 28249284
32. Adolph T, Grander C, Moschen A, Tilg H (2018) Liver-microbiome axis in health and disease. *Trends in Immunology* 39:712-713. PMID: 29843959
33. Gadelha C, Bezerra A (2019) Effects of probiotics on the lipid profile: systematic review. *Jornal Vascular Brasiliense* 18:e20180124. PMID: 31447899
34. Tiderencel K, Hutcheon D, Ziegler J (2020) Probiotics for the treatment of type 2 diabetes: a review of randomized controlled trials. *Diabetes/Metabolism Research and Reviews* 36:e3213. PMID: 31465625
35. Costello M, Robinson P, Benham H, Brown M (2015) The intestinal microbiome in human disease and how it relates to arthritis and spondyloarthritis. *Best Practice and Research. Clinical Rheumatology* 29:202-212. PMID: 26362739
36. Verwoerd A, Haar N, de Roock S et al. (2016) The human microbiome and juvenile idiopathic arthritis. *Pediatric Rheumatology Online Journal* 14:55. PMID: 27650128
37. Rosenbaum J, Asquith M (2016) The microbiome: a revolution in treatment for rheumatic diseases? *Current Rheumatology Reports* 18:62. PMID: 27641915
38. Chotirmall S, Gellatly S, Budden K et al. (2017) Microbiomes in respiratory health and disease: an Asia-Pacific perspective. *Respirology* 22:240-250. PMID: 28102970
39. Salisbury M, Han M, Dickson R, Molyneaux P (2017) The microbiome in interstitial lung disease: from pathogenesis to treatment target. *Current Opinion in Pulmonary Medicine* 23:404-410. PMID: 28650861
40. Wang L, Hao K, Yang T, Wang C (2017) Role of the lung microbiome in the pathogenesis of chronic obstructive pulmonary disease. *Chinese Medical Journal* 130:2107-2111. PMID: 28741603
41. Lu L, Liu J (2016) Human microbiota and ophthalmic disease. *Yale Journal of Biology and Medicine* 89:325-330. PMID: 27698616
42. Rowan S, Taylor A (2018) The role of microbiota in retinal disease. *Advances in Experimental Medicine and Biology* 1074:429-435. PMID: 29721973
43. Cantore S, Ballini A, De Vito D et al. (2018) Clinical results of improvement in periodontal condition by administration of oral probiotics. *Journal of Biological Regulators and Homeostatic Agents* 32:1329-1334. PMID: 30334434

44. Inchincolo F, Dipalma G, Cirulli N et al. (2018) Microbiological results of improvement in periodontal condition by administration of oral probiotics. *Journal of Biological Regulators and Homeostatic Agents* 32:1323-1328. PMID: 30334433
45. Soares L, de Carvalho E, Tinoco E (2019) Clinical effect of *Lactobacillus* on the treatment of severe periodontitis and halitosis: a double-blinded, placebo-controlled, randomized clinical trial. *American Journal of Dentistry* 32:9-13. PMID: 30834725
46. John G, Mullin G (2016) The gut microbiome and obesity. *Current Oncology Reports* 18:45. PMID: 27255389
47. Crovesy L, Ostrowski M, Ferreira D et al. (2017) Effect of *Lactobacillus* on body weight and body fat in overweight subjects: a systematic review of randomized controlled clinical trials. *International Journal of Obesity* 41:1607-1614. PMID: 28792488
48. Menni C, Jackson M, Pallister T et al. (2017) Gut microbiome diversity and high-fibre intake are related to lower long-term weight gain. *International Journal of Obesity* 41:1099-1105. PMID: 28286339
49. Seganfredo F, Blume C, Moehlecke M et al. (2017) Weight-loss interventions and gut microbiota changes in overweight and obese patients: a systematic review. *Obesity Reviews* 18:832-851. PMID: 28524627
50. Khan S, Ansari F (2007) Probiotics—the friendly bacteria with market potential in global market. *Pakistan Journal of Pharmaceutical Sciences* 20:76-82. PMID: 17337434
51. Zhang Y, Leveille S, Edward J (2020) Wisdom teeth, periodontal disease, and C-reactive protein in US adults. *Public Health* 187:97-102. PMID: 32942171
52. Whittaker P, Tufaro P, Rader J (2001) Iron and folate in fortified cereals. *Journal of the American College of Nutrition* 20:247-254. PMID: 11444421
53. Reifen R, Matas Z, Zeidel L et al. (2000) Iron supplementation may aggravate inflammatory status of colitis in a rat model. *Digestive Diseases and Sciences* 45:394-397. PMID: 10711457
54. Carrier J, Aghdassi E, Platt I et al. (2001) Effect of iron supplementation on oxidative stress and colonic inflammation in rats with induced colitis. *Alimentary Pharmacology & Therapeutics* 15:1989-1999. PMID: 11736731
55. Yu S, Feng Y, Shen Z, Li M (2011) Diet supplementation with iron augments brain oxidative stress status in a rat model of psychological stress. *Nutrition* 27:1048-1052. PMID: 21454054
56. Gao W, Li X, Gao Z, Li H (2014) Iron increases diabetes-induced kidney injury and oxidative stress in rats. *Biological Trace Element Research* 160:368-375. PMID: 24996958

57. Volani C, Paglia G, Smarason S et al. (2018) Metabolic signature of dietary iron overload in a mouse model. *Cells* 7:264. PMID: 30544931
58. Korkmaz V, Ozkaya E, Seven B et al. (2014) Comparison of oxidative stress in pregnancies with and without first trimester iron supplement: a randomized double-blind controlled trial. *Journal of Maternal-Fetal & Neonatal Medicine* 27:1535-1538. PMID: 24199687
59. Lymeraki E, Tsikopoulos A, Makedou K et al. (2015) Impact of iron and folic acid supplementation on oxidative stress during pregnancy. *Journal of Obstetrics and Gynaecology* 35:803-806. PMID: 25692315
60. Scholl T (2005) Iron status during pregnancy: setting the stage for mother and infant. *The American Journal of Clinical Nutrition* 81:1218S-1222S. PMID: 15883455
61. Tiwari A, Mahdi A, Chandyan S et al. (2011) Oral iron supplementation leads to oxidative imbalance in anemic women: a prospective study. *Clinical Nutrition* 30:188-193. PMID: 20888091
62. Weinberg E (2000) Iron-enriched rice: the case for labeling. *Journal of Medicinal Food* 3:189-191. PMID: 19236176
63. Weinberg E (2009) Is addition of iron to processed foods safe for iron replete consumers? *Medical Hypotheses* 73:948-949. PMID: 19628337
64. Levy T (2019) *Magnesium: Reversing Disease*, Henderson, NV: MedFox Publishing
65. Lucas C, Barnich N, Nguyen H (2017) Microbiota, inflammation and colorectal cancer. *International Journal of Molecular Sciences* 18:1310. PMID: 28632155
66. Piotrowski I, Kulcenty K, Suchorska W (2020) Interplay between inflammation and cancer. *Reports of Practical Oncology and Radiotherapy* 25:422-427. PMID: 32372882

Chapter 10

1. Levy T (2011) *Primal Panacea*, Henderson, NV: MedFox Publishing
2. Marik P, Khangoora V, Rivera R et al. (2017) Hydrocortisone, vitamin C, and thiamine for the treatment of severe sepsis and septic shock. A retrospective before-after study. *Chest* 151:1229-1238. PMID: 27940189
3. Vergauwen B, Herbert M, Van Beeumen J (2006) Hydrogen peroxide scavenging is not a virulence determinant in the pathogenesis of *Haemophilus influenzae* type b strain Eagan. *BMC Microbiology* 6:3. PMID: 16430767
4. Santos C, Bannitz-Fernandes R, Lima A et al. (2018) Monitoring H₂O₂ inside *Aspergillus fumigatus* with an integrated microelectrode: the role of peroxiredoxin protein Prx1. *Analytical Chemistry* 90:2587-2593. PMID: 29345916

5. Nishimura M (2016) High-flow nasal cannula oxygen therapy in adults: physiological benefits, indication, clinical benefits, and adverse effects. *Respiratory Care* 61:529-541. PMID: 27016353
6. Oliver T, Murphy D (1920) Influenza pneumonia: the intravenous injection of hydrogen peroxide. *The Lancet* 195:432-433.
7. Levine M, Padayatty S, Espey M (2011) Vitamin C: a concentration-function approach yields pharmacology and therapeutic discoveries. *Advances in Nutrition* 2:78-88. PMID: 22332036
8. Halliwell B, Clement M, Ramalingam J, Long L (2000) Hydrogen peroxide. Ubiquitous in cell culture and *in vivo*? *IUBMD Life* 50:251-257. PMID: 11327318
9. Yoon J, An S, Kyeong I et al., (2011) Oxidative modification of ferritin induced by hydrogen peroxide. *BMB Reports* 44:165-169. PMID: 21429293
10. Antunes F, Cadenas E (2000) Estimation of H₂O₂ gradients across biomembranes. *FEBS Letters* 475:121-126. PMID: 10858501
11. Chen Q, Espey M, Krishna M et al. (2005) Pharmacologic ascorbic acid concentrations selectively kill cancer cells: action as a pro-drug to deliver hydrogen peroxide to tissues. *Proceedings of the National Academy of Sciences of the United States of America* 102:13604-13609. PMID: 16157892
12. Chen Q, Espey M, Sun A et al. (2007) Ascorbate in pharmacologic concentrations selectively generates ascorbate radical and hydrogen peroxide in extracellular fluid *in vivo*. *Proceedings of the National Academy of Sciences of the United States of America* 104:8749-8754. PMID: 17502596
13. Root R (1975) H₂O₂ release from human granulocytes during phagocytosis: I. Documentation, quantitation, and some regulating factors. *The Journal of Clinical Investigation* 55:945-955. PMID: 1123431
14. Root R, Metcalf J (1977) H₂O₂ release from human granulocytes during phagocytosis. Relationship to superoxide anion formation and cellular catabolism of H₂O₂: studies with normal and cytochalasin B-treated cells. *The Journal of Clinical Investigation* 60:1266-1279. PMID: 199619
15. Evans R, Currie L, Campbell A (1982) The distribution of ascorbic acid between various cellular components of blood, in normal individuals, and its relation to the plasma concentration. *The British Journal of Nutrition* 47:473-482. PMID: 7082619
16. Carr A, Maggini S (2017) Vitamin C and immune function. *Nutrients* 9:1211. PMID: 29099763
17. Ang A, Pullar J, Currie M, Vissers M (2018) Vitamin C and immune cell function in inflammation and cancer. *Biochemical Society Transactions* 46:1147-1159. PMID: 30301842

18. Pei Z, Wu K, Li Z et al. (2019) Pharmacologic ascorbate as a pro-drug for hydrogen peroxide release to kill mycobacteria. *Biomedicine & Pharmacotherapy* 109:2119-2127. PMID: 30551469
19. Rowen R, Robins H, Carew K et al. (2016) Rapid resolution of hemorrhagic fever (Ebola) in Sierra Leone with ozone therapy. *African Journal of Infectious Diseases* 10:49-54.
20. Gonzalez M, Miranda-Massari J, Berdiel M et al. (2014) High dose intravenous vitamin C and Chikungunya fever: a case report. *Journal of Orthomolecular Medicine* 29:154-156.
21. Marcial-Vega V, Gonzalez-Terron G, Levy T (2015) Intravenous ascorbic acid and hydrogen peroxide in the management of patients with Chikungunya. *Bulletin of the Medical Association of Puerto Rico* 107:20-24. PMID: 26035980
22. Gonzalez M, Berdiel M, Miranda-Massari J et al. (2016) High dose intravenous vitamin C treatment for Zika fever. *Journal of Orthomolecular Medicine* Volume 31.
23. Gonzalez M, Berdiel M, Duconge J et al. (2018) High dose intravenous vitamin C and influenza: a case report. *Journal of Orthomolecular Medicine* Volume 33
24. Kashiouris M, L'Heureux M, Cable C et al. (2020) The emerging role of vitamin C as a treatment for sepsis. *Nutrients* 12:292. PMID: 31978969
25. Fowler A, Kim C, Lepler L et al. (2017) Intravenous vitamin C as adjunctive therapy for enterovirus/rhinovirus induced acute respiratory distress syndrome. *World Journal of Critical Care Medicine* 6:85-90. PMID: 28224112
26. Matthay M, Aldrich J, Gotts J (2020) Treatment for severe acute respiratory distress syndrome from COVID-19. *The Lancet. Respiratory Medicine* 8:433-434. PMID: 32203709
27. Zhao B, Ling Y, Li J et al. (2020) Beneficial aspects of high dose intravenous vitamin C on patients with COVID-19 pneumonia in severe condition: a retrospective case series study. *Annals of Palliative Medicine* Nov 17. Online ahead of print. PMID: 33222462
28. <http://orthomolecular.org/resources/omns/index.shtml>
29. Rose R, Pinkston P, Skornik W (1989) Altered susceptibility to viral respiratory infection during short-term exposure to nitrogen dioxide. *Research Report (Health Effects Institute)* 24:1-24. PMID: 2557864
30. Carter M, Chapman M, Gabler F, Brandi M (2015) Effect of sulfur dioxide fumigation on survival of foodborne pathogens on table grapes under standard storage temperature. *Food Microbiology* 49:189-196. PMID: 25846930
31. Vo H, Imai T, Ho T et al. (2015) Potential application of high pressure carbon dioxide in treated wastewater and water disinfection: recent overview and further trends. *Journal of Environmental Sciences (China)* 36:38-47. PMID: 26456604

32. Akhlaghi M, Dorost A, Karimyan K et al. (2018) Data for comparison of chloride dioxide and chlorine disinfection power in a real dairy wastewater effluent. *Data in Brief* 18:886-890. PMID: 29900255
33. Mayer D, Mithofer A, Glawischnig et al. (2018) Short-term exposure to nitrogen dioxide provides basal pathogen resistance. *Plant Physiology* 178:468-487. PMID: 30076223
34. Besinis A, De Peralta T, Handy R (2014) The antibacterial effects of silver, titanium dioxide and silica dioxide nanoparticles compared to the dental disinfectant chlorhexidine on *Streptococcus mutans* using a suite of bioassays. *Nanotoxicology* 8:1-16. PMID: 23092443
35. Shirai R, Miura T, Yoshida A et al. (2016) Antimicrobial effect of titanium dioxide after ultraviolet irradiation against periodontal pathogen. *Dental Materials Journal* 35:511-516. PMID: 27252009
36. Yamaneli L, Kaldirim U, Oztas Y et al. (2011) Ozone therapy and hyperbaric oxygen treatment in lung injury in septic rats. *International Journal of Medical Sciences* 8:48-55. PMID: 21234269
37. Pryor W, Church D (1991) Aldehydes, hydrogen peroxide, and organic radicals as mediators of ozone toxicity. *Free Radical Biology & Medicine* 11:41-46. PMID: 1937128
38. Elvis A, Ekta J (2011) Ozone therapy: a clinical review. *Journal of Natural Science, Biology and Medicine* 2:66-70. PMID: 22470237
39. Kontorshchikova K, Belova A, Dudenkova V et al. (2017) The level of hydrogen peroxide in HeLa cells in an ozonated medium. *Bulletin of Experimental Biology and Medicine* 163:570-573. PMID: 28853083
40. Cattel F, Giordano S, Bertiond C et al. (2021) Ozone therapy in COVID-19: a narrative review. *Virus Research* 291:198207. PMID: 33115670
41. Franzini M, Valdenassi L, Ricevuti G et al. (2020) Oxygen-ozone (O₂-O₃) immunoceutical therapy for patients with COVID-19. Preliminary evidence reported. *International Immunopharmacology* 88:106879. PMID: 32795898
42. Zheng Z, Dong M, Hu K (2020) A preliminary evaluation on the efficacy of ozone therapy in the treatment of COVID-19. *Journal of Medical Virology* 92:2348-2350. PMID: 32437014
43. Tascini C, Sermann G, Pagotto A et al. (2020) Blood ozonization in patients with mild to moderate COVID-19 pneumonia: a single centre experience. *Internal and Emergency Medicine* Nov 1. Online ahead of print. PMID: 33131033
44. Reed N (2010) The history of ultraviolet germicidal irradiation for air disinfection. *Public Health Reports* 125:15-27. PMID: 20402193
45. Welch D, Buonanno M, Grilj V et al. (2018) Far-UVC light: a new tool to control the spread of airborne-mediated microbial diseases. *Scientific Reports* 29426899

46. Yang J, Wu U, Tai H, Sheng W (2019) Effectiveness of an ultraviolet-C disinfection system for reduction of healthcare-associated pathogens. *Journal of Microbiology, Immunology, and Infection* 52:487-493. PMID: 28951015
47. Heilingloh C, Aufderhorst U, Schipper L et al. (2020) Susceptibility of SARS-CoV-2 to UV irradiation. *American Journal of Infection Control* 48:1273-1275. PMID: 32763344
48. Hamblin M, Abrahamse H (2020) Oxygen-independent antimicrobial photoinactivation: type III photochemical mechanism? *Antibiotics (Basel)* 9:53. PMID: 32023978
49. Rowen R (1996) Ultraviolet blood irradiation therapy (photo-oxidation), the cure that time forgot. *Int J Biosocial Med Res* 14:115-132.
50. Kuenstner J, Mukherjee S, Weg S et al. (2015) The treatment of infectious disease with a medical device: results of a clinical trial of ultraviolet blood irradiation (UVBI) in patients with hepatitis C infection. *International Journal of Infectious Diseases* 37:58-63. PMID: 26092299
51. Zhadnov V, Mishanov R, Kuznetsov A et al. (1995) [Effectiveness of chemotherapy in combination with electrophoresis and ultraviolet irradiation of blood in newly diagnosed patients with destructive pulmonary tuberculosis.] Article in Russian. *Problemy Tuberkuleza* 3:20-22. PMID: 7617626
52. Shurygin A (2009) [The efficiency of ultraviolet autologous blood irradiation used in the complex therapy of infiltrative pulmonary tuberculosis in children and adolescents.] Article in Russian. *Tuberkulez i Bolezni Legkikh* 9:20-23. PMID: 19882857
53. Hamblin M (2017) Ultraviolet irradiation of blood: "The cure that time forgot"? *Advances in Experimental Medicine and Biology* 996:295-309. PMID: 29124710
54. Budowsky E, Bresler S, Friedman E, Zheleznova N (1981) Principles of selective inactivation of viral genome. I. UV-induced inactivation of influenza virus. *Archives of Virology* 68:239-247. PMID: 7271457
55. Conner-Kerr T, Sullivan P, Gaillard J et al. (1998) The effects of ultraviolet radiation on antibiotic-resistant bacteria *in vitro*. *Ostomy/Wound Management* 44:50-56. PMID: 9866596
56. Boretti A, Banik B, Castelletto S (2020) Use of ultraviolet blood irradiation against viral infections. *Clinical Reviews in Allergy & Immunology* Oct 7. Online ahead of print. PMID: 33026601
57. Memar M, Yekani M, Alizadeh N, Baghi H (2019) Hyperbaric oxygen therapy: antimicrobial mechanisms and clinical application for infections. *Biomedicine & Pharmacotherapy* 109:440-447. PMID: 30399579
58. Hink J, Jansen E (2001) Are superoxide and/or hydrogen peroxide responsible for some of the beneficial effects of hyperbaric oxygen therapy? *Medical Hypotheses* 57:764-769. PMID: 11918444

59. Moon R, Weaver L (2020) Hyperbaric oxygen as a treatment for COVID-19 infection? *Undersea & Hyperbaric Medicine* 47:177-179. PMID: 32574432
60. Grant W, Lahore H, McDonnell S et al. (2020) Evidence that vitamin D supplementation could reduce risk of influenza and COVID-19 infections and deaths. *Nutrients* 12:988. PMID: 32252338
61. Name J, Souza A, Vasconcelos A et al. (2020) Zinc, vitamin D and vitamin C: perspectives for COVID-19 with a focus on physical tissue barrier integrity. *Frontiers in Nutrition* 7:606398. PMID: 33365326
62. De Smet D, De Smet K, Herroelen P et al. (2020) Serum 25(OH)D level on hospital admission associated with COVID-19 stage and mortality. *American Journal of Clinical Pathology* Nov 25. Online ahead of print. PMID: 33236114
63. Vassiliou A, Jahaj E, Pratikaki M et al. (2020) Low 25-hydroxyvitamin D levels on admission to the intensive care unit may predispose COVID-19 pneumonia patients to a higher 28-day mortality risk: a pilot study on a Greek ICU cohort. *Nutrients* 12:3773. PMID: 33316914
64. Cangiano B, Fatti L, Danesi L et al. (2020) Mortality in an Italian nursing home during COVID-19 pandemic: correlation with gender, age, ADL, vitamin D supplementation, and limitations of the diagnostic tests. *Aging* Dec 22. Online ahead of print. PMID: 33353888
65. Mariana J, Gimenez V, Bergam I et al. (2020) Association between vitamin D deficiency and COVID-19 incidence, complications, and mortality in 46 countries: an ecological study. *Health Security* Dec 14. Online ahead of print. PMID: 33325788
66. Dancer R, Parekh D, Lax S et al. (2015) Vitamin D deficiency contributes directly to the acute respiratory distress syndrome (ARDS). *Thorax* 70:617-624. PMID: 25903964
67. Chandran M, Maung A, Mithal A, Parameswaran R (2020) Vitamin D in COVID-19: dousing the fire or averting the storm? – a perspective from the Asia-Pacific. *Osteoporosis and Sarcopenia* 6:97-105. PMID: 32838048
68. Mercola J, Grant W, Wagner C (2020) Evidence regarding vitamin D and risk of COVID-19 and its severity. *Nutrients* 12:3361. PMID: 33142828
69. Rhodes J, Subramanian S, Laird E et al. (2021) Perspective: vitamin D deficiency and COVID-19 severity—plausibly linked by latitude, ethnicity, impacts on cytokines, ACE2 and thrombosis. *Journal of Internal Medicine* 289:97-115. PMID: 32613681
70. Neveu A (1959) [La Polio Guerie! Traitement Cytophyllactique de la Poliomyelite par le Chlorure de Magnesium]. Book in French. (“Polio healed. Cytophyllactic treatment of polio with magnesium chloride”). Paris, France: La Vie Claire

71. Rodale J (with Taub H) (1968) *Magnesium, the Nutrient that could Change Your Life*. Pyramid Publications, Inc.: New York, NY
72. Neveu A (1958) [Le Chlorure de Magnesium: Traitement Cytophylactique des Maladies Infectieuses]. Book in French. ("Cytophylactic treatment of infectious diseases by magnesium chloride"). Paris, France: Librairie Le Francois
73. Neveu A (1961) *Le Chlorure de Magnesium Dans L'Elevage: Traitement Cytophylactique des Maladies Infectieuses*. Paris, France: Librairie Le Francois
74. Dekopol B (2018) [Le Chlorure de Magnesium Histoireet Manuel Pratique: Traitement des maladies infectieuses chez l'homme et les animaux]. Book in French. ("Magnesium chloride history and practical manual: treatment of infectious diseases in humans and animals"). Le Jardin de l'Ataraxie. Amazon Kindle
75. Rapp F, Butel J, Wallis C (1965) Protection of measles virus by sulfate ions against thermal inactivation. *Journal of Bacteriology* 90:132-135. PMID: 16562007
76. Boriskin Y, Steinberg L, Dorofeeva L et al. (1988) Salt-induced enhancement of measles virus yields in cultured cells. *Archives of Virology* 101:131-136. PMID: 3415478
77. Alansari K, Sayyed R, Davidson B et al. (2017) IV magnesium sulfate for bronchiolitis: a randomized trial. *Chest* 152:113-119. PMID: 28286262
78. Pruukkonen H, Tapiainen T, Kallio M et al. (2018) Intravenous magnesium sulfate for acute wheezing in young children: a randomized double-blind trial. *The European Respiratory Journal* 51:1701579. PMID: 29437941
79. Wei Z, Burwinkel M, Palissa C et al. (2012) Antiviral activity of zinc salts against transmissible gastroenteritis virus *in vitro*. *Veterinary Microbiology* 160:468-472. PMID: 22818659
80. Green R (2016) Asthma in adults (acute): magnesium sulfate treatment. *BMJ Clinical Evidence* 2016:1513. PMID: 26761432
81. Dominguez L, Barbagallo M, Di Lorenzo G et al. (1998) Bronchial reactivity and intracellular magnesium: a possible mechanism for the bronchodilating effects of magnesium in asthma. *Clinical Science* 95:137-142. PMID: 9680494
82. Colquhoun I, Berg G, el-Fiky M et al. (1993) Arrhythmia prophylaxis after coronary artery surgery. A randomized controlled trial of intravenous magnesium chloride. *European Journal of Cardiothoracic Surgery* 7:520-523. PMID: 8267992
83. Benzer W, Schmid P, Maehr G, Drexel H (1996) Effects of intravenous magnesium chloride reverse left ventricular end-diastolic pressure in coronary artery disease. *The American Journal of Cardiology* 77:638-640. PMID: 8610617

84. Naik P, Malati T, Ratnakar K et al. (1999) Cardioprotective effect of magnesium chloride in experimental acute myocardial infarction. *Indian Journal of Experimental Biology* 37:131-137. PMID: 10641131
85. Muradov J, Hagg T (2013) Intravenous infusion of magnesium chloride improves epicenter blood flow during the acute stage of contusive spinal cord injury in rats. *Journal of Neurotrauma* 30:840-852. PMID: 23302047
86. Levy T (2019) *Magnesium: Reversing Disease*, Henderson, NV: MedFox Publishing
87. Choi K (2012) Viral polymerases. *Advances in Experimental Medicine and Biology* 726:267-304. PMID: 22297518
88. Yin W, Mao C, Luan X et al. (2020) Structural basis for inhibition of the RNA-dependent RNA polymerase from SARS-CoV-2 by remdesivir. *Science* 368:1499-1504. PMID: 32358203
89. te Velthuis A, van den Worm S, Sims A et al. (2010) Zn(2+) inhibits coronavirus and arterivirus RNA polymerase activity *in vitro* and zinc ionophores block the replication of these viruses in cell culture. *PLoS Pathogens* 6:e1001176. PMID: 21079686
90. Kaushik N, Subramani C, Anang S et al. (2017) Zinc salts block hepatitis E virus replication by inhibiting the activity of viral RNA-dependent RNA polymerase. *Journal of Virology* 91:e00754-17. PMID: 28814517
91. Kumar A, Kubota Y, Chernov M, Kasuya H (2020) Potential role of zinc supplementation in prophylaxis and treatment of COVID-19. *Medical Hypotheses* 144:109848. PMID: 32512490
92. Wessels I, Rolles B, Rink L (2020) The potential impact of zinc supplementation on COVID-19 pathogenesis. *Frontiers in Immunology* 11:1712. PMID: 32754164
93. Read S, Obeid S, Ahlenstiel C, Ahlenstiel G (2019) The role of zinc in antiviral immunity. *Advances in Nutrition* 10:696-710. PMID: 31305906
94. Dabbagh-Bazarbachi H, Clergeaud G, Quesada I et al. (2014) Zinc ionophore activity of quercetin and epigallocatechin-gallate: from Hepa 1-6 cells to a liposome model. *Journal of Agricultural and Food Chemistry* 62:8085-8093. PMID: 25050823
95. Derwand R, Scholz M (2020) Does zinc supplementation enhance the clinical efficacy of chloroquine/hydroxychloroquine to win today's battle against COVID-19? *Medical Hypotheses* 142:109815. PMID: 32408070
96. Qui X, Kroeker A, He S et al. (2016) Prophylactic efficacy of quercetin 3-β-O-d-glucoside against Ebola virus infection. *Antimicrobial Agents and Chemotherapy* 60:5182-5188. PMID: 27297486

97. Sharun K, Tiwari R, Yatoo M et al. (2020) Antibody-based immunotherapy and use of convalescent plasma to counter COVID-19: advances and prospects. *Expert Opinion on Biological Therapy* 20:1033-1046. PMID: 32744917
98. White N, Pukrittayakamee S, Hien T et al. (2014) Malaria. *Lancet* 383:723-735. PMID: 23953767
99. Klinger G, Morad Y, Westall C et al. (2001) Ocular toxicity and antenatal exposure to chloroquine or hydroxychloroquine for rheumatic diseases. *Lancet* 358:813-814. PMID: 11564493
100. Savarino A, Boelaert J, Cassone A et al. (2003) Effects of chloroquine on viral infections: an old drug against today's diseases? *The Lancet. Infectious Diseases* 3:722-727. PMID: 14592603
101. Lofgren S, Nicol M, Bangdiwala A et al. (2020) Safety of hydroxychloroquine among outpatient clinical trial participants for COVID-19. *Open Forum Infectious Diseases* 7:ofaa500. PMID: 33204764
102. Gies V, Bekaddour N, Dieudonne Y et al. (2020) Beyond anti-viral effects of chloroquine/hydroxychloroquine. *Frontiers in Immunology* 11:1409. PMID: 32714335
103. Uzunova K, Filipova E, Pavlova V, Vekov T (2020) Insights into antiviral mechanisms of remdesivir, lopinavir/ritonavir and chloroquine/hydroxychloroquine affecting the new SARS-CoV-2. PMID: 131:110668. PMID: 32861965
104. Xu Y, Xiao G, Liu L, Lang M (2019) Zinc transporters in Alzheimer's disease. *Molecular Brain* 12:106. PMID: 31818314
105. Xue J, Moyer A, Peng B et al. (2014) Chloroquine is a zinc ionophore. *PLoS One* 9:e109180. PMID: 25271834
106. Carlucci P, Ahuja T, Petrilli C et al. (2020) Zinc sulfate in combination with a zinc ionophore may improve outcomes in hospitalized COVID-19 patients. *Journal of Medical Microbiology* 69:1228-1234. PMID: 32930657
107. Rolain J, Colson P, Raoult D (2007) Recycling of chloroquine and its hydroxyl analogue to face bacterial, fungal and viral infections in the 21st century. *International Journal of Antimicrobial Agents* 30:297-308. PMID: 17629679
108. Guan W, Lan W, Zhang J et al. (2020) COVID-19: antiviral agents, antibody development and traditional Chinese medicine. *Virologica Sinica* Sep 30. Online ahead of print. PMID: 32997322
109. Gautret P, Lagier J, Parola P et al. (2020) Hydroxychloroquine and azithromycin as a treatment of COVID-19: results of an open-label non-randomized trial. *International Journal of Antimicrobial Agents* 56:105949. PMID: 32205204
110. Lagier J, Million M, Gautret P et al. (2020) Outcomes of 3,737 COVID-19 patients treated with hydroxychloroquine/azithromycin and other regimens in Marseille, France: a retrospective analysis. *Travel Medicine and Infectious Disease* 36:101791. PMID: 32593867

111. Agarwal M, Ranjan P, Baitha U, Mittal A (2020) Hydroxychloroquine as a chemoprophylactic agent for COVID-19: a clinico-pharmacological review. *Frontiers in Pharmacology* 11:593099. PMID: 33390974
112. Cortegiani A, Ingoglia G, Ippolito M et al. (2020) A systematic review on the efficacy and safety of chloroquine for the treatment of COVID-19. *Journal of Critical Care* 57:279-283. PMID: 32173110
113. Devaux C, Rolain J, Colson P, Raoult D (2020) New insights on the antiviral effects of chloroquine against coronavirus: what to expect for COVID-19? *International Journal of Antimicrobial Agents* 55:105938. PMID: 32171740
114. Meo S, Klonoff D, Akram J (2020) Efficacy of chloroquine and hydroxychloroquine in the treatment of COVID-19. *European Review for Medical and Pharmacological Sciences* 24:4539-4547. PMID: 32373993
115. Boulware D, Pullen M, Bangdiwala A et al. (2020) A randomized trial of hydroxychloroquine as postexposure prophylaxis for COVID-19. *The New England Journal of Medicine* 383:517-525. PMID: 32492293
116. Shah S, Das S, Jain A et al. (2020) A systematic review of the prophylactic role of chloroquine and hydroxychloroquine in coronavirus disease-19 (COVID-19). *International Journal of Rheumatic Diseases* 23:613-619. PMID: 32281213
117. Huang M, Tang T, Pang P et al. (2020) Treating COVID-19 with chloroquine. *Journal of Molecular Cell Biology* 12:322-325. PMID: 32236562
118. Rizzo E (2020) Ivermectin, antiviral properties and COVID-19: a possible new mechanism of action. *Naunyn-Schmiedeberg's Archives of Pharmacology* 393:1153-1156. PMID: 32462282
119. Caly L, Druce J, Catton M et al. (2020) The FDA-approved drug ivermectin inhibits the replication of SARS-CoV-2 *in vitro*. *Antiviral Research* 178:104787. PMID: 32251768
120. Chaccour C, Hammann F, Ramon-Garcia S, Rabinovich N (2020) Ivermectin and COVID-19: keeping rigor in times of urgency. *The American Journal of Tropical Medicine and Hygiene* 102:1156-1157. PMID: 32314704
121. Heidary F, Gharebaghi R (2020) Ivermectin: a systematic review from antiviral effects to COVID-19 complementary regimen. *The Journal of Antibiotics* 73:593-602. PMID: 32533071
122. Sharun K, Dhama K, Patel S et al. (2020) Ivermectin, a new candidate therapeutic against SARS-CoV-2/COVID-19. *Annals of Clinical Microbiology and Antimicrobials* 19:23. PMID: 32473642
123. Hellwig M, Maia A (2020) A COVID-19 prophylaxis? Lower incidence associated with prophylactic administration of ivermectin. *International Journal of Antimicrobial Agents* Nov 28. Online ahead of print. PMID: 33259913

124. Padhy B, Mohanty R, Das S, Meher B (2020) Therapeutic potential of ivermectin as add on treatment in COVID-19: a systematic review and meta-analysis. *Journal of Pharmacy & Pharmaceutical Sciences* 23:462-469. PMID: 33227231
125. Choudhary R, Sharma A (2020) Potential use of hydroxychloroquine, ivermectin and azithromycin drugs in fighting COVID-19: trends, scope and relevance. *New Microbes and New Infections* 35:100684. PMID: 32322397
126. Chaccour C, Abizanda G, Irigoyen-Barrio A et al. (2020) Nebulized ivermectin for COVID-19 and other respiratory diseases, a proof of concept, dose-ranging study in rats. *Scientific Reports* 10:17073. PMID: 33051517
127. Casadevall A (1996) Antibody-based therapies for emerging infectious diseases. *Emerging Infectious Diseases* 2:200-208. PMID: 8903230
128. Casadevall A, Scharff M (1994) Serum therapy revisited: animal models of infection and development of passive antibody therapy. *Antimicrobial Agents and Chemotherapy* 38:1695-1702. PMID: 7985997
129. Casadevall A, Scharff M (1995) Return to the past: the case for antibody-based therapies in infectious diseases. *Clinical Infectious Diseases* 21:150-161. PMID: 7578725
130. Brown B, McCullough J (2020) Treatment for emerging viruses: convalescent plasma and COVID-19. *Transfusion and Apheresis Science* 59:102790. PMID: 32345485
131. Ye M, Fu D, Ren Y et al. (2020) Treatment with convalescent plasma for COVID-19 patients in Wuhan, China. *Journal of Medical Virology* 92:1890-1901. PMID: 32293713
132. Sparrow E, Friede M, Sheikh M, Torvaldsen S (2017) Therapeutic antibodies for infectious diseases. *Bulletin of the World Health Organization* 95:235-237. PMID: 28250538
133. Jahanshahlu L, Rezaei N (2020) Monoclonal antibody as a potential anti-COVID-19. *Biomedicine & Pharmacotherapy* 129:110337. PMID: 32534226
134. Weinreich D, Sivapalasingam S, Norton T et al. (2020) REGN-COV2, a neutralizing antibody cocktail, in outpatients with COVID-19. *The New England Journal of Medicine* Dec 17. Online ahead of print. PMID: 33332778
135. Baum A, Ajithdoss D, Copin R et al. (2020) REGN-COV2 antibodies prevent and treat SARS-CoV-2 infection in rhesus macaques and hamsters. *Science* 370:1110-1115. PMID: 33037066
136. Liu X, Cao W, Li T (2020) High-dose intravenous immunoglobulins in the treatment of severe acute viral pneumonia: the known mechanisms and clinical effects. *Frontiers in Immunology* 11:1660. PMID: 32760407

137. Xie Y, Cao S, Dong H et al. (2020) Effect of regular intravenous immunoglobulin therapy on prognosis of severe pneumonia in patients with COVID-19. *The Journal of Infection* 81:318-356. PMID: 32283154

Chapter 12

1. Wong H (1989) The problems of tonsils and adenoids. *The Journal of the Singapore Paediatric Society* 31:97-102. PMID: 2700589
2. Emerick K, Cunningham M (2006) Tubal tonsil hypertrophy: a cause of recurrent symptoms after adenoidectomy. *Archives of Otolaryngology—Head & Neck Surgery* 132:153-156. PMID: 16490872
3. Hellings P, Jorissen M, Ceuppens J (2000) The Waldeyer's ring. *Acta Oto-Rhino-Laryngologica Belgica* 54:237-241. PMID: 11082757
4. Bitar M, Dowli A, Mourad M (2015) The effect of tonsillectomy on the immune system: a systematic review and meta-analysis. *International Journal of Pediatric Otorhinolaryngology* 79:1184-1191. PMID: 26055199
5. Allen H, Jadeja S, Allawh R, Goyal K (2018) Psoriasis, chronic tonsillitis, and biofilms: tonsillar pathologic findings supporting a microbial hypothesis. *Ear, Nose, & Throat Journal* 97:79-82. PMID: 29554401
6. Isseks J (2005) *Cancer, A Second Opinion: The Classic Book on Integrative Cancer Treatment* Garden City Park, NY: Square One Publishers

Chapter 13

1. Halliwell B (2006) Reactive species and antioxidants. Redox biology is a fundamental theme of aerobic life. *Plant Physiology* 141:312-322. PMID: 16760481
2. Hajeyah A, Griffiths W, Want Y et al. (2020) The biosynthesis of enzymatically oxidized lipids. *Frontiers in Endocrinology* 11:591819. PMID: 33329396
3. Bhatt S, Puli L, Patil C (2020) Role of reactive oxygen species in the progression of Alzheimer's disease. *Drug Discovery Today* Dec 8. Online ahead of print. PMID: 33306995
4. Sies H (1997) Oxidative stress: oxidants and antioxidants. *Experimental Physiology* 82:291-295. PMID: 9129943
5. Checa J, Aran J (2020) Reactive oxygen species: drivers of physiological and pathological processes. *Journal of Inflammation Research* 13:1057-1073. PMID: 33293849
6. Flohé L (2020) Looking back at the early stages of redox biology. *Antioxidants* 9:E1254. PMID: 33317108

7. Levy T (2002) *Curing the Incurable: Vitamin C, Infectious Diseases, and Toxins*. Henderson, NV: MedFox Publishing
8. Susick Jr R, Zannoni V (1987) Effect of ascorbic acid on the consequences of acute alcohol consumption in humans. *Clinical Pharmacology and Therapeutics* 41:502-509. PMID: 3568535
9. Zannoni V, Brodfuehrer J, Smart R, Susick Jr R (1987) Ascorbic acid, alcohol, and environmental chemicals. *Annals of the New York Academy of Sciences* 498:364-388. PMID: 3304067
10. Kao H, Jai S, Young Y (1965) [A study of the therapeutic effect of large dosage of injection ascorbici acidi on the depression of the central nervous system as in acute poisoning due to barbiturates]. Article in Chinese. *Acta Pharmaceutica Sinica* 12:764-765. PMID: 5899011
11. Klenner F (1971) Observations on the dose and administration of ascorbic acid when employed beyond the range of a vitamin in human pathology. *Journal of Applied Nutrition* 23:61-88.
12. Klenner F (1974) Significance of high daily intake of ascorbic acid in preventive medicine. *Journal of the International Academy of Preventive Medicine* 1:45-69.
13. Dwenger A, Pape H, Bantel C et al. (1994) Ascorbic acid reduces the endotoxin-induced lung injury in awake sheep. *European Journal of Clinical Investigation* 24:229-235. PMID: 8050451
14. Cadena S, Rojas C, Barja G (1998) Endotoxin increases oxidative injury to proteins in guinea pig liver: protection by dietary vitamin C. *Pharmacology & Toxicology* 82:11-18. PMID: 9527640
15. De la Fuente M, Victor V (2001) Ascorbic acid and N-acetylcysteine improve *in vitro* the function of lymphocytes from mice with endotoxin-induced oxidative stress. *Free Radical Research* 35:73-84. PMID: 11697119
16. Peterson F, Knodell R (1984) Ascorbic acid protects against acetaminophen- and cocaine-induced hepatic damage in mice. *Drug-Nutrient Interactions* 3:33-41. PMID: 6510239
17. Ilkiw J, Ratcliffe R (1987) Paracetamol toxicity in a cat. *Australian Veterinary Journal* 64:245-247. PMID: 3689263
18. Sprince H, Parker C, Smith G, Gonzales L (1975) Protective action of ascorbic acid and sulfur compounds against acetaldehyde toxicity: implications in alcoholism and smoking. *Agents and Actions* 5:164-173. PMID: 1171591
19. Rebec G, Centore J, White L, Alloway K (1985) Ascorbic acid and the behavioral response to haloperidol: implications for the action of antipsychotic drugs. *Science* 227:438-440. PMID: 4038426
20. White L, Carpenter M, Block M et al. (1988) Ascorbate antagonizes the behavioral effects of amphetamine by a central mechanism. *Psychopharmacology* 94:284-287. PMID: 2832869
21. Beyer C (2001) Rapid recovery from Ecstasy intoxication. *South African Medical Journal* 91:708-709.

22. Rao N, Snyder R (1995) Oxidative modifications produced in HL-60 cells on exposure to benzene metabolites. *Journal of Applied Toxicology* 15:403-409. PMID: 8666725
23. Soliman M, Elwi A, el-Kateb H, Kamel S (1965) Vitamin C as prophylactic drug against experimental hepatotoxicity. *The Journal of the Egyptian Medical Association* 48:806-812. PMID: 5886659
24. Ademuyiwa O, Adesanya O, Ajuwon O (1994) Vitamin C in CCl_4 hepatotoxicity—a preliminary report. *Human & Experimental Toxicology* 13:107-109. PMID: 7908806
25. Mills E, Gunasekar P, Pavlakovic G, Isom G (1996) Cyanide-induced apoptosis and oxidative stress in differentiated PC12 cells. *Journal of Neurochemistry* 67:1039-1046. PMID: 8752110
26. Kanthasamy A, Ardelt B, Malave A et al. (1997) Reactive oxygen species generated by cyanide mediate toxicity in rat pheochromocytoma cells. *Toxicology Letters* 93:47-54. PMID: 9381482
27. Sprince H, Parker C, Smith G (1979) Comparison of protection by L-ascorbic acid, L-cysteine, and adrenergic-blocking agents against acetaldehyde, acrolein, and formaldehyde toxicity: implications in smoking. *Agents and Actions* 9:407-414. PMID: 42290
28. Tamura T, Inoue H, Iida T, Ono H (1969) Studies on the antidotal action of drugs. I. Vitamin C and its antidotal effect against alcoholic and nicotine poisoning. *The Journal of Nihon University School of Dentistry* 11:149-151. PMID: 5268018
29. Halimi J, Mimran A (2000) Systemic and renal effect of nicotine in non-smokers: influence of vitamin C. *Journal of Hypertension* 18:1665-1669. PMID: 11081781
30. Cappelletti G, Maggioni M, Maci R (1998) Apoptosis in human lung epithelial cells: triggering by paraquat and modulation by antioxidants. *Cell Biology International* 22:671-678. PMID: 10452837
31. Vismara C, Vailati G, Bacchetta R (2001) Reduction in paraquat embryotoxicity by ascorbic acid in *Xenopus laevis*. *Aquatic Toxicology* 51:293-303. PMID: 11090891
32. Rappolt R, Gay G, Farris R (1979) Emergency management of acute phencyclidine intoxication. *JACEP* 8:68-76. PMID: 439546
33. Skvortsova R, Pozniakovskii V, Agarkova I (1981) [Role of the vitamin factor in preventing phenol poisoning]. Article in Russian. *Voprosy Pitaniia* 2:32-35. PMID: 6972670
34. Hong S, Anestis D, Ball J et al. (1997) 4-amino-2, 6-dichlorophenol nephrotoxicity in the Fischer 344 rat: protection by ascorbic acid, AT-125, and aminoxyacetic acid. *Toxicology and Applied Pharmacology* 147:115-125. PMID: 9356314
35. Appenroth D, Winnefeld K (1998) Vitamin E and C in the prevention of metal nephrotoxicity in developing rats. *Experimental and Toxicologic Pathology* 50:391-396. PMID: 9784013
36. Laing M (1984) A cure for mushroom poisoning. *South African Medical Journal* 65:590. PMID: 6200941

37. Klenner F (1971) Observations on the dose and administration of ascorbic acid when employed beyond the range of a vitamin in human pathology. *Journal of Applied Nutrition* 23:61-88.
38. Tiwari R, Bandyopadhyay S, Chatterjee G (1982) Protective effect of L-ascorbic acid in lindane intoxicated rats. *Acta Vitaminologica et Enzymologica* 4:215-220. PMID: 6183953
39. Narra V, Howell R, Sastry K, Rao D (1993) Vitamin C as a radio-protector against iodine-131 *in vivo*. *Journal of Nuclear Medicine* 34:637-640. PMID: 8455081
40. Blumenthal R, Lew W, Reising A et al. (2000) Anti-oxidant vitamins reduce normal tissue toxicity induced by radio-immunotherapy. *International Journal of Cancer* 86:276-280. PMID: 10738257
41. Dey P (1967) Protective action of ascorbic acid & its precursors on the convulsive & lethal actions of strychnine. *Indian Journal of Experimental Biology* 5:110-112. PMID: 4383547
42. Klenner F (1954) Recent discoveries in the treatment of lockjaw with vitamin C and Tolserol. *Tri-State Medical Journal* July, pp. 7-11.
43. Dey P (1966) Efficacy of vitamin C in counteracting tetanus toxin toxicity. *Naturwissenschaften* 53:310. PMID: 5986216
44. Jahan K, Ahmad K, Ali M (1984) Effect of ascorbic acid in the treatment of tetanus. *Bangladesh Medical Research Council Bulletin* 10:24-28. PMID: 6466264
45. Mokranjac M, Petrovic C (1964) [Vitamin C as antidote in cases of poisoning by fatal doses of mercury]. Article in French. *Comptes Rendus Hebdomadaires des Seances de l'Academie des Sciences* 258:1341-1342. PMID: 14133923
46. Huggins H, Levy T (1999) *Uninformed Consent: The Hidden Dangers in Dental Care* Charlottesville, VA: Hampton Roads Publishing Company, Inc.
47. Pillemer L, Seifter J, Kuehn A, Ecker E (1940) Vitamin C in chronic lead poisoning. An experimental study. *The American Journal of the Medical Sciences* 200:322-327.
48. Tandon S, Chatterjee M, Bhargava A et al. (2001) Lead poisoning in Indian silver refiners. *The Science of the Total Environment* 281:177-182. PMID: 11778950
49. Korallus U, Harzdorf C, Lewalter J (1984) Experimental bases for ascorbic acid therapy of poisoning by hexavalent chromium compounds. *International Archives of Occupational and Environmental Health* 53:247-256. PMID: 6706420
50. Walpole I, Johnston K, Clarkson R et al. (1985) Acute chromium poisoning in a 2 year old child. *Australian Paediatric Journal* 21:65-67. PMID: 3977793

51. Little M, Gawkrodger D, MacNeil S (1996) Chromium- and nickel-induced cytotoxicity in normal and transformed keratinocytes: an investigation of pharmacological approaches to the prevention of Cr(VI)-induced cytotoxicity. *The British Journal of Dermatology* 134:199-207. PMID: 8746330
52. Chattopadhyay S, Ghosh S, Debnath J, Ghosh D (2001) Protection of sodium arsenite-induced ovarian toxicity by coadministration of L-ascorbate (vitamin C) in mature Wistar strain rat. *Archives of Environmental Contamination and Toxicology* 41:83-89. PMID: 11385593
53. Hudecova A, Ginter E (1992) The influence of ascorbic acid on lipid peroxidation in guinea pigs intoxicated with cadmium. *Food and Chemical Toxicology* 30:1011-1013. PMID: 1473794
54. Shiraishi N, Uno H, Waalkes M (1993) Effect of L-ascorbic acid pretreatment on cadmium toxicity in the male Fischer (F344/NCr) rat. *Toxicology* 85:85-100. PMID: 8303714
55. Chen C, Huang Y, Lin T (1998) Association between oxidative stress and cytokine production in nickel-treated rats. *Archives of Biochemistry and Biophysics* 356:127-132. PMID: 9705202
56. Perminova I, Sinel'shchikova T, Alekhina N et al. (2001) Individual sensitivity to genotoxic effects of nickel and antimutagenic activity of ascorbic acid. *Bulletin of Experimental Biology and Medicine* 131:367-370. PMID: 11550028
57. Swain C, Chainy G (2000) In vitro stimulation of chick brain lipid peroxidation by aluminium, and effects of Tiron, EDTA and some antioxidants. *Indian Journal of Experimental Biology* 38:1231-1235. PMID: 11411045
58. Anane R, Creppy E (2001) Lipid peroxidation as pathway of aluminium cytotoxicity in human skin fibroblast cultures: prevention by superoxide dismutase+catalase and vitamins E and C. *Human & Experimental Toxicology* 20:477-481. PMID: 11776410
59. Klenner F (1954) Case history cure of a 4-year-old child bitten by a mature Highland moccasin. *Tri-State Medical Journal* July
60. Klenner F (1957) The Black Widow spider. Case history. *Tri-State Medical Journal* December, pp. 15-18.
61. Klenner F (1971) Observations on the dose and administration of ascorbic acid when employed beyond the range of a vitamin in human pathology. *Journal of Applied Nutrition* 23:61-88.
62. Klenner F (1974) Significance of high daily intake of ascorbic acid in preventive medicine. *Journal of the International Academy of Preventive Medicine* 1:45-69.
63. Miura N, Saito T, Taira T et al. (2015) Risk factors for QT prolongation associated with acute psychotropic drug overdose. *The American Journal of Emergency Medicine* 33:142-149. PMID: 25445869

64. Ali Z, Ismail M, Nazar Z et al. (2020) Prevalence of QTc interval prolongation and its associated risk factors among psychiatric patients: a prospective observational study. *BMC Psychiatry* 20:277. PMID: 32493330
65. Emamhadi M, Mostafazadeh B, Hassanijirdehi M (2012) Tricyclic antidepressant poisoning treated by magnesium sulfate: a randomized, clinical trial. *Drug and Chemical Toxicology* 35:300-303. PMID: 22309432
66. Levy T (2019) *Magnesium: Reversing Disease*, Henderson, NV: MedFox Publishing
67. Levy T (2002) *Curing the Incurable: Vitamin C, Infectious Diseases, and Toxins*. Henderson, NV: MedFox Publishing
68. Cunningham J, Ellis S, McVeigh K et al. (1991) Reduced mononuclear leukocyte ascorbic acid content in adults with insulin-dependent diabetes mellitus consuming adequate dietary vitamin C. *Metabolism* 40:146-149. PMID: 1988772
69. May J (1998) Reduction of the ascorbyl free radical to ascorbate by thioredoxin reductase. *The Journal of Biological Chemistry* 273:23039-23045. PMID: 9722529
70. Boatright W (2016) Oxygen dependency of one-electron reactions generating ascorbate radicals and hydrogen peroxide from ascorbic acid. *Food Chemistry* 196:1361-1367. PMID: 26593628
71. Evans R, Currie L, Campbell A (1982) The distribution of ascorbic acid between various cellular components of blood, in normal individuals, and its relation to the plasma concentration. *The British Journal of Nutrition* 47:473-482. PMID: 7082619
72. Carr A, Maggini S (2017) Vitamin C and immune function. *Nutrients* 9:1211. PMID: 29099763
73. Ang A, Pullar J, Currie M, Vissers M (2018) Vitamin C and immune cell function inflammation and cancer. *Biochemical Society Transactions* 46:1147-1159. PMID: 30301842
74. Grunewald R (1993) Ascorbic acid in the brain. *Brain Research. Brain Research Reviews* 18:123-133. PMID: 8467348
75. May J (2012) Vitamin C transport and its role in the central nervous system. *Sub-Cellular Biochemistry* 56:85-103. PMID: 22116696
76. Michaelsson K, Melhus H, Lemming E et al. (2013) Long term calcium intake and rates of all cause and cardiovascular mortality: community based prospective longitudinal cohort study. *BMJ* 346:f228. PMID: 23403980
77. Bolland M, Avenell A, Baron J et al. (2010) Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis. *BMJ* 341:C3691. PMID: 20671013

78. Li K, Kaaks R, Linseisen J, Rohrmann S (2012) Associations of dietary calcium intake and calcium supplementation with myocardial infarction and stroke risk and overall cardiovascular mortality in the Heidelberg cohort of the European Prospective Investigation into Cancer and Nutrition study (EPIC-Heidelberg). *Heart* 98:920-925. PMID: 22626900
79. Levy T (2013) *Death by Calcium: Proof of the toxic effects of dairy and calcium supplements*, Henderson, NV: MedFox Publishing
80. Ishimura E, Okuno S, Yamakawa T et al. (2007) Serum magnesium concentration is a significant predictor of mortality in maintenance hemodialysis patients. *Magnesium Research* 20:237-244. PMID: 18271493
81. Reffelmann T, Ittermann T, Dorr M et al. (2011) Low serum magnesium concentrations predict cardiovascular and all-cause mortality. *Atherosclerosis* 219:280-284. PMID: 21703623
82. Wesselink E, Kok D, Bours M et al. (2020) Vitamin D, magnesium, calcium, and their interaction in relation to colorectal cancer recurrence and all-cause mortality. *The American Journal of Clinical Nutrition* 111:1007-1017. PMID: 32190892
83. Sribnick E, Del Re A, Ray S et al. (2009) Estrogen attenuates glutamate-induced cell death by inhibiting Ca²⁺ influx through L-type voltage-gated Ca²⁺ channels. *Brain Research* 1276:159-170. PMID: 19389388
84. Facchinetto F, Borella P, Valentini M et al. (1988) Premenstrual increase of intracellular magnesium levels in women with ovulatory, asymptomatic menstrual cycles. *Gynecological Endocrinology* 2:249-256. PMID: 3227989
85. Xi Q, Hoenderop J, Bindels R (2009) Regulation of magnesium reabsorption in DCT. *Pflugers Archiv* 458:89-98. PMID: 18949482
86. Barrasa G, Canete N, Boasi L (2018) Age of postmenopause women: effect of soy isoflavone in lipoprotein and inflammation markers. *Journal of Menopausal Medicine* 24:176-182. PMID: 30671410
87. Schairer C, Adami H, Hoover R, Persson I (1997) Cause-specific mortality in women receiving hormone replacement therapy. *Epidemiology* 8:59-65. PMID: 9116097
88. Hall J, Jones R, Jones T et al. (2006) Selective inhibition of L-type Ca²⁺ channels in A7r5 cells by physiological levels of testosterone. *Endocrinology* 147:2675-2680. PMID: 16527846
89. Marin D, Bolin A, dos Santos R et al. (2010) Testosterone suppresses oxidative stress in human neutrophils. *Cell Biochemistry and Function* 28:394-402. PMID: 20589735
90. Sharma R, Oni O, Gupta K et al. (2015) Normalization of testosterone level is associated with reduced incidence of myocardial infarction and mortality in men. *European Heart Journal* 36:2706-2715. PMID: 26248567

91. Doulamis I, Tzani A, Konstantopoulos P et al. (2019) Experimental hypogonadism: insulin resistance, biochemical changes and effect of testosterone substitution. *Journal of Basic and Clinical Physiology and Pharmacology* 30. PMID: 31054251
92. Rosenthal S (1968) Acceleration of primary wound healing by insulin. *Archives of Surgery* 96:53-55. PMID: 5635406
93. Oryan A, Alemzadeh E (2017) Effects of insulin on wound healing: a review of animal and human evidences. *Life Sciences* 174:59-67. PMID: 28263805
94. Vatankhah N, Jahangiri Y, Landry G et al. (2017) Effect of systemic insulin treatment on diabetic wound healing. *Wound Repair and Regeneration* 25:288-291. PMID: 28120507
95. Paolisso G, Ravussin E (1995) Intracellular magnesium and insulin resistance: results in Pima Indians and Caucasians. *The Journal of Clinical Endocrinology and Metabolism* 80:1382-1385. PMID: 7714114
96. Cunningham J (1998) The glucose/insulin system and vitamin C: implications in insulin-dependent diabetes mellitus. *Journal of the American College of Nutrition* 17:105-108. PMID: 9550452
97. Dai L, Ritchie G, Bapty B et al. (1999) Insulin stimulates Mg²⁺ uptake in mouse distal convoluted tubule cells. *The American Journal of Physiology* 277:F907-913. PMID: 10600938
98. Zor U, Her E, Talmon J et al. (1987) Hydrocortisone inhibits antigen-induced rise in intracellular free calcium concentration and abolishes leukotriene C4 production in leukemic basophils. *Prostaglandins* 34:29-40. PMID: 3685396
99. Hyde G, Seale A, Grau E, Borski R (2004) Cortisol rapidly suppresses intracellular calcium and voltage-gated calcium channel activity in prolactin cells of the tilapia (*Oreochromis mossambicus*). *American Journal of Physiology. Endocrinology and Metabolism* 286:E626-E633. PMID: 14656715
100. Chincholikar S, Ambiger S (2018) Association of hypomagnesemia with hypocalcemia after thyroidectomy. *Indian Journal of Endocrinology and Metabolism* 22:656-660. PMID: 30294577
101. Wang K, Wei H, Zhang W et al. (2018) Severely low serum magnesium is associated with increased risks of positive anti-thyroglobulin antibody and hypothyroidism: a cross-sectional study. *Scientific Reports* 8:9904. PMID: 29967483
102. Ige A, Chidi R, Egbeluya E et al. (2019) Amelioration of thyroid dysfunction by magnesium in experimental diabetes may also prevent diabetes-induced renal impairment. *Helyon* 5:e01660. PMID: 31193031
103. Ballard B, Torres L, Romani A (2008) Effect of thyroid hormone on Mg(2+) homeostasis and extrusion in cardiac cells. *Molecular and Cellular Biochemistry* 318:117-127. PMID: 18604605

104. Gammie M, Franklyn J, Logan S (1987) Effects of amiodarone and thyroid dysfunction on myocardial calcium, serum calcium and thyroid hormones in the rat. *British Journal of Pharmacology* 92:363-370. PMID: 3676598
105. Zinman T, Shneyvays V, Tribulova N et al. (2006) Acute, nongenomic effect of thyroid hormones in preventing calcium overload in newborn rat cardiocytes. *Journal of Cellular Physiology* 207:220-231. PMID: 16331687
106. Moon S, Kim M, Yu J et al. (2018) Subclinical hypothyroidism and the risk of cardiovascular disease and all-cause mortality: a meta-analysis of prospective cohort studies. *Thyroid* 28:1101-1110. PMID: 29978767
107. Levy T (2019) *Magnesium: Reversing Disease*, Henderson, NV: MedFox Publishing
108. Barnes B, Galton L (1976) *Hypothyroidism: The Unsuspected Illness*. New York: NY: Harper & Row
109. Starr M (2009) *Hypothyroidism Type 2: The Epidemic*. Columbia, MO: Mark Starr Trust

Chapter 14

1. Brownstein D, Ng R, Rowen R et al. (2020) A novel approach to treating COVID-19 using nutritional and oxidative therapies. *Science, Public Health Policy, and the Law* 2:4-22.
2. Issels J (2005) *Cancer: A second opinion*. Garden City Park, NY: Square One Publishers
3. Kulacz R, Levy, T (2014) *The Toxic Tooth: How a root canal could be making you sick*. Henderson, NV: MedFox Publishing
4. Levy T (2017) *Hidden Epidemic: Silent oral infections cause most heart attacks and breast cancers*. Henderson, NV: MedFox Publishing
5. Cunningham J (1998) The glucose/insulin system and vitamin C: implications in insulin-dependent diabetes mellitus. *Journal of the American College of Nutrition* 17:105-108. PMID: 9550452
6. Qutob S, Dixon S, Wilson J (1998) Insulin stimulates vitamin C recycling and ascorbate accumulation in osteoblastic cells. *Endocrinology* 139:51-56. PMID: 9421397
7. Mikirova N, Levy T, Hunninghake R (2019) The levels of ascorbic acid in blood and mononuclear blood cells after oral liposome-encapsulated and oral non-encapsulated vitamin C supplementation, taken without and with IV hydrocortisone. *Journal of Orthomolecular Medicine* volume 34, number 1
8. Riordan H, Hunninghake R, Riordan N et al. (2003) Intravenous ascorbic acid: protocol for its application and use. *Puerto Rico Health Sciences Journal* 22:287-290. PMID: 14619456

9. Marcial-Vega V, Gonzalez-Terron I, Levy T (2015) Intravenous ascorbic acid and hydrogen peroxide in the management of patients with Chikungunya. *Boletin de la Asociacion Medica de Puerto Rico* 107:20-24. PMID: 26035980
10. Levy T (2013) *Death by Calcium: Proof of the toxic effects of dairy and calcium supplements*. Henderson, NV: MedFox Publishing
11. Levy T (2017) *Hidden Epidemic: Silent oral infections cause most heart attacks and breast cancers*. Henderson, NV: MedFox Publishing

