HERBAL ANTIVIRALS

NATURAL REMEDIES FOR EMERGING & RESISTANT VIRAL INFECTIONS

2ND Edition
Revised and Expanded

- Treatment protocols for influenza, enteroviruses, encephalitis, SARS, Covid-19, dengue, the herpes group, Zika, and more
- Comprehensive guide to the most potent antiviral herbs
- Effective ways to strengthen the immune system

STEPHEN HARROD BUHNER
author of *Herbal Antibiotics*

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WHY THIS BOOK EXISTS

It serves little purpose merely to be scared by viruses. But it serves a good deal of purpose to understand them.

—**Frank Ryan, MD, *Virus X: Tracking the New Killer Plagues***

If you aren't skeptical about your skepticism, you aren't a skeptic.

For several decades now, I have been deeply interested in antibiotic resistance, the intelligence of bacteria, and the use of treatment approaches that are, ultimately, more elegant than pharmaceuticals. Plant-based medicines, unlike pharmaceuticals, don't cause resistance problems, they are much safer, *and* they are ecologically sound—they are biodegradable *and* renewable, which most pharmaceuticals are not.

My long-term interest in herbal antibacterials resulted, after a considerable time (and an early initial look at the topic), in a very deep exploration of systemic herbal antibacterials for resistant infections (*Herbal Antibiotics*, second edition, Storey Publishing, 2012). And during that exploration, many aspects of plant medicine not hitherto developed in the West began to reveal themselves (such as the importance of plant synergists).

This book is the beginning, for me, of a similar exploration into the world of viruses, emerging and resistant viral diseases, and more ecologically responsible (and often more effective) forms of treatment. In this book you will find information on some of the best broad-spectrum, systemic, antiviral herbs on Earth. As with herbal antibiotics, they are easy to use, easy to grow, and easy to make into medicines for yourself, your family, your patients. And they are very, very effective for emerging and resistant viral infections. For the plants themselves learned long ago, just as they did with bacteria, how to stop viruses from killing them. Plants can't run but they sure can do chemistry.

The concept of herbal antibiotics as primary interventives has, over the past several decades, become common in cultures outside the Western industrialized nations. Medical systems in Africa, Asia, and South and Central America are turning away from pharmaceuticals as a first-line treatment for bacterial infections because of resistance problems and, most especially, because pharmaceutical corporations make a great deal too much money off the suffering of their populations.

Cultures other than those in the West have realized that they just can't afford corporate greed any longer—*and* they are unwilling to let the poorer members of their populations die because of it. Researchers in cultures across the globe have found that plant antibacterials are often *more* effective than pharmaceuticals. So they are exploring which ones are most potent, which forms of preparation are most effective, and how best to grow them. Then they are traveling throughout their regions (especially in Africa), giving seeds to local villages, teaching them all they have learned, and letting them get on with their healing. There is no middleman raking off profits in the process. A new model of health care is coming into being—and it's about time.

It is my hope that this same kind of movement will begin in the treatment of viral diseases. (And in China, they are already years ahead of us; they *see* the writing on the wall.) We need a new paradigm of healing. We need new ways of thinking about viruses, their emergence, and their treatment—just as we have needed them about bacteria. (Even in the herbal communities in the West, our approaches to viral infections and viruses have been extremely shallow.) There is a lot we can do to create a more effective healing paradigm in our world, one that is ecologically sustainable while at the same time being more human friendly . . . if we step outside the box that our thinking has been trapped by.

I hope that you find the material in this book stimulating to your thinking. I hope that you begin, yourself, to add to this emerging paradigm of healing, one that is slowly extricating itself from the outmoded thinking of the past. We have a great opportunity to create something new, something that reflects more accurately the world around us, something that truly addresses the healing needs of the people who come to us.

I think the viruses are going to be pretty insistent that we do so. And soon.

1

EMERGING VIRUSES: WHAT WE ARE FACING

It is naive to think we can win.

—David Livermore, MD

Viral diseases, caused by pathogenic virus infections which have high morbidity and mortality rates, are still the leading cause of death in humans worldwide. . . . Moreover, the emergence of viral resistance to drugs, as well as the serious adverse effects induced by antiviral drugs, has caused serious medical problems, particularly when [the drugs are] administered in combination over prolonged treatment periods. . . . And these drugs are quite costly, thus limiting their use in developing countries, where infection is most prevalent.

—Kaio Kitazato et al., "Viral Infectious Disease and Natural Products with Antiviral Activity"

For much of the twentieth century, infectious diseases in human populations of Western countries have been in retreat, as we learnt to sanitize our cities, cleanse our water supplies, improve domestic hygiene, use antibiotics, control vector organisms and vaccinate. As a result the developed world became rather complacent, naively welcoming the false dawn of a life mostly free of infectious disease. Since the 1980s things have looked much less secure, however, with the emergence of many previously unrecognized infectious diseases, and the re-emergence of known infectious diseases that were thought to be under control. This trend has continued until the present time and many infectious pathogens, predominantly viruses, have been newly identified.

—Thijs Kuiken et al., "Emerging Viral Infections in a Rapidly Changing World"

During the summer of 2006 a hitherto little-known viral disease swept across a large and diverse range of islands in the Indian Ocean. On the island of Réunion 265,000 people became critically ill—out of a population of 770,000. Very few of those infected were asymptomatic; the illness was, in nearly every case, severe. Health-care workers and the island's hospital system were overwhelmed. Even if they had not been, there was little they could do. So, they offered "supportive" care. In other words, they watched. They waited. Either the person's immune and bodily systems would fight it off, or they wouldn't. For many, they didn't.

The virus soon jumped to India, where an estimated 1.3 million people became ill. The culprit? A relatively little-known viral disease, chikungunya fever.[1] The virus is known to medical science but not well; it's not a common disease. But it had mutated. Later analysis showed that the mutation had occurred sometime between the spring and fall of 2005. Within 6 months it had become pandemic in the region. By the end of 2006 over two million people had become infected.

The disease is attended by severe joint pain (somewhat like dengue fever). The ankles and wrists are the most impacted; conjunctivitis and a rash often occur. The pain in the joints can last for weeks . . . or months, and it is debilitating. There is no treatment and there is no cure. The physicians recommended the use of acetominophen for the pain. The cause of death for many of those who died? Liver damage . . . from the acetominophen.

People visiting the region who traveled back to their homes in the United States and Europe brought the disease with them. Over 1,000 in the United States were diagnosed with it; person-to-person transmission occurred in a number of instances, infecting new hosts.

The disease is primarily spread by mosquitoes (similarly to most of the diseases discussed in this book), specifically *Aedes albopictus*. This is a mosquito that was once limited in its geographical range but has, in the past 50 years, spread to every continent on Earth.

This is an example of just how fast a new viral pathogen can spread in the global village. It began with an African virus entering an Asian mosquito that traveled with people by plane and boat to the Indian Ocean and India. And from there, it went everywhere. This same dynamic is now playing out everyplace on Earth. The chikungunya outbreak is not an uncommon event. West Nile encephalitis virus

emerged in the United States in a large outbreak in 1999. It soon spread throughout the world and is now endemic in Europe and Asia. In the fall of 2002, SARS emerged in China and quickly spread throughout the pan-Asian region. The epidemiologists who studied the SARS outbreak discovered that it had initially emerged in a small region in China. The physician who attended the ill then visited Hong Kong, where he infected 16 people. Some of them traveled and in doing so spread the disease worldwide in a matter of weeks.

Then came swine flu and headlines like these:

Doctors shocked by spread of swine flu—and its severity

—Jeremy Lawrence, *The Independent*, December 22, 2010

460 flu victims fighting for life as experts admit 24 deaths from swine strain may be only a fraction of the true number

—Sophie Borland, *Mail Online*, December 24, 2010

Flu crisis hits cancer surgery: Hospitals struggle to cope as deaths rise and Britain teeters on the brink of an epidemic

—Sophie Borland, *Mail Online*, December 27, 2010

Only a year later, in the fall of 2011, a worldwide epidemic of dengue fever began in northern Africa. It soon spread to the Philippines and Puerto Rico, crossing the ocean in both directions. Finally it hit Brazil. By April of 2012 over 50,000 people had been admitted to hospitals. Five hundred newly infected people were being admitted each week.

And then, of course, in late 2019 the Covid-19 (SARS-CoV-2) pandemic began. It began, as SARS did, in China. In a matter of months most countries, and people, around the globe were affected. Shutdown orders for both businesses and public gatherings were instituted in an attempt to stop the spread of the pathogen. That slowed the spread but did not stop it . . . then it began to resurge. Countries believed free of the infection suddenly were not. And in some countries 10,000 new infections, and 1,000 deaths, were being reported every day. (As the pandemic spread over the next 18 months infections and deaths would increase exponentially.)

The world's economies went into free fall as its peoples and governments struggled to come to grips with the worst pandemic since the 1918 influenza.

Viral diseases such as these are emerging in new and potent forms every place on Earth. Covid-19 will not be the last; it is only the beginning of what we face. There are few medical treatments for these kinds of pathogens if a reliable vaccine does not exist (or cannot be created) . . . and for most of them it does not.

Welcome to the twenty-first century.

But We Won . . . Didn't We?

When the first antibiotic, penicillin, came into common use in 1946 it was heralded as the beginning of the end for infectious diseases. And as each new antibiotic was discovered, and ever more diseases conquered, the voices proclaiming the end of infectious diseases grew louder. The success of antibiotics stimulated concerted medical assaults on epidemic viral diseases, primarily through the development and use of vaccines. The first widespread success was the polio vaccine.

Though it is not widely known, nearly everyone in a given region, when the poliovirus spread to that area, became infected. It was, in fact, a very common virus with very few symptoms for most people. For over 90 percent there were no symptoms at all. Only about 8 percent or so had mild to moderate symptoms, generally a self-limited flu-like condition (which nearly every virus causes). But for about 1 percent of the population, the virus entered the nervous system and those people developed what most people think of as polio. It caused shrunken limbs or paralysis or even, for some, the loss of the capacity to breathe on their own. For those, the rest of life was lived encased in a shell that raised and lowered the chest mechanically.

Oddly enough, given the memories that some people still have of the disease—and the fear it engendered prior to the late twentieth century—it was relatively uncommon. Polio epidemics, as such, were unknown throughout most of human history. But by 1910 major epidemics of the disease began to sweep the world. It became the scourge of the industrialized nations.

The success of antibiotics after World War II combined with the deep public fear of the disease drove a powerful medical movement in the search for a cure. And as with most viral diseases, the focus was on a vaccine. It didn't take long. The Salk vaccine was discovered in 1955 and,

after trials, in 1962 it was licensed for widespread use. Worldwide epidemics soon faded from memory, and infections declined from the millions to the hundreds of thousands, and by 2007 to a mere 1,652, almost all in Asia and Africa. It was a powerful success story and the belief that medical science could defeat all infectious diseases spread.

A 1963 comment by the Australian physician Sir F. Macfarlane Burnet, a Nobel laureate, is typical. By the end of the twentieth century, he said, humanity would see the "virtual elimination of infectious disease as a significant factor in societal life."[2] Seven years later, Surgeon General William Stewart testified to Congress that "it was time to close the book on infectious diseases."[3] And for a while, it seemed they might be right, for the next viral disease they attacked was smallpox.

Though they rarely get credit for it, it was Russian physicians in 1958 who began to press for a worldwide program to permanently eradicate smallpox. By 1967 the program was in full swing with some 250 million vaccinations occurring throughout the world each year. Within a decade, mostly due to the efforts of an American physician and epidemiologist, Donald Henderson, the last regions still endemic for the disease were cleared. And the world celebrated. For the first time a major human disease pathogen had been eradicated from the planet. (Though to be fair to the virus, both the Russians and the Americans kept just a little around, in case they needed it later—you know, for the kids.)

The triumph over smallpox was the apex of the success of the medical assault on microbial disease pathogens—though few knew it at the time. It was thought to be the beginning of the end for every disease pathogen on Earth. The hubris level, already immense by 1963, grew larger. If humans could defeat smallpox, they could defeat every virus on Earth. The word spread; the newspapers were filled with optimistic scenarios of a future without disease. Researchers were quoted extensively (as they still are) as saying that, soon, in just a few years, none of us would die from infectious diseases. Most people in the industrialized world accepted this at face value . . . as they still do. It is, regrettably, part of the utopian future-myth of science (especially medical science) that many people take for granted. But it never has, and never has had, much to do with reality. As physician and researcher Frank Ryan comments, "Perhaps it reflected, in part, a regrettable separation of clinicians from basic scientists." He goes on to say:

> In fact those people whose living depended upon a study of microbes, of their potential and durability, were never deluded. A prescient few, such as René Dubos, warned us openly that the optimism was unjustified. But on the whole people were not inclined to listen. Most doctors, never mind members of the public, were infected with the prevailing overconfidence, hardly perceiving the growing threat of social changes to the "global village." They seemed unable to grasp the new potential afforded to a very ancient peril arising from world travel. . . . Today, as one after another of the dismissed plagues returns to haunt us, as new plagues every bit as deadly as anything seen in previous history threatens our species, it is obvious that the postwar years were an age of delusion. It was comforting, a very understandable delusion, but a delusion nonetheless.[4]

As it happens, the scientific and medical beliefs about the Earth and its interrelated life forms, including bacteria and viruses, that have been widely spread are not very accurate. Lynn Margulis and Dorion Sagan, in their book *What Is Life?*, note that once "the germ theory of contagion finally caught on, it did so with a vengeance. Different types of bacteria were implicated in anthrax, gonorrhea, typhoid, and leprosy. Microbes, once amusing little anomalies, became demonized. . . . [They] became a virulent 'other' to be destroyed."[5] But there are many problems with this belief about microbes. Two stand out for me. The first comes from the particular medical paradigm in use in the West and the second from a very inaccurate, outmoded nineteenth-/early-twentieth-century view of nature.

The medical paradigm problem is simple enough, though it's rarely recognized for the problem it is. Specifically: Most physicians and medical researchers commonly speak of the "cause of death" when speaking of mortality. The assumption, deeply embedded within that communication, is that the bacteria or virus (or heart disease or stroke) caused the death. Even deeper is the communication that if all "causes" of death were defeated, then there would be no more death. As Harvard researcher and zoologist Richard Lewontin puts it,

> The claims made by medicine imply this possibility without explicitly stating it. Medical scientists speak of 'preventing' deaths by curing disease, but the evidence is that death cannot be prevented, only postponed at best. Moreover, the postponement has not been as effective as is sometimes claimed during the last fifty years of great progress in physiology, cell biology, and medicine. . . . [The truth is] that although the proximate causes of death can be dealt with, death itself cannot. So, there must be a cause of death as a *phenomenon*, as distinct from the individual cases.[6]

In other words, if *every* "cause" of death were removed, in spite of what physicians (and news reports) say, there would still be death. Death is inherent in this place. The unstated and deeply buried assertion that microbes "cause" death is not only false, it stimulates people to view microbes as enemies, as participants in a war against us—and this is very far from the truth.

Bacteria and viruses are not a "virulent other." They are, instead, intimately interwoven into the underpinnings of life on this planet. They cannot be killed off without killing off every form of life on Earth. This is the great error of the nineteenth-/early-twentieth-century view of nature that continues to plague us. Or, as Lynn Margulis once put it, "The more balanced view of microbe as colleague and ancestor remains almost unexpressed. Our culture ignores the hard-won fact that these disease 'agents,' these 'germs,' also germinated all life."[7] We are, counter-intuitively and most upsettingly, only a form of bacteria ourselves—in extremely elegant, symbiogenic, innovative shapes. Bacteria are the foundation of every life form on this planet. Had bacteria not developed resistance to antibiotics all life on this planet would have already become extinct simply from the millions of tons of antibiotics now present in the environment.

With bacteria the paradigm problem is bad enough, but when viruses enter the picture, the complexity rises by orders of magnitude. Viruses are not and never have been parasites, though they may act as or seem to be parasites when we fall sick with one. They, in fact, perform highly elegant ecosystem functions—as everything on this planet does. Viruses, as Frank Ryan comments, "weave in and out of the genomes of every form of life on earth. As a result, terrestrial life [has] become a dense web of genetic interactions."[8]

DNA is not, and never has been, a computer program (and neither is RNA). It is, as Nobel Prize winner Barbara McClintock once noted, a living organ of the cell. DNA and RNA are both similar structures and they are deeply interactive with the world around them. DNA is a double-stranded molecule, RNA is single-stranded, but both of them are intimately involved in the structural formation of life forms. DNA contains information about the genetic development of living organisms. RNA is a messenger molecule used to carry genetic information that directs, in part, the synthesis of proteins, proteins that are needed for the structural formation of organisms. But again, these nucleic

acids are not fixed in form. They change. We live in the midst of constant genetic rearrangements. And these gene rearrangements occur not only in response to impulses within the organism but also, as Barbara McClintock observed, to communications from the environment around it.

There is, in fact, no discrete inside and outside, no "us" and "them," even though it seems (within our nineteenth-century paradigm) that there is. The life forms on this planet are living organisms and that means they possess soft boundaries, *very* soft boundaries. There is a constant exchange (of energy, for example) between the inside and outside in all living systems. But to be more direct there is a constant inflow and outflow of life through those soft boundaries. The nature of the ecological reality of this world demands it.

As Richard Lewontin observes:

> Even virus particles, which do not metabolize energy, can reproduce only when they become integrated into the metabolic apparatus of the cells they infect. At the time of viral replication, there is a complete abolition of the previously existing boundary between the virus and its cellular environment. . . . Organisms do not find already existent ecological niches to which they adapt, but are in the constant process of defining and remaking their environments. At every moment natural selection is operating to change the genetic composition of populations in response to the momentary environment, but as that composition changes it forces a concomitant change in the environment itself. Thus organism and environment are both causes and effects in a coevolutionary process. . . . Small changes in environment lead to small changes in the organism which, in turn, lead to small changes in the environment. . . . In general the organism and the environment must track each other continuously or life would long ago have become extinct.[9]

Specifically, viruses have the capacity to enter cells, snip off sections of DNA (or RNA), and weave them into their own genetic structure. They can then weave those sections, as well as sections of their own genome, into other living organisms. One of their main functions in fact is the genetic intermingling of all life forms on Earth. Or as Frank Ryan puts it, "Viruses are vehicles for genetic exchange between the disparate species that make up the matrix of life on earth."[10] Our genome, as that of all life on this planet, contains snippets of the genetic codes of multiple other life forms. It also contains snippets of viral genes. Our forms, our shapes, are an expression of a communication that has been ongoing since life has been. We *are* the enemy we have been fighting.

In short, the perspectives of most scientists at the beginning of the antibiotic era were limited, more limited than even the most prescient

knew. And the view of the world that they disseminated was deeply flawed. Viruses are deeply interwoven into the ecological matrix of this planet and they serve functions that are essential. Further, they are extremely adaptable. They can alter their structure very rapidly and take on new forms seemingly at will. All based on their analysis of the environment that surrounds them. They are highly intelligent. They are not in fact stupid. But we certainly were (and are) when we thought them so.

The paradigm of medicine that emerged in the twentieth century was wrong and we are now paying the price for applying it so comprehensively. We now face the emergence of epidemics more devastating than any known before. If *we* are to adapt, we have to see with different eyes, understand through a different paradigm. We have to realize that viruses are not what we thought them to be. We have to learn to see them as they are.

Viruses

To begin with, if you have ever read much about viruses, or if you have looked closely at some of the words I have used while writing this, you might have noted that many people refer to viruses as "particles." There is a widely held belief among scientists that viruses are not alive. Many insist that they are merely organic structures that interact with living organisms. Some say they are "organisms at the edge of life" but really aren't organisms in any meaningful sense. This is, they say, because they lack a cellular structure, don't have their own metabolism, and can only reproduce *inside* cellular structures—and besides they don't use cellular division to do it either. If asked where the definition of life comes from, a definition that precludes viruses, biologists will admit, under duress, that people invented the definition but nevertheless, it's true anyway. (This explains why manufacturers put a warning label on those sun shields people occasionally place in the front windows of their cars to protect the interior from the sun—"Warning: do not drive with shield in place.")

Oddly enough some bacteria, such as rickettsia and chlamydia, are considered living despite having similar limitations. (And the Earth is not alive, though it may "act" like a living system. Why? Because it consumes

its own waste, which, as everyone knows, living organisms *never* do . . . apparently they have not met my neighbor's dog.)

There are a lot of viruses on this planet. Estimates are that the Earth contains 10^{31} viruses, that is, a 10 with 31 zeros after it. Technically (for wonks) that is somewhere between a nonillion and a decillion (though if you just say bajillion you are close enough). Essentially—a whole lot. And there are multitudes of different kinds. In every 200 liters of water (about 50 gallons or the contents of a typical hot water tank) some 5,000 different viral genotypes are present. There are viruses in the coldest, most inhospitable parts of this planet and viruses inside boiling hot springs. There are viruses high in the atmosphere and in the deepest wells on Earth. They live on top of mountains and in the furthest depths of the ocean. And, sometimes, they even travel into space. They are a part of life on this planet; there is no avoiding them.

Viruses, unlike bacteria, have no nucleus and no cell wall. They are the minimum of life honed to a structural simplicity. Though there are many kinds, in general, a virus is a strand of DNA or RNA surrounded by a mathematically elegant polyhedron, called a capsid, whose shape is virus specific. For what are called "enveloped" viruses, the capsid is surrounded by one or more protein envelopes. This simplified structure makes them different than bacteria, for example, but no less alive. They *are* a unique life form (but that is no reason to discriminate against them). They are very much like seeds (or spores): They only grow when they find the right soil in which to do so. And like seeds, even though in a suspended state, they constantly monitor the exterior world around them.

The surface of the viral protein envelope is studded with receptors, specific kinds of sensory organs that tell viruses about their surroundings. Viruses use these elegant sensory organs to analyze the environment in which they find themselves and to help them find the cells they are most suited for. As physician and researcher Frank Ryan comments:

> Viruses have a kind of sensation that could be classed as intermediate between a rudimentary smell or touch. . . . They have a way of detecting the chemical composition of cell surfaces. . . . This gives a virus the most exquisite ability to sense the right cell surfaces [allowing it to find its unique host cell]. It recognizes them through a perception in three-dimensional surface chemistry.[11]

Viruses have a highly sophisticated capacity to sense the environment around them, to determine its nature, to find the cellular organisms

within which they can most easily reproduce, and to then stimulate the organisms in which those cells reside in order to spread the viruses to new hosts. And they are very good at surviving. They can analyze the nature of the immune response that occurs against them and they can alter themselves—or alter the host immune defense itself—in order to avoid it. They can *reason* by any useful definition of the term, that is: analyze inputs and create new behaviors based on what they have determined the meaning of those inputs to be.

Viruses are typed in a number of ways: by size or shape, presence or absence of an enclosing capsule (not all have one), whether DNA or RNA based (and from that whether single or double stranded, positive or negative sense), their type of protein structure, and their manner of replication. DNA viruses are fairly reliable as viruses go because they have a kind of "copy-check" mechanism that RNA viruses lack. This means that when a DNA virus is making more of itself within a host cell, it uses a biofeedback loop to make sure the copies of itself are reasonably accurate. In contrast an RNA virus can't. It tends to make a whole lot of copies that vary, sometimes a great deal, from the original. Some of these copy differences are initiated intentionally by RNA viruses to increase their genetic variation and, hence, survivability in the host. Because of this, while it is often possible to come up with a lasting vaccine for a DNA virus (smallpox, for example), it is much harder, and sometimes impossible, to make one for most RNA viruses. (Notable exceptions are those for measles, mumps, rubella, and polio, which all have very stable genomic structures.) This also makes RNA viruses very hard to treat with pharmaceuticals; they, like bacteria, begin creating solutions to synthetic drugs the moment they encounter one. Evidence indicates that the mutation rate of the hepatitis C virus, for example, accelerates in response to interferon and ribavirin therapy in much the same way that bacterial alteration occurs in the presence of antibiotics. Infection

> We live in an era of rapidly changing global landscapes and local environments. Viruses with RNA as their genetic material can quickly adapt to and exploit these varying conditions. . . . It comes as no surprise, then, that several prominent recent examples of emerging or re-emerging viruses are caused by RNA viruses.
>
> —Stuart Nichol et al., "Emerging Viral Diseases"

with an RNA virus like West Nile or Japanese encephalitis is actually quite different than infection with a DNA virus.

While DNA viruses make billions more of themselves, most RNA viruses make billions of similar but not identical viruses. It is something like a swarm of honeybees—all similar but all different. In fact it is much more accurate to think of an RNA viral infection as infection by a viral swarm, each member of the swarm possessing a slightly different genetic structure.

(The most stable RNA viruses have a very low "mutation" rate; they make new copies of themselves that tend to be very similar to each other. In essence they tend to conserve certain genomic structures from generation to generation. And it is those stable structures that vaccines teach our immune systems to recognize, which is why the vaccines work.)

There is also evidence that both DNA and RNA viruses, like bacteria, share information among themselves in order to remain unaffected by medical treatments or immune systems. Similar viruses will actively share genetic structure to create very difficult-to-treat infections. Influenza viruses (for instance) specifically (and intentionally) both rearrange their genetic structures and insert entirely new genes within themselves on a regular basis in order to remain invisible to the human immune system. And they gather these new gene sequences from pigs and birds in Asia. This is why a new vaccine is needed every year for the flu.

Viruses, when not in a living cell, go into a state of hibernation much like plant seeds. In this state of dormancy they move with air currents, in water, or simply rest dormant on the ground until they come into contact with a life form that contains the cells they need to awaken from their long sleep. At that moment a virus's first task is to get inside the new host organism, bypass its protective mechanisms, and find the proper host cell. Viruses use highly elegant analysis to address these challenges; they actually begin experimenting with new combinations of genes to adapt to the environment they face. Most of them have also generated a genetic structure that facilitates their entry into other host organisms after an initial infection begins. The rabies virus, for instance, affects a part of the brain that then causes uncontrolled biting. At the same time, the virus swarms in its billions into the saliva of infected animals. Then, every time the animal bites something the virus is transmitted to a new host. Influenza, and other respiratory viruses, enter respiratory droplets and then stimulate coughing or sneezing.

Those droplets are then breathed in by new hosts. And still other viruses, spread by mosquitoes, flood into the blood and there stimulate the release of chemicals through the host skin surface that calls mosquitoes to the infected host so that the virus can be picked up and spread to others. Viruses are very good at getting from *here* to *there*.

Viruses spread by ticks or mosquitoes take advantage of the compounds in the arthropod saliva to facilitate their entry into the new host. The salival compounds reduce certain immune responses in the host to allow the arthropod to feed and often anesthetize the bite location as well. The lowered immune responses at that location allow the viruses to enter the new host in a place where there will be little resistance. Once inside the viruses will make their way to the draining lymph node nearest the bite location and be carried to the spleen via the lymph. There they will begin altering the host's immune function, reducing the capacity of immune cells to recognize and kill invading microbes. Once that occurs, the viruses will catch a ride on immune cells, macrophages or monocytes usually, and begin spreading throughout the body. This is common for encephalitis viruses, for example. They will then travel in the lymph to the barrier between the brain and the rest of the body, release compounds that make the barrier more porous, enter the brain, and find the cells they really prefer: brain neurons.

> The present period of unprecedented ecological change and the growing economic and social crises that are driving vast movements of hosts are together contributing to the resurgence of old pests and the appearance of new ones. Important components in this rapid evolution are the vulnerabilities of ecosystems and instabilities in climate.
>
> —Paul Epstein, "Emerging Diseases and Ecosystem Instability: New Threats to Public Health"

Other viruses enter through being inhaled (influenza) or through sex (HIV) or through being eaten on food (enteroviruses). Once in the body, they hitch rides on whatever cells they have developed a taste for (usually immune cells, for those cells travel *everywhere*) and actively seek out their preferred location. Such is the case with HIV, which views T4 lymphocytes as the perfect host cells, or the Epstein-Barr virus, which has an affinity for human B cells, or the Japanese encephalitis virus, which loves monocytes.

To hitch rides, a virus uses chemotactic compounds that allow it to stick to its preferred "taxi" cell. The receptors on the surface of the virus fool the cell into thinking it is a compatible protein that has attached, and through a series of chemical communications, the virus gets the cell to let it inside. Basically, it gets the cell's confidence, then abuses it. From there the virus is carried everyplace it might need to go in the body. Once near its preferred location, it leaves its ride, attaches to the cell that is most specific for it, and once again fools the cell into taking it inside. Now it begins to replicate in its millions.

Once inside the primary habitat cell, the virus sheds its protein coat and begins taking over the cell. First it stops the cell from dying, which infected cells are programmed to do, and there it remains, protected from the rest of the immune system. It then breaks off pieces of itself and sends them into the nucleus of the cell, which is then tricked into making copies of the virus, using the viral proteins as a template. These new viral particles exit the nucleus, travel to the interior of the cell wall, and bubble out (viral budding, they call it). The cell dies during this process and bursts apart, and the viruses take up parts of the cell membrane and make themselves new viral protein coats with receptors for new host cells. And it all happens very quickly.

Thus the ancient struggle begins: finding out which is in better shape—the organism's immune system or the replicating virus. If the virus is particularly strong or if the immune system is compromised in any way, the virus can really take hold and illness, sometimes severe illness, is inevitable.

Emerging Pathogenic Viruses

Similarly to antibiotic-resistant bacteria, many of the viruses long thought conquered are making comebacks. They are doing this through genetic rearrangements, through learned resistance to antivirals, and, most of all, because of changes in the world in which all of us live. And those changes are profound.

Following are some of the major alterations affecting the planet that researchers have identified as being behind the emergence of so many new (and old) pathogenic viruses. There is no place on the planet that has escaped them.

- **Demographic changes:** human population and refugee increases, accelerated mobility, worldwide urbanization, increasing population density in confined spaces such as inner cities and prisons
- **Medical care and technology:** medical-related infections in hospitals, concentrated microbial intermingling in hospitals and nursing homes, blood transfusions, organ transplantation, reuse of medical equipment, pharmaceutical contaminants, viral and antibiotic resistance
- **Economic and commercial trends:** overly extensive industrial agriculture with the resultant damage of ecosystem homeodynamis; worldwide disbursement of commercial food animals, food plants, and agricultural pharmaceuticals
- **Ecosystem disturbance:** deforestation, waterway disturbances, reduction in predator populations, destruction of wild plant populations
- **Climatic changes:** disruptions in climate homeodynamis by anthropogenic factors such as global warming, CO_2 increases, and pollutant gases

Viruses have lived for millions of years in balance with their host species (such as wild bee or buffalo populations). The disruption of healthy ecosystems by human incursions and the resultant loss of host species and their habitats stimulates viruses to jump species. And one of the species into which they jump is us (or the animals that survive well with us: pigeons, mice, pigs, and chickens—from whom they then easily move into us). After all, there are a lot more of *us* now (and our food animals) than any other large life form; we aren't that hard to find. As a matter of fact many of us live in the same places the viruses' former hosts did. And a home is a home. Our bodies are not all that different from the other animals on this planet. It is a simple adjustment for the viruses to make *us* their new hosts.

Some of the most common emerging, resistant, or newly virulent viruses are dengue virus (which infects millions worldwide every year), hepatitis C, enterovirus 71, HIV, and the eight members of the herpes family that affect humans, including cytomegalovirus and Epstein-Barr virus. But by far the ones that are causing the most worldwide concern (outside HIV) are the influenza and encephalitis viruses.

Influenza, which often seems a somewhat mild disease in many people's minds, just another "case of the flu," is actually a very potent viral pathogen. Epidemiologists have been warning, with increasing insistence, that a worldwide pandemic similar to the one that covered the globe in 1918, a pandemic that infected over 500 million people and caused some 100 million deaths, is due soon. Our factory farms (in whose pigs and chickens the viruses increase their virulence), our growing populations, the hubris and outdated nature of our medical system, and the intelligence of viruses make it certain that a pandemic strain will emerge again. It is only a matter of time. As Robert Heinlein once said, "Population problems have a horrible way of solving themselves."

The viruses are learning. We should, too.

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VIRAL RESPIRATORY INFECTIONS AND THEIR TREATMENT

Persistent host-specific viral agents are the origin of emerging acute epidemic disease following adaptation of that virus to new host species. . . . These acute viruses have a high dependence on host population structure as described by the apparently accurate mathematical models that resemble predator-prey dynamics in which the viruses act as predators on their host prey. . . . Acute human influenza A represents a host species jump of a persisting viral agent of aquatic birds.

—Luis Villarreal et al., "Acute and Persistent Viral Life Strategies and Their Relationship to Emerging Diseases"

The major animal reservoirs of Influenza A are migratory birds and the majority of all the possible combinations of HA-NA subtypes have been isolated from them.

—Andrew Pekosz and Gregory Glass, "Emerging Viral Diseases"

Most of us think of the flu as a fairly minor disease, and for most of us it is. At worst we lie in bed for a week or so, feeling miserable. But for the old and the very young the flu can be deadly; it kills those with the weakest or least developed immune systems, some 30,000 people a year in the United States. But sometimes a real pandemic happens and the death rate rises. It has never risen more than it did in 1918.

The 1918 world influenza pandemic is the most deadly plague that human beings have *ever* experienced. It began in 1918, just as war was drawing to a close, and lasted until December of 1920. World War I (which ended in 1918) killed some 17 million people. In contrast, the influenza pandemic, spread around the world by returning soldiers, killed six times as many in half the time—perhaps as many as 130 million people. The first wave of the pandemic began in January of 1918 and it was fairly routine. People became ill but only the very old and very young died; it was, so far, a pretty typical flu. But the virus soon mutated. And the second wave? It was deadly. It killed those with the strongest immune systems. Half of those who died were between the ages of 20 and 40; nearly all were under age 65. And it killed them by the millions.

Instead of the usual respiratory infection, with death occurring as the lungs filled with fluid, massive hemorrhages took place. The infected lung cells, and those nearby, damaged by the virus-stimulated cytokine storm (see page 32 for more on cytokines), literally burst open from the inflammation. And unlike most viral influenzas this one did not stay confined to the respiratory system. It spread to the GI tract, the brain, and every mucous membrane system in the body. First it destroyed the infected mucosal epithelial cells, then the blood vessels that fed them inflamed and burst open. Bleeding was extensive from the nose, stomach, and intestines; hemorrhages from the skin and ears were common. The infected literally bled out. And nothing physicians tried would stop it.[1]

To understand the impact, consider the fact that, in a world reeling from war, *one-third* of the entire world's population contracted the disease—over 500 million people. In some places half the population was bedridden. As troops demobilized, the ships returning them home from war stopped at hundreds of ports along the way, and the infection spread across the globe. On the islands of Western Samoa 90 percent of the population fell ill—*simultaneously*. Thirty percent of the men, 22 percent of the women, and 10 percent of the children died.

In an attempt to stop the infection, port quarantines were put into effect around the world. Most were too late to do any good. One out of every three persons on Earth fell ill. One out of every five of those died. Five percent of the total world population—one out of every 20 people—did not survive the pandemic. Within the first 6 months 25 million

people died, more than were killed in 5 years of war. Entire towns and cities were shut down.

There were few professionals to help the sick. Doctors and nurses were the first responders and they succumbed immediately. (The morticians followed soon after.) The infected filled the hospitals, school gymnasiums, auditoriums—every large building that could hold masses of people was pressed into service. The beds and the floors were awash in blood as the people died . . . and hemorrhaged by the hundreds and the thousands while doing it. And the bodies piled up. Steam shovels were brought in and mass graves dug—row after row after row of identically sized holes stretching across empty fields in a terrible mockery of industrial expediency. Then the trucks came, the bodies piled high on the wooden beds, and the masked workers dropped them in, hour by hour, day by day, month after month. And behind them, the steam shovels covered them over . . . one by one, day by day, for the 2 terrible years of the pandemic. There were few coffins and often no headstones. The system was completely overwhelmed. Not even a full century of the Black Plague had killed like this. In the history of human habitation of Earth, never had a disease spread so quickly around the world nor killed so many in so short a time. Only one place on Earth reported *no* infections: the tiny island of Marajó near Brazil in South America. And then, as inexplicably as it had begun, in a 1-month period of time, between November and December of 1920, the pandemic ended, simultaneously, around the globe.

Much effort has been expended in recent years in an attempt to understand what made that particular influenza strain so much more deadly than all the others people have known. It turns out that there were two interrelated events that came together in just the right way at just the right time in a terrible serendipity of the universe. From that intermingling came the worst pandemic the human species has ever experienced.

The first event was the emergence of a new strain of influenza at just the right time in human history. An analysis of the viral genome from 1918 has revealed that a new influenza strain had jumped species (from birds) just prior to 1913. By 1915 the virus had split into two types: one infecting pigs, the other humans. The second event was the war itself, which began with perfect timing in 1914.

Normally, when people fall ill with the flu, they go home and rest. The soldiers could not and the new influenza strain rapidly spread throughout the troops on both sides of the conflict. Constrained in cramped, unhealthy conditions, in hospital tents and in trenches, the soldiers were a perfect breeding ground for the virus. And sometime between 1915 and late 1917 the virus mutated again, this time into a form that could powerfully infect just that kind of population: the young. Then, the war over, the soldiers, millions of whom were infected, were crowded together in ships (there was no air travel then) that sailed from port to port to port, infecting as they went. Once home port was reached, the soldiers took trains, buses, and cars to their individual towns and cities. And the virus went with them, infecting everyone.

Re-created forms of the strain, patiently assembled in laboratories, when given to primates, have been found to generate the same symptoms as those described, in depth, by the physicians who treated the 1918 pandemic. An analysis of the physiological damage that occurs found that the reason the disease is so severe is that the virus creates a tremendously potent cytokine cascade in the body—a cytokine storm. A perfect storm. These cytokines are immunoregulatory proteins stimulated by the body's innate immune system in response to infection. The cytokine cascade is how the body attempts to kill off the invading pathogen. But this was much more than the usual immune response. The immune reaction was extreme, somewhere between 100 and 1,000 times what would be normal in those who were infected. And that overreaction, much more pronounced in those with strong immune systems, is what killed so many so quickly.

It is just this kind of influenza pandemic that epidemiologists and viral researchers fear will emerge once more. Given the current population density (and the crowding in prisons, nursing homes, hospitals, day care centers, and inner cities), the ecological disruptions that are occurring worldwide, the number of viruses that are jumping species, the rate of mutation, *and* the vast and very rapid movement of people via air travel, they say it is only a matter of time. And in spite of the many advances in medical technology, there is very little that modern medicine can do to treat a widespread pandemic of deadly influenza. Pharmaceutical antivirals are only partially effective for this kind of infection and the stocks of those antivirals are insufficient to deal with a true pandemic. And vaccines? Vaccines take time.

Flu vaccines have to be made for the specific virus that emerges *in that year*. This means that the disease will already be moving throughout the world before production even begins. And if it is a true pandemic of a deadly strain, by the time the vaccine is produced and shipped (normally a 3- to 6-month process), the infrastructure of the world will already be failing. The health-care workers, hospitals, and transportation workers will be the first to fall. Then the morticians and cemetery workers. The system will begin to shut down. Quarantines, forcing people to stay in their homes, will be put into effect to try and stop the spread. And people will survive as best they can, just as they always have.

The Influenza Virus

The influenza virus is a member of the Orthomyxoviridae family. It is an RNA virus and that means it alters its genetic structure very quickly. That is why a new flu shot is needed every year (for those in the Western world who have such things available). The old vaccine can only help prevent infection by the strain that has emerged in that particular year. The next year, it is not the same virus, merely a similar one. Influenza viruses spread around the world every year in seasonal epidemics; 250,000 to 500,000 people die from them each time.

About one-third of people who are infected remain asymptomatic; the rest get some degree of the "flu." The first symptoms are usually a feeling of being cold or achy and perhaps the beginnings of a fever. High fever alternating with severe chills sets in as the infection spreads. As the virus enters the lungs and sinus tissues mucus congestion begins. Coughing, body aches, fatigue, headache, and irritated eyes, nose, and throat are common. Some people will have diarrhea and abdominal pain. Vomiting. Sometimes. Yes.

The symptoms of the infection usually begin the third day after infection. But the virus is already well established by then. It starts replicating the second day, then begins "shedding" viral particles that are released in increasing numbers for the next 5 to 7 days. The higher the fever, the more viral organisms that are being released. Children are *extremely* infectious compared to adults, with very high viral loads. They also tend to have very high fevers.

As the virus invades the lungs it stimulates inflammation in the tissues. The lung cells, filled with viruses, soon bulge outward and explode—the essence of viral shedding. Then the virus stimulates coughing, spreading the virus to new hosts via respiratory droplets. Pneumonia, a severe inflammation of the lungs accompanied by massive fluid retention and an inability to breathe, is the main cause of death. People, in essence, drown.

There are three different groups of influenza viruses, denoted A, B, and C. Influenza A is the most virulent. Influenza B is a relatively stable virus and mutates much more slowly than A. Most people develop, in childhood, at least some immunity to it; it is much less dangerous. Influenza C is fairly rare. It does infect people, sometimes severely, but it usually causes only a mild illness, generally in children. When people talk about an influenza pandemic, what they are talking about is influenza A in one of its many genetically altered forms. The 1918 pandemic was caused by an influenza A strain.

There have been numerous pandemics of influenza over the years, each caused by a different strain of the virus. The one in 1918 was the beginning of the modern influenza pandemic era; such pandemics were much less common before then. There was a long rest after 1918. Since 1957, however, they have been occurring with greater frequency.

The most dangerous strains, currently, are H1N1, which caused the flu pandemic of 1918; H2N2, which caused the Asian flu pandemic in 1957; H3N2, which caused the Hong Kong flu pandemic in 1968; and a relatively new one, H5N1, known as avian or bird flu, which caused a pandemic in 2004. Then H1N1 came again. It was the source of the swine flu pandemic in 2009 and is a modified descendant of the 1918 H1N1 strain.

The influenza virus alters its genetic structure rather significantly every year by passing through both pigs and birds. And on that trip it exchanges genetic material with other viruses and reworks its own. Then it spreads around the world again by plane and boat, rail and car, infecting millions, causing what we call the yearly flu season. But every so often it develops a much more virulent strain, sometimes through unique genetic rearrangements, sometimes through species jumps, sometimes through both. The Asian flu pandemic in 2004 was a species jump. The swine flu epidemic of 2009 was a unique genetic

rearrangement. It occurred when the virus took advantage of giant agribusiness animal crowding.

Viral geneticists have traced the lineage of the 2009 swine flu epidemic, a virulent H1N1 strain, to an H3N2 strain that emerged in 1998 in U.S. factory farms, specifically huge hog farms in which the animals are so tightly packed together that they literally cannot move. This H3N2 strain combined with another swine strain, a European H1N2 variant, rearranged genetic material into a new and very potent H1N1 form, and then emerged into the human population. The earliest infections occurred in La Gloria, Veracruz, Mexico, just adjacent to a huge hog farm. The workers became infected with the new strain, went home, infected others, many of whom traveled to other cities and towns, and the pandemic began. And it was particularly deadly for those who were infected. Among those hospitalized, depending on location, up to 31 percent were in intensive care units, and as many as 46 percent of those receiving intensive care died.

One of the main fears that epidemiologists and viral geneticists have is the possibility of a combined swine and avian flu strain. The crowding of human food animals, similar to the crowding of soldiers in trenches in World War I, continually allows for the emergence of potently virulent strains. Chicken farms, in which unique avian flu strains can emerge, and hog farms, in which unique swine strains can emerge, are perfectly positioned to allow the combination of the two into one potent, and very deadly, influenza strain. This kind of combined strain can then pass easily into farm workers and thence into the population at large.

Researchers have found that, indeed, the H3N2 swine flu virus easily combines with H5N1 strains of avian flu. When that occurs, a tremendously pathogenic form of the virus emerges. It is, they insist, only a matter of time until it occurs on its own. In fact, studies of pigs on large farms adjacent to poultry farms have found such viral combinations already infecting pigs. That combined viral strain has not infected people . . . yet.

Infection Dynamics and the Cytokine Cascade

Cytokines are physiological signaling molecules produced by the body for a variety of reasons. They are produced in the largest numbers during infections. Cytokines (and their cousins, chemokines) are generally part of the innate (rather than the adapted) immune system. They are intended to respond to incursions into our bodies by viruses and bacteria. Another way to think of them is as inflammatory molecules. They cause various sorts of inflammation in the body—they are why, when you cut yourself, the wound gets red and tender and swells. The cytokines rushing to the area create conditions in which many bacteria and viruses find it difficult to survive. Unfortunately for us, bacteria and viruses have also learned how to use our own immune responses for their purposes. They subvert them, quite often, to facilitate their infection of the body *and* their destruction of certain areas of the body. This facilitates their reproduction and allows them to gather nutrients. Influenza viruses love the lungs and it is where they cause the greatest damage.

Unlike encephalitis viruses, which love brain neurons but have to find their way to the brain after being injected into people by mosquitoes, influenza viruses don't have to work nearly so hard. They are taken to the location they like best simply because we need to breathe.

Once inhaled, the viruses begin attaching to lung epithelial cells. They use a kind of agglutinin (a substance that glues things to itself—its name shares a root with the English word "glue"), a hemagglutinin, to bind to what are called sialic acid linkages on the surface of airway epithelial cells. (This is one mechanism by which plants such as Chinese skullcap and ginger stop influenza infections; they are hemagglutinin inhibitors.) All viruses do this in their own way; they have an affinity for a unique receptor on the surface of specific cells and in one way or another they get to that location and those particular cells. Once there, they attach to that part of the cells. In a sense they use that part of the host cells' membrane as a docking port.

As soon as it is attached to a cell, the virus begins to alter the permeability of the cell wall, inducing alterations in the cell's cytoskeleton and initiating endocytosis. In other words, it makes the cell surface more

soft, causes the skeletal structure of the cell to bend apart, and tricks the cell into taking the virus inside it where it can't be found by the immune system. It does this by using a particular kind of enzyme, neuraminidase—which is sometimes also called a sialidase because such enzymes catalyze, or break apart, the sialic acid linkages on the host cell surface. This is why neuraminidase inhibitors (such as Tamiflu, i.e., oseltamivir) are effective in the treatment of influenza; they inhibit the ability of the virus to enter host cells. This stops the infection. (Chinese skullcap, elder, licorice, rhodiola, ginger, isatis, *Lespedeza bicolor*, *Angelica keiskei*, *Amorpha fruticosa*, quercetin, *Alpinia zerumbet*, *Erythrina addisoniae*, and *Cleistocalyx operculatus* are all neuraminidase inhibitors.) Neuraminidase inhibitors are effective against both influenza A and B strains.

During the process of endocytosis, the virus stimulates the cell to create what is called a vacuole, essentially a sealed bubble that will be held inside the cell. Cells do this to sequester substances that can damage them. Microbes have learned to use such vacuoles for their own purposes, usually to protect the virus or bacteria from intracellular antimicrobial actions.

The virus uses its hemagglutinin to bind itself to the inside of the vacuole membrane, where it opens a pore to the cell's cytoplasm, i.e., its interior spaces. To do this the virus uses what is called the M2 ion channel—ion channels are tiny pores in cells that allow charged molecules to enter and exit cells, bringing food in and allowing waste out. Using an M2 inhibitor blocks this process and literally stops the virus from replicating. (Lomatium is one of the most potent M2 inhibitors known, stronger than the pharmaceutical amantadine.) Use of the M2 channel is specific to the influenza A virus, which is why the development of blockers for it was considered crucial. Unfortunately, the extensive use of chemical M2 inhibitors such as amantadine in poultry farms has now created nearly complete resistance to them in all influenza A strains.

Once the pore is open, the virus disassembles itself and releases viral RNA and core proteins into the cytoplasm. (Chinese skullcap inhibits this kind of viral RNA release.) The core proteins and viral RNA form a complex that is taken into the nucleus of the cell, where the cell is stimulated to begin making copies of the viral RNA (each slightly different). The new viral RNA is combined with other newly manufactured virus

components such as neuraminidase and hemagglutinin and assembled into new viruses. These attach to the inside of the host cell membrane, a bulge forms in the membrane, and the new viruses are expressed (viral budding or shedding) into the extracellular matrix surrounding the cell.

The cell is taken over by the virus in this process, its own components depleted during the creation of new viruses. Once its resources are gone, the cell dies and the newly created viruses move on to new host cells, beginning the process all over again.

The alveolar epithelial cells are specific sites for this process to occur. The alveoli are tiny sacs that are the terminal end of the respiratory tree. The air we breathe travels throughout the bronchial tree, eventually emerging into the alveoli, where the oxygen transfuses across very thin membranes into the blood. This is how our bodies remain oxygenated. In the cells lining those tiny sacs the viruses breed. They cause extreme inflammation, or swelling, of the cells in that location with resulting edema (fluid accumulation). All the infected cells burst open and die as new viruses are made. So, fewer alveoli are functional. Breathing is more difficult and the infected person has much less energy because oxygen is not making it into the blood in sufficient quantities. (This is why hospitals sometimes give the infected oxygen.) Pneumonia is when this process becomes severe, the sacs filling with increasing amounts of fluid while there are fewer and fewer functional alveoli.

Throughout the cellular infection and replication process, the virus is also stimulating the release of cytokines by the cell. These cytokines make the tight junctions between cells (and the cellular membranes) more porous and allow easier movement of viral particles through the extracellular matrix (and into the cells themselves). The cytokines are also stimulated in just such a way as to keep the parts of the immune system that can kill the viruses suppressed for as long as possible.

Toll-like receptors (TLRs) are pattern recognition receptors that can identify different types of microbes. The virus particles stimulate TLR3, which begins inducing the release of nuclear factor kappa-B (NF-κB) cytokines. NF-κB is an upstream cytokine, meaning that it is a powerful initiator of other inflammatory cytokines. NF-κB *begins* very specific types of cytokine cascades. Other types of initiators such as RIG-1, NOD2, and MDA5 are also released as part of the body's reaction to a viral infection. Normally, these would strongly stimulate type 1

interferon (IFN) production (IFN-α and IFN-ß). And influenza viruses are generally very susceptible to these interferons. However, the influenza virus uses a protein, the NS1 protein, which blocks the induction of type 1 IFNs long enough to get established in the body. (Upregulating the production of type I interferons with herbs such as licorice will help reduce the severity of the infection.) The virus also inhibits dendritic cell maturation and activation, lowering the response levels of T and B cells. (Increasing T cell counts is particularly effective in reducing influenza severity. Licorice, elder, red root, and zinc are specific for this.) These cells are part of the adaptive immune response; suppressing them protects the virus from attack. The body response also stimulates the release of type III interferons, to which the virus is less susceptible and which it does nothing to suppress. These interferons have general, rather than specific, antiviral qualities and are upregulated within 3 to 6 hours of infection. This is what begins causing the general flu-like feelings that presage a full-blown flu episode. The virus itself does not make you feel "fluey."

During this same time period, the infected airway cells (tracheobronchial and alveolar epithelial cells) begin generating specific cytokines and chemokines: interleukin-1 beta (IL-1ß), IL-6, IL-18 (which causes spikes in IFN-γ production), C-C chemokine ligand 5 (CCL5, also known as RANTES, "regulated and normal T cell expressed and secreted"), C-X-C chemokine ligand 10 (CXCL10). Then, some 12 to 16 hours later, other cytokines are produced: tumor necrosis factor alpha (TNF-α), IL-8, and CCL2 (also known as monocyte chemoattractant protein-1 or MCP-1). The expressed cytokines make the epithelial structures more porous. This assists faster viral penetration of the cells. It also stimulates the migration of immune cells to the sites of infection.

Interferon-gamma (IFN-γ) is a type 2 interferon, sometimes called macrophage-activating factor. It is this IFN that is crucial in the cytokine overinflammation that occurs during severe influenza. By stimulating it, the virus initiates a positive feedback loop in the cytokine process that leads, in severe infections, to cytokine storms.

CCL2 causes the migration of blood-derived monocytes into the alveolar airspaces. TNF-α and IL-1ß upregulate adhesion molecules (which include intercellular adhesion molecule-1, a.k.a. ICAM-1, and E-selectin) on the surface of the endothelial cells that line blood vessels.

This helps the endothelial lining become more porous and stimulates the transendothelial migration of neutrophils to those locations. TNF-α induces monocyte and neutrophil movement across the epithelium through ICAM-1 and VCAM-1 (vascular cell adhesion molecule-1) upregulation. The consequence of this is increasing amounts of white-blood-cell-filled mucus in the lungs. (This is what we cough up during a flu infection.)

The size of the drainage lymph nodes in the lungs begins to increase. This helps, during a healthy resolution of infection, to drain more of the fluids from the lungs, preventing suffocation. Within those lymph nodes, areas called the geminal centers increase their size and development. The germinal centers are the sites where B lymphocytes are produced and are differentiated in order to attack the specific infection that is occurring. This is part of the adaptive humoral immune response. These lymph node locations (as well as those in peripheral tissues) can become overfull during severe infections, slowing drainage and healthy adaptive immune responses. They can also be specifically attacked and damaged so that they do not function at all. This is a contributor to the mortality that sometimes occurs during cytokine storms. (This is why herbs such as red root, inmortal, and pleurisy root are useful; they all support the lymph structures in the lungs and periphery. Red root—*Ceanothus* spp.—is particular useful in the periphery for spleen and lymph enlargement and lymph drainage; inmortal—*Asclepias asperula*—is specific for optimizing lymph drainage from the lungs; pleurisy root—*Asclepias tuberosa*—is specific for reducing inflammation in the pleurae and lungs. They can be used interchangeably to some extent.) The lymph centers in the lungs are heavily affected during influenza, much more so than the periphery.

Similarly to many viruses, while influenza viruses reproduce most efficiently in the alveolar epithelial cells, they can also infect other cells, specifically dendritic cells, monocytes, macrophages, neutrophils, T cells, B cells, and natural killer (NK) cells. In response to being infected those cells also begin releasing cytokines and chemokines: IFNs, IL-1α and IL-1ß, IL-6, TNF-α, CXCL8, CCL2 (MCP-1), CCL3 (a.k.a. macrophage inflammatory protein-1 alpha, or MIP-1α), CCL4, CXCL9, and CXCL10 through the ERK-1, ERK-2 (extracelluar-signal-regulated kinase 1 and 2), p38 MAPK (p38 mitogen-activated protein kinase), and JNK (c-Jun N-terminal kinase) pathways.

TNF-α, IL-1ß, IL-6, and IFN-γ are responsible for most of the negative effects of the cytokine cascade. Mice that are unable to produce TNF-α consistently show decreased mortality, a reduced symptom picture, and less severe course of the disease. This holds true even if they are infected with the reconstituted, and very virulent, 1918 virus. Inhibition of TNF-α (especially) and IL-1ß has been found to significantly reduce the cytokine-based inflammation that occurs during influenza, alleviating symptoms and inhibiting viral spread. (Herbs specific for inhibiting TNF-α are kudzu, Chinese senega root, Chinese skullcap, elder, ginger, houttuynia, licorice, boneset, and cordyceps. Herbs specific for inhibiting IL-1ß are Japanese knotweed, Chinese senega root, Chinese skullcap, cordyceps, kudzu, and boneset.)

The virus can also inhibit the production of macrophages over time. This occurs because, over time, macrophages will begin producing anti-inflammatory cytokines such as IL-4 and IL-10. Once the bodily system is macrophage-depleted a prolonged inflammatory process occurs, keeping the infection going. Lung levels of IL-1ß, IL-6, and TNF-α all increase considerably at that point. Stimulating monocyte and dendritic cell maturation (cordyceps) and inducing IL-4 and IL-10 (Chinese skullcap, elder, houttuynia, licorice, cordyceps) will help counteract this.

The virus is exceptionally sophisticated in its impacts. There are three stages of chemokine stimulation. The first, 2 to 4 hours postinfection, is attended by the production of CXCL16, CXCL1, CXCL2, and CXCL3. These chemokines are specific for attracting neutrophils, cytotoxic T cells, and NK cells. At 8 to 12 hours postinfection CXCL8, CCL3, CCL4, CCL5, CXCL9, CXCL10, and CXCL11 are being produced, which attract effector memory T cells. At 24 to 48 hours postinfection, when dendritic cells are most present in the lymphoid tissues, the chemokine profile changes again in such a manner as to attract naive T and B cells. The effect of all this is the virus playing the immune system as a virtuoso plays a violin. Eventually the immune system catches up (usually) and the infection is stopped as influenza-specific antibodies are created.

Plants that reduce the other main cytokines that the virus stimulates will also help lessen disease severity and prevent lung damage. I think the most important are inhibitors of NF-κB (Chinese senega root, Chinese skullcap, ginger, houttuynia, kudzu, licorice, boneset, astragalus), IL-6 (kudzu, Chinese skullcap, isatis), IL-8 (cordyceps, isatis, Japanese

knotweed), RANTES (licorice, isatis), MCP-1 (houttuynia), CXCL10 (boneset), CCL2 (boneset), the ERK pathway (kudzu, Chinese skullcap, cordyceps), the p38 pathway (Chinese skullcap, houttuynia, cordyceps), and the JNK pathway (Chinese skullcap, cordyceps, lion's mane). The reduction of these cytokines and pathways will reduce IFN-γ.

Each type of influenza has a slightly different cytokine profile with slightly different cytokines more strongly represented. However, the protocols herein, directed to this form of cytokine profile, will be specific enough for every strain, including the low pathogenic avian strain H9N2, which strongly upregulates transforming growth factor beta 2 (TGF-ß2), a different dynamic entirely. Medicinal plants already in use in the developed protocol are, however, specific for TGF-ß2, i.e., astragalus (the strongest) and Chinese skullcap. *Magnolia officinalis, Ginkgo biloba, Folium syringae, Nigella sativa, Paeonia lactiflora,* and *Lonicera japonica* are other plants specific for inhibiting TGF-ß2. (This is why lonicera, or Japanese honeysuckle, is commonly used in the treatment of respiratory infections in China—it alleviates wind heat and expels wind heat invasion. In other words, it reduces inflammation in the lungs and expels the virus or bacteria responsible.)

Normally, influenza viruses stay in the upper respiratory tract. However, during more severe infections they will infect the lower respiratory tract as well. Pneumonia is one serious complication from that. So are cytokine storms, should the disease really take hold.

Cytokine Storms

The more serious pandemic viruses (1918 H1N1, 2009 H1N1, and 2004 H5N1) cause severe pulmonary injury and inflammation. In these cases, the cytokine cascades become storms and the death rate correspondingly climbs. H5N1, for example, has around a 60 percent death rate in those who are infected, usually from acute respiratory distress and organ failure. The 1918 rate had a much lower mortality rate, around 20 percent, but the strain is much more infective, reaching about one-third of the population. (The emergence of a highly infective avian H5N1 strain is one of the things that keeps viral researchers up at night.)

While there is (usually) not a corresponding increase in viral replication during a viral storm, the cytokine increases in severe pandemic

influenzas are significant and this is where the mortal damage comes from. IFN-γ production is usually increased, as is the expression of TNF-α, IL-1ß, CXCL10, RANTES, MIP-1α, MCP-1, MCP-3, and IL-6. The IFN-γ levels and the virus synergistically interact to significantly increase CXCL10 in airway epithelial cells. This causes a tremendous infiltration of immune cells into the airways. Blocking IFN-γ through the use of inhibitors has been found to significantly reduce airway infiltrates (houttuynia, cordyceps, Chinese skullcap, and licorice; note that licorice is an IFN-γ *modulator*—it inhibits its production when levels are high and stimulates its production, especially in T cells, when levels are low).

In particular, the inhibition of TNF-α, IFN-γ, IL-1ß, and IL-6 is crucial during infection with severe influenza pandemic strains. Those cytokines are found in exceptionally high levels in such instances and damage to the lungs is specific to them. If their levels rise high enough, the inflammation does not stay confined to the respiratory system but goes systemic. This kind of condition is called sepsis, essentially a whole-body inflammatory state. If severe enough it can lead to organ failure and cardiac arrest.

A particular cytokine-like protein has been implicated in sepsis-induced cytokine storms: high-mobility group box 1 protein (HMGB1). This cytokine-like protein is highly elevated in all patients who die from sepsis, including sepsis generated by influenza. HMGB1 is also unique in that once stimulated, its secretion continues for a very long time. TNF-α, in comparison, lasts at peak levels for about 90 minutes once stimulated. HMGB1 peak levels last 18 hours before they begin to decline. Once HMGB1 is released it stimulates further cytokine releases *and* has the additional property of being synergistic with the other cytokines already present in the body, amplifying their effects. HMGB1 release is stimulated by macrophages and monocytes when a particularly potent cytokine cascade begins, specifically with high levels of NF-κB, TNF-α, RANTES, IL-6, and IFN-γ, in pretty much that order. The amount released is directly dose-dependent. In other words, the higher the cytokine levels, the more HMGB1 is released. And the more that is released, the higher the cytokine levels go. As examples, levels of IL-6 are nearly four times higher, IL-8 nearly three times higher, and IFN-γ more than two times higher during severe infections than in

milder cases. Higher IL-6 concentrations are positively correlated with prolonged illness and hospitalization. As the storm progresses levels of IL-8, MCP-1, and H_2O_2-myeloperoxidase also significantly increase. Endothelial cells are strongly stimulated and begin to amplify the storm's cytokines. Hyperactivation of p38 MAPK with an accompanying inhibition of the adaptive immune system is a marker for these kinds of cytokine storms.

HMGB1 is also released when the nuclei of cells are damaged, as they are during influenza infections. HMG proteins are held in the nucleus to help in forming DNA complexes and regulating gene expression. When HMGB1 is expressed in lung tissue, as it is during severe influenza episodes, it causes massive neutrophil infiltration into the lungs and acute lung injury. As the storm progresses respiratory failure (requiring mechanical ventilation), acute renal failure, and systemic shock all occur. In severe cases such as these, antivirals (oseltamivir), antibiotics, and corticosteroids have all been found to be ineffective.

Common steroidal drugs (e.g., dexamethasone and cortisone) have consistently been found to have no effect on HMGB1 levels, and the same can be said for NSAIDs such as aspirin, ibuprofen, and indomethacin—even at superpharmacological concentrations. However, a number of herbs and herbal constituents do have direct suppressive actions against the protein.

Direct inhibition of HMGB1 with herbs such as *Angelica sinensis* and *Salvia miltiorrhiza* protects mice both before and 24 hours after infection with normally lethal influenza viruses. The licorice constituent glycyrrhizin directly binds HMGB1, inactivating its actions in the body. The green tea component epigallocatechin gallate (EGCG) also inhibits HMGB1, as does quercetin. Counterintuitively, nicotine also significantly lowers HMGB1 in the lungs. The pharmaceutical minocycline has also shown the ability to reduce HMGB1 levels; its use should be explored in hospital and pharmaceutical settings.

During severe influenza infections reducing HMGB1 is essential.

Lung and Tissue Pathology during Severe Influenza Infections

Influenza viruses specifically invade lung tissues and cause both direct and inflammatory-mediated damage. There are four primary pathological changes that occur: 1) diffuse alveolar damage; 2) necrotizing bronchiolitis; 3) intense alveolar hemorrhage; and 4) severe fluid accumulation.

The viruses infect specific cellular structures, in fact any that possess linked sialic acids (alpha-2,6 and alpha-2,3) on their surface membranes. But cells in the respiratory system express those acids differently and different influenzal strains create different infection profiles. The nonciliated cells of the lungs contain a higher proportion of alpha-2,6-linked sialic acids while ciliated cells contain both alpha-2,6- and alpha-2,3-linked sialic acid. H3N2 viruses prefer the nonciliated-cell sialic acids while the avian flu types (H5N1) exclusively infect ciliated cells. This is part of the reason that the avian strains tend to be more deadly. The cilia, when infected, are often killed and their ability to move mucus up and out of the lungs destroyed. This substantially increases mucus buildup in the lungs. The H5N1 strains prefer the alpha-2,3-linked sialic acids that are most strongly present on the ciliated cells but those acids exist on ciliated cells in higher quantities in the lower respiratory tract. So, the H5N1 strains infect not only the cilia but also the lower respiratory tract, causing a much deeper infection.

In severe cases, irrespective of strain, alveolar hemorrhage is often present, as is intra-alveolar edema and interstitial inflammation. The tissues surrounding blood vessels and lymph nodes and channels all inflame (perivasculitis). Microthrombi or tiny blood clots occur throughout the blood vessels in the lungs. IFN-γ levels are high in macrophages, alveolar epithelial cells, and vessels. TNF-α levels are high in alveolar macrophages and bronchial and vascular smooth muscle. There are massive infiltrates surrounding airways and in the alveolar walls. The spleen typically atrophies and presents with nonreactive white pulp. In the lymph nodes nonreactive follicles and sinusoidal erythrophagocytosis are common.

Protecting spleen and lymph structures and their function, cilial structures, and mucous membrane structures is essential.

Medical Interventions

If influenza is pharmaceutically treated, neuraminidase inhibitors such as oseltamivir (Tamiflu) or zanamivir (Relenza) are usually used. Sometimes adamantanes (amantadine and rimantadine) are as well; they inhibit the M2 ion channels. These pharmaceuticals are commonly referred to as antivirals but they are not, at least not in the same way that an antibiotic is an antibiotic, that is, something that specifically kills bacteria. They, more accurately, inhibit viral penetration of host cells (thus stopping or slowing the infection) or prevent the vacuole-enveloped virus from releasing viral proteins into the host cell interior (thus stopping or slowing the infection). They don't directly kill the virus. Ribavirin, a drug that interferes with RNA metabolism, is sometimes used but its effects are mixed and it has many serious side effects.

If there is significant inflammation, corticosteriods may be used to try and reduce it—but if HMGB1 levels are in play, corticosteroids will do nothing to reduce them. Hospitalization is common in severe cases, but other than passive care and the use of oxygen, little can be done. Intravenous liquids may be given but they may have serious side effects, since the main approach has been the use of a combination nutrient solution/glucose IV in an attempt to keep the patient's nutrient/energy levels high. Unfortunately, it turns out that the use of glucose during influenza infections significantly increases viral load and illness parameters. Insulin, on the other hand, reduces them considerably and also has the added benefit of lowering HMGB1 levels.

There is the bare beginnings within hospital settings of the use of cytokine inhibitors such as minocycline and HMGB1 inhibitors (e.g., anti-IFN-γ antibodies, intravenous immunoglobulin) but their use is not widespread. If microthrombi proliferate in the lungs then anticoagulants may be used. Interestingly, both antithrombin III and thrombomodulin decrease HMGB1 in vitro. Very few of these HMGB1 interventions are commonly used, or known of, by practicing physicians.

To make matters worse, many influenza strains are developing resistance to the primary neuraminidase inhibitor used to treat them, oseltamivir, as well as to the primary adamantane M2 ion channel inhibitor, amantadine. Influenza virus samples from the 2007–2008 season showed 0.06 percent resistance, from the 2008–2009 season

1.5 percent resistance, and from the 2009–2010 season 28 percent resistance—a normal exponential learning curve for resistance. Research in late 2009 began finding strains resistant to the other major neuraminidase inhibitor, zanamivir. Resistance has become common as well to the other primary M2 ion channel inhibitor, rimantadine. Some areas report 100 percent resistance to amantadine and over 90 percent resistance to oseltamivir. Besides their overuse in agribusiness, there is another reason for resistance: human excretion.

Oseltamivir is immediately metabolized in the body to oseltamivir carboxylate. It is only active in this form. Unfortunately this form *is* the metabolized form and it is excreted out of the human body without any further alterations. It flows unaffected through wastewater treatment plants and ends up in waterways in low doses, where it comes into contact with waterfowl and thus is exposed to avian influenza strains. The avian strains develop resistance, and as the avian, human, and swine strains commingle the resistance is passed on into strains that can infect humans.

These drugs are also often used in large quantities during epidemic outbreaks. And the viruses quickly develop resistance to them. About 30 percent of those treated will develop resistant strains and will shed them for days afterward. The newly infected are then resistant to the drugs.

Less severe cases of the flu, if one sees a physician, are rarely treated (though, irresponsibly, some physicians will prescribe antibiotics, which are not active against viruses, for the flu). The usual medical advice is to "rest in bed, drink plenty of fluids, and take over-the-counter medications as needed." In other words, it is left up to the individual's immune system and some very limited self-care options to treat the infection. The Chinese don't have this kind of technological bias in place. Unlike those of us in the West, they have been developing both herb-alone and herb/pharmaceutical combination approaches in their treatment protocols. And their outcomes are very good when compared to Western approaches.

There are a great many interventions that are possible with plant medicines and unlike pharmaceuticals, viruses don't develop resistance to them.

Natural Treatment Protocols for Influenza

Again, just to emphasize this: *there are thousands of combinations of plant medicines that can be created to treat respiratory infections.* These are just the ones I have found useful. Please feel free to experiment, combine, innovate, and find your own unique combinations. There is no one right way to the truth.

An influenza infection can run the range from extremely mild to extremely severe. I break the disease down into four types, each needing a different approach: 1) early onset; 2) mild infection; 3) moderate infection; and 4) severe infection. I will go into some of the unique aspects of treating severe infections at the end of this section.

Early-Onset Treatment

I have found two approaches that can short-circuit a developing episode before it gets a good hold in the body: oscillococcinum and an herbal tincture combination.

OSCILLOCOCCINUM

I have found this homeopathic remedy to be extremely good for stopping the development of the flu *if you take it at the first signs of the flu,* that is, the *moment* you feel that first tingling sensation in your body that tells you that you are about to get sick.

Oscillococcinum comes as little sugar granules in tiny tubes. Take one tube every 6 hours, three per day, for 2 or 3 days in a row. This is often enough to stop the infection.

HERBAL TINCTURE COMBINATION

For many years I used a particular tincture combination: *Echinacea angustifolia* (now I use fresh ginger juice tea—see facing page; and note: *E. purpurea* is useless for this; it won't work), red root, and licorice, in equal parts. The dosage is a full dropperful of the tincture (30 drops) *every hour*, every day, until the symptoms resolve themselves.

I have found this useful for stopping the development of a flu infection *if you take it at the first signs of tingling or soreness in your throat.* The

tincture mix should be held in your mouth, liberally mixed with saliva, then swallowed, *slowly*, letting it dribble down the back of the throat.

For *Echinacea angustifolia* to work for a cold or flu *the herbal tincture must touch the affected membranes.* Echinacea *is* antiviral; it's been found active against HIV and influenza H5N1, H7N7, and H1N1 (swine origin). However, in order to inactivate the influenza strains, it needs *direct contact* with the affected cells just prior to or right at the moment of infection. Echinacea inhibits the receptor cell binding activity of the virus, interfering with its entry into the cells while at the same time strengthening the protective power of the mucous membranes through hyaluronidase inhibition. In essence, it strengthens the cellular bonds in the mucous membranes and makes it harder for a virus to penetrate. If the virus does penetrates deeper into the body, the herb just won't work because direct contact is not possible.

Goldenseal has some similar actions on mucous membranes, which is why the deplorable echinacea/goldenseal combinations are so common. They are only effective at the first signs of infection. (I reiterate: They are *only* effective at the first signs of infection. If the infection is full-blown, you are just wasting your money.) Again, *E. purpurea* (in the form in use in most of the West) will not work. The Germans use *only* the fresh, stabilized juice of the stalks, not the root, and it is the root that nearly every American herbalist and company use in their products. (Capsules, of any species, are completely useless for viral and bacterial infections.)

Mild Infection

If you do get sick but have a relatively mild case developing, then the following protocol, composed of two parts, will usually get rid of it.

FRESH GINGER JUICE TEA

Ginger is useful for the flu *only* if the juice of the fresh root is used. Dried ginger is useless.

At the first signs of an infection that is not going to stop, juice 1 to 2 pounds of ginger. (Squeeze the remaining pulp to get all the juice out of it, and keep any leftover juice refrigerated.) Pour 1 to 2 ounces of the juice into a mug, and add one-quarter of a lime (squozen), a large

tablespoon of honey, ⅛ teaspoon of cayenne, and 8 to 10 ounces of hot water. Stir well. Drink 2 to 6 cups daily.

This will usually end the infection within a few days. *If it does not* it is still tremendously useful as it will thin the mucus, slow the spread of the virus in the body, and help protect mucous membranes from damage.

Comment: Some people find that an elderberry syrup will provide the same effects.

HERBAL TINCTURE COMBINATION

Tincture combination of 2 parts lomatium, 2 parts red root, 2 parts licorice, and 1 part isatis (e.g., 2 ounces of each of the first three, 1 ounce of the latter). Dosage: 30–60 drops each hour until the condition improves.

Moderate and Severe Infections

I treat moderate and severe influenza infections similarly, though with severe infections there needs to be a great deal of focus and persistence. The doses often need to be higher as well and additional formulations used as symptoms develop.

The primary interventions are:

- Direct antivirals that will inhibit viral penetration of host cells and replication. (The primary antiviral herbs for this are Chinese skullcap, isatis, licorice, houttuynia, lomatium, cordyceps, astragalus, rhodiola, boneset, elder, *Strobilanthes cusia*, *Forsythia suspensa*, and *Sophora flavescens*.)
- Reducing cytokine levels, thus inhibiting damage in tissues.
- Thinning the mucus and promoting fluid drainage from the lungs.
- Repair of damaged tissues.
- Normalization of immune responses.
- If sepsis is a potential problem, large quantities of HMGB1 inhibitors should be used.

Treatment of moderate to severe influenza is composed of three main formulations, to which others can be added if necessary. These are an antiviral tincture formulation, an antiviral ginger juice tea, and an immune complex tincture formulation.

ANTIVIRAL TINCTURE FORMULATION

Equal parts of Chinese skullcap, isatis, licorice, houttuynia, lomatium, red root, yerba santa (*Eriodictyon* spp.), elephant tree (*Bursera microphylla*), osha (*Ligusticum porteri*), and either inmortal (*Asclepias asperula*) or pleurisy root (*Asclepias tuberosa*).

This formulation contains potent antivirals, specifically Chinese skullcap, isatis, licorice, houttuynia, lomatium. These are designed to kill the virus and inhibit its entry into the body. And of course many of them have alternate actions as well. Licorice, for example, is mucoprotective, strongly anti-inflammatory, and expectorant. Chinese skullcap is potently anti-inflammatory for the cytokine cascades that influenza creates, provides splenic protection and activation, will help lower fevers, and is an expectorant. All of these antiviral herbs have multiple functions in respiratory diseases.

The four herbs added to this formulation that are not discussed in depth in this book (yerba santa, osha, elephant tree, and inmortal or pleurisy root) do not have to be included, though they do help considerably, primarily through helping with the tastiness of the formulation, thinning the mucus, stimulating expectoration, and promoting lymph drainage from the lungs.

Both yerba santa and osha are added for taste as well as their medicinal actions (isatis really does taste foul to me). Osha is a relative of lomatium and has its own antiviral and expectorant actions. It has strong impacts on inflammation in the lungs and increases the degree of oxygen intake during respiration. It also has the added benefit of anesthesizing the throat tissues, helping reduce throat soreness. Yerba santa is a very good expectorant, bronchial dilator, and decongestant. Elephant tree is anti-inflammatory, thins and softens bronchial mucus, and stimulates expectoration. It is a major source of copal and a close relative of myrrh. (Myrrh can be substituted for elephant tree in this formulation if the tincture is stabilized with 20 percent glycerin.) I consider all three of these herbs to be specific for maintaining the mucous membranes of the lungs, thinning the mucus, and increasing expectoration. Inmortal (or, as an alternative, pleurisy root) improves cilia function and is a bronchial dilator, an expectorant, a febrifuge (lowering fevers), and most especially a potent medicinal for stimulating lymph drainage from the lungs.

Dosage needs to be high for two reasons. The first is that there are so many herbs in the formulation that each herb has a reduced presence in the formulation. The second is the nature of moderate to severe influenza infections. As the disease progresses up the scale of severity, the cytokine cascade increases in intensity. The body needs to be *bathed* in the plant compounds in high enough quantities that the cytokine cascade is potently inhibited. In addition, the body needs to be suffused

Other Anti-Influenza Herbs and Supplements

A number of other plants have been found effective for influenza during in vitro, in vivo, or human studies:

Achillea millefolium (yarrow)
Aegle marmelos (bael)
Agathosma betulina
Agrimonia pilosa
Allium oreoprasum
Allium sativum (garlic)
Alpinia officinarum
Andrographis paniculata
Androsace strigilosa
Angelica keiskei
Aronia melanocarpa
Asparagus filicinus
Azadirachta indica (neem)
Bergenia ligulata
Camellia sinensis (green tea, EGCG)
Cephalotaxus harringtonia
Chaenomeles sinensis
Cistus incanus
Clinacanthus siamensis
Cocos nucifera (coconut oil, monolaurin)
Commelina communis
Eleutherococcus senticosus
Elsholtzia rugulosa
Geranium sanguineum
Ginkgo biloba
Holoptelea integrifolia
Hypericum japonicum
Justicia pectoralis
Myrica rubra
Narcissus tazetta
Nerium indicum
Ocimum sanctum (holy basil)
Olea europaea (olive)
Panax spp. (ginseng)
Pandanus amaryllifolius
Phyllanthus emblica
Propolis
Prunus mume
Punica granatum (pomegranate)
Rhinacanthus nasutus
Sanicula europaea
Saponaria officinalis
Schefflera heptaphylla
Terminalia chebula
Thalictrum simplex
Tinospora cordifolia
Toddalia asiatica
Trachyspermum ammi
Tussilago farfara (coltsfoot)
Uncaria rhynchophylla
Verbascum thapsus (mullein)

with enough of the antiviral compounds that the viral entry into host cells and its presence in the body are severely curtailed.

For moderate influenza: 60 drops or 3 ml (a little over ½ teaspoon) every hour.

For severe influenza: 1–2 teaspoons every hour.

Dividing the formulation: You can if you wish divide the formulation in two. The first would contain Chinese skullcap, isatis, licorice, houttuynia, and lomatium and would be primarily an antiviral formulation (and would taste from okay to bad). The second would contain red root, yerba santa (*Eriodictyon* spp.), elephant tree (*Bursera microphylla*), and either inmortal (*Asclepias asperula*) or pleurisy root (*Asclepias tuberosa*) and would taste very good. (I would skip the osha if the formulation is split in two.) This second formulation would primarily be for lymph and spleen optimization and protection, expectorant and decongestant actions, mucus thinning, cilia protection, and lymph drainage from the lungs. The dosage for each would be half the dosage as when combined.

FRESH GINGER JUICE TEA

This is the same as discussed earlier, in essence: ginger juice tea, hot. Again, ginger is useful for the flu *only* if the juice of the fresh root is used. Dried ginger is useless. Prepare the tea as directed on page 39. Drink 4 to 6 cups daily.

Ginger in this form is potently antiviral for influenza. The fresh juice tea will also thin the mucus, help protect mucous membranes from damage, *and* act as a potent diaphoretic, lowering fever during the infection.

IMMUNE COMPLEX TINCTURE FORMULATION

Equal parts of the tinctures of astragalus, cordyceps, and rhodiola.
All of these herbs are active against influenza viruses. They are also potently adaptogenic, that is, they increase the resistance of organisms to stressors, whether microbial or external. Additionally, astragalus and cordyceps are highly specific for the cytokine cascades that are initiated by influenza. These herbs will help through their antiviral actions, modulate the overactive immune response, lower cytokine levels, and enhance a healthy immune response to the infection.

Again, dosage levels should be highish, for the same reasons as outlined above.

For moderate influenza: ½ teaspoon of the tincture 3x daily.

For severe influenza: 1–2 teaspoons of the tincture 6x daily.

Supportive Additions

There are a few additional things that can be very helpful during acute influenza episodes. These are treatments for high fever, severe headache, cough, high HMGB1 levels during cytokine storms, and protecting cilial structures and mucous membranes in the lungs. Two supplements have also been found to be helpful (in a number of studies). And essential oil inhalants can help with the infection in the lungs, coughing, and mucus flow and secretion.

For Fever

There are a number of interventions that can help. Fresh ginger juice tea (see page 39) can often lower the high fevers that occur during influenza, but if you want more:

- **Boneset tea:** Boneset is specific for influenza and a number of the cytokines it stimulates. It is also highly specific for diseases that alternate fevers and chill episodes. If I am going to use boneset, and I am already ill, it is easier to make up a lot at one time. Getting out of bed over and over again is too difficult. So . . . add 3 ounces of dried boneset herb to 1 gallon of hot water, let steep 30 minutes, then drink 8 ounces every few hours. *The tea must be consumed hot for it to be effective for fever.* The herb will stimulate sweating, thus lowering the fever. It will help interrupt the chill/fever, chill/fever cycles. It's bitter, so add honey.
- **Pasque flower (*Pulsatilla patens*) tincture:** 10 drops each hour as needed.
- **Any diaphoretic (causes sweating) tea:** Peppermint is a good choice, children like it, and it will also help calm the stomach. Yarrow is more bitter but is also good for this. Both of them do have some antiviral activity against influenza.

- **Wet cloth:** A wet washcloth, applied regularly over the entire body, will mimic the action of sweat in helping lower a fever. The higher the fever, the more often you have to do it.

For Headache

- **Indian pipe (*Monotropa uniflora*) tincture:** The best I have found for the kinds of recalcitrant headaches that can sometimes occur during the flu. Dosage is from 30 drops to 1 teaspoon every few hours.
- **Coral root (*Corallorhiza* spp.) tincture:** Up to 1 teaspoon every few hours. It does help and for some people it is specific.
- **Tinctures of motherwort (*Leonurus cardiaca*) and American wood betony (*Pedicularis* spp.):** Combined, equal parts. Dosage is up to 1 ounce (yes, that's right) of the combination at a time, in water. I usually use ¼ ounce but the high dosage can help occasionally when nothing else does. Go slow and work up, every 4 hours or so.

Complex Formulations

For nearly 30 years I tended to use formulations that contained only three herbs, occasionally five. With the emergence of more intense forms of influenza, and my increasing age, I have found that a more complex formulation works better. I do think a major factor in that is aging. There are, in myself and in many of the people I help, considerable age-related alterations in our physiology. There are preexisting inflammations in many parts of our bodies, from age-related memory dysfunction to arthritis. Our bodies are wearing out, biodegrading, and that deterioration makes them more susceptible to infections such as influenza, and in more severe forms. Further, our immune systems are not as vital as they once were and have a great deal more trouble counteracting the infection.

For Cough

I make a cough syrup every fall just before the flu season. It comes in handy. The recipe varies all the time, depending on what I have on hand and what I have wild-harvested in any particular year. But the recipe below gives you a good idea of what kinds of herbs are in it. I do keep it refrigerated though it will last awhile if it is not.

Cough Syrup Recipe

INGREDIENTS

- 3 ounces horehound
- 2 ounces cherry bark
- 2 ounces elderberries
- 2 ounces elecampane
- 2 ounces licorice
- 2 ounces mallow (or marshmallow)
- 1 ounce Russian or slippery elm bark
- 1 ounce vervain
- 1 ounce lomatium (or osha)
- 7 pints water
- 3 ounces glycerin
- Wildflower honey
- 2 ounces mullein tincture
- 1 ounce yerba santa tincture

To make:

Combine the horehound, cherry bark, elderberries, elecampane, licorice, mallow, elm bark, vervain, and half of the lomatium with the 7 pints of water in a large pot. Bring to a boil. Stir frequently as it heats to prevent sticking. Once it boils, reduce the heat and let simmer, stirring constantly. Cook until the liquid is reduced by half. Remove from the heat and let cool. (You can put the pot in a bath of cold water to cool it faster. Don't let it tip over.) Strain the liquid, pressing the marc (the spent plant matter) through a cloth to get as much liquid as you can.

(With mucilaginous herbs—the licorice, mallow, and elm bark—as part of the mix, it can be hard for the liquid to pass through the weave of the cloth you are using. So, alternatively, you can keep the mucilaginous herbs out of the mix and once the marc is pressed, heat all the liquid again, adding the licorice, mallow, and elm to the pot in a muslin bag to keep them out of the liquid. Bring to a boil and simmer, stirring constantly, for 30 minutes. Remove the bag, let it cool, then squeeze out the liquid as best you can.)

Warm the liquid again, just enough to dissolve the honey and glycerin. Add the glycerin, then the honey to taste. Grind the remaining lomatium (or osha) to a fine powder—a nut or coffee grinder or mortar and pestle

is good for this—then add it to the liquid. Let the mix cool, then add the mullein and yerba santa tincture. (Keep in mind that you can substitute similar herbs for any used in this recipe.)

The honey, glycerin, and two tinctures help stabilize the syrup, keeping it from going bad. I do keep the whole thing in the refrigerator though. It will last a year very easily. Generally, it is best to make this kind of a syrup in the fall, after the berries are ripe and ready for harvest, and just before flu season. It is very effective.

To use:

I keep this cough syrup by the bed and take as desired. Really, none of that 1-tablespoon-at-a-time stuff—that won't help at all. Just drink it as needed, right out of the bottle. It will help soothe the mucous membranes, reduce coughing, and ease the aches and pains that come with the flu.

To Reduce HMGB1 Levels during Cytokine Storms

The herbs that are already being used will help this considerably. However, if the condition significantly worsens then the following specific intervention is warranted. Take both formulations.

Formulation 1: Tincture combination of *Angelica sinensis* and *Salvia miltiorrhiza,* in equal parts. Dosage: 1 tablespoon every hour. And . . .

Formulation 2: Strong infusion of the two herbs, 4 ounces of each in 1 gallon of just-boiled water. Remove from the heat, let sit 4 hours, and strain. Dosage: Drink 12 ounces every hour.

To Protect Cilial Structures and Lung Mucosa

There are a number of herbs that are specific for protecting the cilia: cordyceps, olive oil and leaf, the berberine plants, and, my favorite, *Bidens pilosa*. Bidens is a very strong systemic antibiotic that is used in Asia and Africa for systemic bacterial infections (including respiratory) *and* influenza (though it has not been tested against that virus). If the mucous membranes have been infected by a microbe and you start to get well, relapse, start to get well, relapse, this is the herb to use. The herb is specific for healing and protecting mucous membrane structures, including the cilia. Tincture of the fresh herb should be used. The dry herb is not as antimicrobial though it will still help the mucous membranes' tone. Dosage: ¼–½ teaspoon up to 6x daily.

Supplements

Zinc and selenium are very helpful during influenza infections. Both have been found to protect mice from severe influenzal strains. Dosage: 200 mcg daily of selenium; 25–40 mg daily of zinc.

Essential Oil Inhalants

Essential oils of thyme, eucalyptus, rosemary, and sage can all help. They are all antiviral for influenza (to varying extents), will help reduce the coughing reflex, thin and help expectorate mucus, and improve airflow in the bronchial tract. To use: Bring a gallon of water to a boil in a pot on the stove. Turn off the heat, add 20 drops of each of the essential oils to the pot, and bring the pot to a comfortable location where you can sit with your head over it. Hold your head over the pot and breathe in the steam for as long as you can take it, every few hours.

A Few Other Respiratory Viral Infections

The main ones that people encounter are the adenoviruses, parainfluenza viruses, respiratory syncytial virus, and rhinoviruses.

Adenoviruses

Adenovirus infections tend to be mild and are generally easy to treat. However, acute conditions such as pharyngoconjunctival fever, acute respiratory disease, pneumonia, and meningitis can also occur.

Adenovirus 14 is an emerging serotype that can cause serious infection, essentially acute respiratory disease, which can sometimes lead to death. Conjunctivitis, high fever, pneumonia, and gastrointestinal involvement can all occur. The virus sheds in both respiratory droplets and feces and can remain highly infective in feces for long periods.

The herbs specific for adenovirus infections are astragalus, Chinese skullcap, elder, isatis, and licorice. Other herbs that are active are *Ardisia squamulosa, Artemisia princeps, Boussingaultia gracilis, Caesalpinia pulcherrima, Ocimum basilicum,* and *Serissa japonica*.

Treatment: The same as for mild influenza. If it becomes serious, the same as for moderate to severe influenza.

Parainfluenza Viruses

Parainfluenza viruses generally cause what is called croup. It is an acute infection of the upper respiratory tract accompanied by barking cough (the croup part) and hoarseness. The throat is often swollen, which can interfere with breathing. The herbs specific for parainfluenza are Chinese skullcap, elder, and licorice. *Allium sativum* and *Cicer arietinum* have also been found active.

Treatment: Tincture combination of elderberry, Chinese skullcap, and licorice tinctures, in equal parts. Dosage: 30 drops every hour.

Respiratory Syncytial Virus

Respiratory syncytial virus is also a single-strand, enveloped RNA virus with high variation in its genome. It is a very common infection, especially in young children, throughout the world. It causes bronchiolitis and other types of respiratory infections, especially in the lower respiratory tract. It generally presents as a common cold but can sometimes become serious, turning into pneumonia if left untreated.

The herbs specific for respiratory syncytial virus infections are Chinese skullcap, *Eleutherococcus senticosus*, elder, isatis, licorice, and *Sophora flavescens*. Other herbs found active are *Barleria prionitis*, *Blumea laciniata*, *Elephantopus scaber*, *Laggera pterodonta*, *Markhamia lutea*, *Mussaenda pubescens*, *Narcissus tazetta*, *Selaginella sinensis*, *Scutellaria indica*, and *Schefflera octophylla* (in vitro).

Treatment: The same as for mild influenza. If it becomes serious, the same as for moderate to severe influenza.

Rhinoviruses

These viruses cause the common cold. The herbs/supplements specific for rhinovirus infections are ginger, *Echinacea angustifolia*, elder, *Eleutherococcus senticosus*, quercetin, *Papaver pseudocanescens*, and *Raoulia australis*. A Japanese traditional formulation, hochu-ekki-to, has been found highly effective, as has *Prunus mume*.

Treatment: I have found the use of an *E. angustifolia*, licorice, and red root tincture combination (see page 38) *and* the prolific use of fresh ginger juice tea (see page 39) to be very effective.

Some Comments on Treating Severe Influenza

When people become severely ill with influenza, they often present with high fever, extreme lethargy, and significantly reduced vital energy. They are usually bedridden and very, very afraid.

The interventions in such cases need to be highly focused and attentive. It takes a lot of work. These patients need to be nurtured continuously, fed if they will eat (chicken broth is very good), helped to the bathroom (if they can even get out of bed), and ministered to. The fever will often need to be brought down and you will have to monitor the plant medicine intake. It needs to be constant (every hour at minimum), and in fairly high dosages in order to lower the cytokine cascade, reduce the viral load, and get the immune system back online. It will often take a week to begin to turn the situation around, and several more weeks before the person really begins to get well. It can be done. Of all the herbs useful for this, lomatium, licorice, Chinese skullcap, and cordyceps are the most essential.

3

COVID-19 AND THE CORONAVIRUSES

The illness went on and on. The symptoms changed, it was like an advent calendar, every day there was a surprise, something new. A muggy head; acutely painful calf; upset stomach; tinnitus; pins and needles; aching all over; breathlessness; dizziness; arthritis in my hands; weird sensation in the skin with synthetic materials. Gentle exercise or walking made me worse—I would feel absolutely dreadful the next day.

—Paul Garner, professor of infectious diseases, Liverpool School of Tropical Medicine

It's like nothing I've ever seen before. **—Nick Caputo, MD**

COVID is here to stay. **—Thomas Frieden, MD, former head of the CDC**

No one knows how many coronaviruses there are, perhaps hundreds, perhaps thousands. Only seven (at this time) are known to infect people. Four of those (such as one form of the common cold) usually cause only mild to moderate infections. Three are far more serious. They are all members of the SARS group: the original SARS coronavirus (an acronym for Sudden Acute Respiratory Syndrome coronavirus, a.k.a. SARS-CoV, or SARS-CoV-1), emerged in November of 2002; MERS coronavirus (an acronym for Middle East Respiratory Syndrome coronavirus, a.k.a. MERS-CoV), which was identified in early fall of 2012 (and which is spread mostly by camels); and SARS-CoV-2 (a.k.a. Covid-19), which emerged in late fall or early winter of 2019.

SARS-CoV-1 is, in its impacts in the body, very similar to acute influenza and at first was thought to be an emerging influenzal strain. The

disease was characterized by fever followed by respiratory symptoms and, ultimately for some of the infected, progressive respiratory failure leading to death. Many of those who recovered from the acute phase suffered long-term physical damage from the pathogen's effects on, for instance, the lungs, liver, and kidneys. The same was found to be true of MERS. Eventually researchers found that SARS-CoV-1 was in fact a coronavirus and that it had jumped species . . . into us. Later the same was found to be true of MERS.

SARS-CoV-1 spread to 26 countries within a few months of emergence, carried primarily by travelers who were infected. But for some reason (there are lots of guesses) it disappeared sometime in 2004. MERS-CoV is still around but has remained pretty much limited to the Middle East. But SARS-CoV-2 has neither disappeared nor limited its range. On March 11, 2020, the World Health Organization declared it a global pandemic.

SARS-CoV-2

I am writing this in March of 2021. The coronavirus pandemic is still ongoing; there is no clear end in sight (though there are hopes that by spring of 2022 things will be back to normal). Despite early successes, there has been a viral resurgence in many countries that believed they had it under control. Much has been learned about SARS-CoV-2 since the pandemic began but there is still a great deal more to learn. Nevertheless, even in this short time, herbal protocols have been found to help; the ones that follow this lengthy discussion of SARS-CoV-2 (see page 97) are based on a depth understanding of how the virus infects people and what it then does in the body, and, as well, which plant medicines can subvert those processes and help protect and restore health. (A year of feedback on these protocols has shown a 95 percent success rate in preventing severe symptoms and shortening the duration of illness.)

To begin with, while SARS-CoV-2 is an acronym, it is also known as Covid-19 (and sometimes as just *the* coronavirus or even SARS-2), which does make things confusing. Regrettably, Covid-19 is also an acronym. It stands for **CO**rona**VI**rus **D**isease of 20**19**, usually written

COVID-19 (but also Covid-19). (It doesn't mean that there were 18 Covids before this one.)

Again, SARS-CoV-2 is a coronavirus. Coronaviruses are some of the largest viruses known. They possess some important differences to our most familiar virus—influenza. With SARS-CoV-2, one primary difference is that it has a very low mutation rate. (Influenza mutates around four times faster than SARS-CoV-2.) Nevertheless, problems have arisen.

A Rather Frightening Look at What Covid-19 Infection Does to the Body

This particular virus, it is becoming clear, is far more dangerous than was first believed. It is also a great deal more aggressive than influenza, to which it has been erroneously compared. And further, it is far more complex and subtle in its actions and much more damaging to the human body during infection than any pandemic respiratory pathogen since 1918. It combines the behavior of stealth pathogens and their associated systemic effects (similarly to organisms such as *Borrelia*, i.e., Lyme disease) with that of some of the more deadly forms of influenza. While the virus is primarily thought of as a respiratory pathogen, it is becoming clear that the nose/mouth/lungs are merely the entry point for the organism. It often spreads outward from those locations, infecting and damaging a wide variety of organs in the body as it travels. This is especially true for those who show no symptoms of infection yet are positive for the virus.

At minimum, 30 percent of those infected have *no* fever, respiratory distress, or cough. They pass through all casual testing processes, are believed to be uninfected, and continue to spread the infection. Further, newer studies are finding that children, who tend to become less ill than adults, are far more commonly infected than was believed and have a far greater viral load in their upper respiratory system (e.g., the nose) than even those adults who are seriously ill do. (This is apparently true of the asymptomatic who are in their teens, 20s, and 30s as well.) It now appears that they are acting as asymptomatic spreaders of the infection.

At this point it is known that the lungs, kidneys, heart, brain, GI tract, skin, and blood cells/circulatory system are the main organs affected by

the organism. From 20 to 50 percent of people hospitalized for Covid-19 have some form of heart damage or arrhythmias. About 20 percent have skin rashes. Significant blood clotting is occurring throughout the body for many people and this is probably the most serious common problem. As Dr. Jeffrey Laurence, a hematologist at Weill Cornell Medicine in New York City, commented, "The number of clotting problems I'm seeing in the ICU, all related to Covid-19, is unprecedented. Blood clotting problems appear to be widespread in severe Covid" (Rettner, 2020). Worryingly, many people in their 20s, 30s, and 40s, without any other symptoms, also have this kind of clotting but it is only discovered after they have a severe heart attack or stroke (which may be brought on by physical exertion or may simply occur on a day that seems like any old day at all).

The virus's preferred attachment point is what are called angiotensin converting enzyme 2 (ACE-2) receptors on our bodies' cells. (I will go into more depth on this later.) Damage to the endothelial cells, which are high in ACE-2 receptors and which line blood vessels, veins, and arteries, is far more common than was suspected, even in those with mild or no symptoms. This is the main source of the clotting problems people experience—though the inflammatory cascade the virus initiates plays a part as well, as it often does in many different types of microbial infections.

In consequence, it's clear that in addition to the testing necessary to determine if people are infected, a blood test for D-dimer levels is crucial. D-dimer is a fibrin degradation product that is present in the blood after clots are degraded by fibrinolysis in the body. Levels of D-dimer in the blood give a good indication of how pervasive clotting is, especially in those with no symptoms. Without this, the only other test that can give an indication of problems is the use of an oximeter, which measures blood oxygen levels. Healthy readings should run around 96 to 98 percent. If those levels begin to decrease, it indicates problems in lung/blood oxygen exchange. (Oximeters are very inexpensive and can be ordered online—it is a very good idea to get one.)

Neurologists removing large clots from the brains of fairly young people infected with the virus have found that as fast as they remove the clots, more form. Many of those who have died from the virus have been found to have hundreds if not thousands of tiny blood clots throughout

the lungs and, many times, in other organs such as the brain and the kidneys.

Damage to the blood vessels close to the surface of the skin is the source of the rash that is now known to be relatively common, occurring in around 20 percent of those infected. Infection of the GI tract can present merely as mild gastrointestinal upset, transient or continuing diarrhea, bloody diarrhea, vomiting, and severe abdominal pain. Damage to the GI tract can be severe. (The virus infects the GI tract, in part, so it can spread through feces excretion.)

Diagnostic imaging of the GI tract of those infected with SARS-CoV-2 (even in those with no pulmonary symptoms) has found severe damage to the bowel in a number people who were admitted to hospitals. Extensive clotting has led to the loss of circulation to portions of the bowel (ischemia) with portions of the bowel becoming necrotic (dead) in consequence. As Rajesh Bhayana notes, "Some findings were typical of bowel ischemia, or dying bowel, and in those who had surgery we saw small vessel clots beside areas of dead bowel" (Palmer, 2020). There is no way, as yet, to determine how many people's bowels have been seriously affected; virtually no diagnostic imaging of this sort is being utilized at this point in time.

Half of the infected show signs of kidney damage with up to a third needing temporary or permanent dialysis. (Eighty-two percent of those with subsequent kidney damage had no history of renal problems.) Due to the extensive clotting the virus causes, dialysis catheters often clog with clots during treatment and have to be continually cleared from the machines. Kidney failure is a common contributing factor to death from the virus. The virus also infects the endothelial cells of the bladder; it extensively sheds viruses from this location, spreading in expressed urine. The infection in the bladder can lead to recurring urinary troubles such as bladder pain (cystitis) and frequent urination. Proteinuria, hematuria, and elevated serum creatine and urea nitrogen have all been reported as well (though in lesser numbers) and still occur for some postinfection "long haulers."

In the brain, excessive clotting is the source of the mild to severe strokes that sometimes occur. Some of the first signs of this are slurring of speech and difficulty walking. Serious strokes leading to necrotizing

hemorrhagic encephalopathy, incapacitation, and death are also being reported. But the impact on the neurological system can be far broader.

Neurological symptoms can run the gamut from mild to severe. Somewhere between one-third and one-half of those infected display some form of neurological effects. These can be as mild as loss of smell or taste, muscle weakness, headache, nerve pain, depressed levels of consciousness, dizziness, tingling/fizzing sensation, hair and scalp pain, confusion, and a sense of not being one's self or as serious as encephalitis, seizures, and long-term mental impairment.

This virus, like SARS-CoV-1, apparently attaches to olfactory neurons in the nose. To infect neurons the virus doesn't utilize ACE-2 but a different cellular receptor—CD147—and from there spreads to the brain. (There is some confusion, a.k.a. argument, in the literature as to whether or not neurons express ACE-2; some say yes, some say no, fisticuffs at 4 behind the playground.) It spreads outward from the olfactory bulb in the brain to regions closely affiliated with that initial site.

Once the virus accesses the brain cells, it begins to replicate in some while killing substantial numbers of nearby cells. Colloquially, it appears to "suck up all the oxygen nearby," thus causing hypoxia, and death, in neighboring cells. However, the cells in which the virus replicates survive, allowing further reproduction. Synaptic connections decline; as neuroscientist Alysson Muotri comments, "Days after infection and we already see a dramatic reduction in the amount of synapses" (A. Mandavilli, 2020).

Portions of the brain as well as the brainstem, spinal cord, and cerebral spinal fluid all show viral infection. Autopsies have found damaged brain neurons and multifocal lesions in the brainstem, cerebral white matter, and cerebellum. (Infection of the cardiorespiratory center in the medulla, which has been found to occur, is possibly the reason for sudden respiratory failure in a number of the infected. This may also explain the extremely odd circumstance where some of the infected present with blood oxygen levels as low as 50 percent, which should cause unconsciousness, but they show no signs of respiratory distress—a.k.a. "happy hypoxia.")

The virus has also been found to attach itself to another receptor on cells, neuropilin-1. This can substantially reduce the experience of pain during Covid-19 infection. During viral infections, vascular

endothelial growth factor A (VEGF-A) is often released. VEGF-A binds to neuropilin-1, generating hyperexcitability of neurons—and pain. By binding to neuropilin-1 the virus inhibits VEGF-A binding, which stops the hyperexcitability, thus inhibiting the pain response. Thus, people are infected but feel none of the normal range of pain effects that infection usually causes. In other words, they remain asymptomatic. Because the pain is suppressed, they go about their day, further spreading the virus.

There is no evidence yet of demyelination of the neural structures of the brain, something that often occurs with acute viral infections, but research into the neurological impacts of infection is in its early stages—such damage is often seen only weeks or months later. A number of specialists are suggesting that anyone who has had the disease have regular neurological monitoring, perhaps for as long as a year after infection.

Some of those who have recovered still show neurological deficits, which seems likely to be a continuing aspect of what is now becoming known as post-coronavirus syndrome (as are various forms of damage to the kidneys, GI tract, heart, and lungs).

Nearly all the infected show elevated liver enzymes and this can continue for months after apparent resolution of the infection. About one-third of those who become ill experience liver problems. The problems are usually mild but in some cases have led to severe hepatitis. The endothelial cells in the gallbladder ducts are particularly susceptible to infection by the virus, which leads to what is called cholangiocyte injury, a bile duct inflammatory condition that can cause severe damage. This is the most common serious liver problem. (Postinfection, the use of standardized milk thistle seed to protect the liver and normalize its functioning is probably a very good idea.)

The virus does circulate through the spleen and lymph system but there is no data yet on whether it damages that system, or the bones, or pancreas, and so on. Those struggling with post-coronavirus syndrome commonly report enlarged lymph nodes as a continuing problem.

It's becoming clear that the virus may play a far more serious role in the reproductive system than is currently thought to be the case. ACE-2 receptors are very high in both ovaries and testicles. They are also high in the uterus, endometrium, and vagina. ACE-2 expression also varies with the menstrual cycle and there is evidence that Covid-19 infection or post-coronavirus syndrome are causing alterations in that cycle. But

concerns are also arising that the damage to the female reproductive system might be far worse—that is, viral infection of the ovaries could affect fertility. Some specialists are now recommending that women practice birth control (or abstain from sex) during infection and for at least 8 months after infection clears. There is no way to know, at this point in time, the effects of the virus during the early stages of pregnancy but ACE-2 receptors are highly expressed in the placenta, which could lead to problems in fetal development.

In men, ACE-2 is highly expressed in the testes, in Leydig cells and Sertoli cells. ACE-2 is very high in spermatogonia—the early cells that later become sperm. And the virus has been found living quite happily in sperm. Sexual transmission is apparently common and can come via either the male or female. Orchitis (inflammation of the testicles) is a potential long-term problem (both from viral infection and so-called autoimmune orchitis). Free testosterone levels tend to be significantly lower in some men who are infected; there is growing evidence that infection of the testicles is damaging fertility. Emerging research continues to show a wide variety of negative, potentially long-term impacts on both female and male reproductive systems.

There are early indications that the virus is damaging endocrine function in the body—that is, upsetting its complex hormonal processes. Those experiencing long-term post-coronavirus syndrome have reported problems in endrocrine functioning, and at least one group of researchers (Mongioi et al., 2020) have expressed concern about possible long-term endocrine-metabolic problems after infection.

Musculoskeletal problems have also been reported by the infected as well as those struggling with postinfection problems. Muscle pain (myalgia) is common in around 60 percent of the infected, and arthralgia (joint pain) in around one-third. Many people, postinfection, continue to experience this, usually on a cyclical pattern.

And, of course, infection and damage to the lungs can be extreme. The long list of complications that can occur in the body and its organs, both short and long term, are explored in the various sections that follow.

Similarly to borrelial infections, early suspicions are arising that the virus may sequester itself in protected locations in the body only to reemerge later, after treatment has ceased and the infected person is considered cured.

There are scores of people now, in the United States, in the United Kingdom, and throughout the world, who appear to have recovered only to "relapse" days, weeks, or even months later. Further, to make things worse, the symptom picture continually changes, sometimes with every resurgence. As Paul Garner, a professor of infectious diseases who himself experienced these types of relapses, comments, "Every day there was a surprise, something new. . . . I spoke to others experiencing weird symptoms, which were often discounted by those around them as anxiety, making them doubt themselves." (This is typical of those with recurring stealth-type infections—as many in the Lyme community have discovered. They seem better, the disease resurges, fatigue and other symptoms recur, and everyone, including their doctors, default to "it's all in your head." This is known as gaslighting.)

As he goes on to say:

> The least helpful comments were from people who explained to me that I had post viral fatigue. I knew this was wrong. There was a pattern in that period from two weeks to six weeks: feeling absolutely dreadful during the day; sleep heavily, waking with the bed drenched in sweat; getting up with a blinding headache, receding during the day, turning me into a battered ragdoll in the evening.
>
> I joined a Facebook page (Covid19 Support Group (have it/had it)) full of people with these stories, some from the UK, some from the US. People suffering from the disease, but not believing their symptoms were real; their families thinking the symptoms were anxiety; employers telling people they had to return to work, as the two weeks for the illness was up. And the posts reflect this "I thought I was going crazy for not getting better in their time frame"; "the doctor said there is zero reason to believe it lasts this long." And too, people report that their families do not believe their ever changing symptoms, that it is psychological, it is the stress. (Garner, 2020)

As Luke Harding reports, "According to the latest research, about one in 20 Covid patients experience long-term on-off symptoms. It's unclear whether long-term means two months, or three or longer. [Note: Emerging data indicates it may be much longer.] The best parallel is dengue fever, [Paul] Garner suggests—a 'ghastly' viral infection of the lymph nodes which he also contracted. 'Dengue comes and goes. It's like driving around with a handbrake on for six to nine months.'" Or, as Lynne Turner-Stokes, professor of rehabilitation medicine at King's College, London, puts it (in typically convoluted language), for a percentage of those infected there is a "recrudescence of symptomatology" (Harding, 2020). The virus, in these cases, may

be sequestering itself (as Lyme bacteria, and others, do) and then reemerging after treatment, or the organism may be generating a new form that the immune system does not recognize, or perhaps the immune system antibodies have become less effective over time. (There are "long haulers" who now have been ill for as long as 12 months.)

Some people are testing positive for months and never seem to throw off the infection. As reporter Roxanne Khamsi comments, "One doctor had multiple positive coronavirus tests 90 days out from her initial diagnosis." Some researchers are speculating that there might be people who remain infected for very long periods of time. Virologist Richard Randall, for example, comments that it's not impossible that there might be people who can remain infective for 6 months or even as long as a year. "Those people may act as seeds or reservoirs for the virus and potentially could be the source of a local outbreak. I am not saying it is happening for Covid-19 because the data's not there. But that happening would not be surprising" (Khamsi, 2020).

Newer viral research is indeed finding that many viruses can remain active in various locations in the body for many years. And some coronaviruses can remain active in test animals' liver and central nervous system for exceptionally long periods of time. Kenneth Witwer, a molecular biologist at Johns Hopkins University, thinks that SARS-CoV-2 sequesters itself: "I still think that this phenomenon is likely explained by a persistent cellular reservoir of low-level replication, not by residual virus particles." (Noninfective viral particles can lead to a positive PCR test.) As he notes, viral RNA degrades very quickly, and there is no other reason for it to be found for months after infection (and thus leading to a positive test) unless new viruses are releasing particles (Khamsi, 2020).

Despite the increasing evidence for viral sequestering, some are suggesting that such may not be the reason; it may be reinfection after cure. (This is not uncommon with coronaviruses.) This would mean that previous infection does not confer immunity or that it is of very short duration. (The current speculation by researchers is 3 months of immunity after infection.) Looking at other coronaviruses: For those that recovered from SARS-CoV-1, immunity lasted for 2 years; immunity from coronaviruses that cause the common cold fades in a year. No one knows how long immunity to SARS-CoV-2 will last. (In late April of 2020, the World Health Organization issued a statement that it

should *not* be assumed that previous infection would confer immunity to repeat infections. Currently, numerous countries are reporting reinfection after recovery, especially with the mutated variants.)

And if all this were not enough, a great many people (between 30 and 40 percent of those infected) have been found to be asymptomatic for the disease and yet be silent carriers. People have been known to carry and spread the virus for weeks before symptoms arise (if they ever do) and for up to 4 weeks after infection is thought to have cleared (and perhaps a great deal longer).

The true rates of infection and death from the virus are not yet known and probably won't be known for 1 to 2 years. The reasons actual figures can't be known for so long is due to a variety of factors. Those are: early, erroneous beliefs about the virus and what it does in the body, very poor tests, low testing rates, and in the United States, regrettably, the CDC criteria for both infection and death, which nearly always are, and in this instance very much are, far too conservative and limited in scope.

Low testing rates (in the United States and in a number of other countries) give a false picture of infection in the general population. Figures change weekly, often in response to complex research papers that are utilizing various forms of statistical analysis. Few of them agree. I have seen figures speculating that true infection rates are ten times official numbers; others insist it is one thousand times official numbers. Death rates range from 0.05 percent in Singapore to 29 percent in Yemen.

While the elderly (due to simple aging of the body, its immune system, and its organs) and those with underlying conditions (obesity, diabetes, etc.) or immune dysfunction are the most likely groups of people to die from the virus, significant numbers of people in their late 20s, 30s, and 40s are also succumbing to the disease. Far more, in fact, than first thought. (Children of all ages are far more susceptible to infection than first believed, and while rates are low, death is occurring in this group as well.) There are two main reasons for death rates being far higher than is currently thought—though at root it comes down to the same thing, lack of testing.

The first is that, because of its system-wide impacts the virus is causing a great many heart attacks, strokes, and incidences of kidney failure. Unless those who die from causes other than respiratory failure

are tested for coronavirus, the listed cause of death is going to be incorrect. Secondly, a great many people are dying at home. Few of them are being tested for coronavirus. In fact, until recently, unless they had previously been tested for coronavirus and found positive, a death at home was not considered to be coronavirus related.

It is helpful here to look at normal background deaths at home in New York City and deaths at home during the pandemic. Normal deaths at home in that city average around 25 per day. During this pandemic early studies have found daily deaths running from 150 to 275, depending on the week and how diligently apartments are being checked. The true coronavirus death rate is much higher than believed, something that is now being widely recognized. (Most sources are now accepting that true death rates are at minimum 30 percent higher than official figures.)

Covid-19 Mutations

Somebody said that people in many rich countries have got used to thinking that they've conquered all infectious disease, and so there's this hubris about that, and I think that we found that hubris was more profound than we realized. We felt far too safe, and there was really quite a great degree of arrogance in there. . . . [Then] an old colonialist-thinking legacy [arose], discounting Asian science and experience, and that's a large part of what this whole theme is. Just that assumption that you are Americans or Europeans and know best over and over again. If this pandemic has taught us anything, it should be not to think that anymore, and yet, people keep doing it.

—Hilda Bastian, scientist, medical researcher, and health consumer advocate

Like all microbes, and most especially viruses, Sars-CoV-2 is a master of mixing its genome; it is highly adaptable. Viruses, when they move into large populations of people, enter a new ecological territory. They, as we do, learn the new terrain and adapt to it. Viruses are immeasurably older than we are; they have a great deal of experience altering their genomes to better survive in new ecological niches. It's become common among researchers to speak of viruses as having trouble reproducing identical copies of themselves. They say the viruses make "typos" or "copy-and-paste" errors or even that they engage in bad "proofreading." This makes the viruses seem rather stupid: "Oh, the poor things. Can't even copy themselves correctly." The truth is very different.

What is more ecologically accurate is that viruses are a form of swarm intelligence—the individual members are not the entity, the swarm is. One of the primary adaptation patterns the swarm uses is to generate millions of slightly different offspring very quickly in order to produce more highly adaptable forms. The viral swarm also possess the capacity to create new genomic forms through highly sophisticated examination and analysis of their new hosts' ecologies. They *respond* to our responses to them. And they do this in a number of ways.

Analysis of SARS-CoV-2 has found that it took up residence in a number of immunocompromised people around the world, learning a great deal from them in the process. The virus then recombined its genome, creating more adaptable forms that are better able to live within us. In one Boston hospital a 45-year-old severely immunocompromised man remained ill with the infection for 5 months. Doctors sequenced the virus from the beginning of his infection and found that more and more mutations occurred—21 by the end. The virus was experimenting with alterations in the spike protein to find the ones best suited to evade immune responses. After the man was given a new antibody drug, the virus immediately developed alterations to evade it. This exact same process occurred nearly simultaneously in countries throughout the world. As molecular epidemiologist Emma Hodcroft commented, "It becomes almost like a training course for how to live with the human immune system" (S. Zhang, January 18, 2021).

The virus has a plethora of other ways to create variations. Sometimes the virus utilizes the genomes of more adaptable "typo" forms, recombining several different ones in order to create better survivors. The virus also shares genomic sequences with other coronaviruses (such as the corona cold virus) that have already adapted to the human body. (This is especially worrying in that there are thousands of unknown coronaviruses that live in wild ecosystems. Developing a spike protein adaptation that allows easy human infection will, if shared, allow other members of the genus to infect us.) And our medical responses to them, including vaccines, also stimulate adaptation.

These are the main reasons that similar-to-identical variants have emerged, essentially simultaneously, throughout the world. When these variants meet each other in new hosts they innovate again. They share genomic information, creating even more adaptable variations. This is

a very ancient viral adaptation strategy. All microbes, despite what we were taught in school, are highly intelligent, and they have been surviving and adapting for far longer than our species has existed.

A significant number of variations (so-called mutations) emerged at the time of this writing, in the early months of 2021. There is every reason to expect this will be a continuing problem. The first three variations of serious concern in 2021 were the UK, the South African, and the Brazilian. But others soon emerged as well: the powerful California variant, another in New York, one in Spain, and still another in Brazil. The genomic innovations the variants possess are not identical, though at this point in time they are fairly close. Nevertheless, each possesses an alteration in their spike protein (called N501Y). This alteration allows the virus to attach to ACE-2 receptors more easily and more firmly, which makes infection more likely.

The wild or initial form of Covid-19 had a less sophisticated attachment method; the new ones are far more elegant. A bad analogy is that the virus (male) and the cell (female) form a kind of male/female attachment point. The fit wasn't perfect in the original form but it worked well enough. The immune system responded by creating an antibody that "capped" the spike on the virus (the condom), making it more difficult or impossible for the virus to attach itself. But in time the virus adapted itself to the cap (the new variants), altering the spike's shape so that it could not be capped by the antibody. At the same time a much firmer, tighter fit to the ACE-2 receptors occurred. Still other alterations made the virus significantly more transmissible. Viral loads in the infected are up to a thousand-fold higher. Further innovations in the South African and Brazilian variants (the E484K mutation) make them even more capable of avoiding immune system antibodies. The California variant is now known to be extremely transmissible as well, perhaps far more than the initial three variants. The variants are 40 to 70 percent more transmissible, and in some cause much more severe illness.

More troubling, previous infection does not confer immunity to the new variants. People in some areas of Brazil, having already survived infection from earlier forms, are now succumbing to infections from the new variant. The same dynamic appears to be playing out in South Africa. Again: *Previous infection is not conferring immunity*. Nor does donor plasma seem to work against the new variants.

The new variants are unfortunately increasing "exponentially" in the human population. The UK variant, which initially had accounted for only 1 to 4 percent of infections in the United States, had, within a month, surged to 30 to 40 percent. As Dr. Celine Gounder said on *CNN Newsroom* (March 6, 2021), "We are probably right now on the tipping point of another surge." A day later, in an appearance on NBC's *Meet the Press*, Michael Osterholm commented that we are in the "eye of the hurricane." He expects a dangerous upswing infections between early summer and fall 2021. This process—an upsurge, then a downswing, then an upsurge—is likely to continue indefinitely. Newer variants are going to emerge in an endless repeating cycle. The California variant is expected to make up 90 percent of infections on the West Coast by summer of 2021. It, like a number of the others, is far more contagious than earlier forms. As Paul Duprex at the Center for Vaccine Research at the University of Pittsburgh puts it, it was a mistake to "think that we are cleverer than evolution." Kevin McCarthy, also at Pittsburgh, comments, "We've been underestimating the capacity of the virus to evolve since the beginning of the pandemic" (Johnson, March 7, 2021).

The more people the virus infects worldwide, the more it learns, and the more successful variants it will create. Many researchers now believe that *herd immunity is unlikely*. Given the virus's adaptability and its very fast learning curve, the best case scenario is that it may be more accurate to think of it similarly to the flu virus, for which we require a new and different vaccination each year. As the *Washington Post* recently commented, "The pandemic continues. For how long? At this point, anyone giving a confident answer is guilty of hubris" (Achenbach, Cha, and Sellers, 2021).

SARS-CoV-2 is going to be with us for a very long time. The crucial question is, will we be able to adapt to it as well as it is adapting to us?

The Vaccines

[This same kind of arrogance is] happening with vaccines, especially thinking it's all about the vaccines of a few big EuroAmerican multinationals galloping to the world's rescue. One of the most fascinating stories is Cuba. I mean, there's this really interesting juxtaposition between Cuba and Canada, ironically. In Canada there's a debate about why did they let their capacity to produce vaccines dwindle away next to nothing. Cuba had the exact opposite. Cuba had to become self-sufficient

at pretty well everything, and that included producing drugs and producing medical teams. . . . They're going to have a massive amount more vaccine than they need. They're not going to have any trouble vaccinating their population with home-grown vaccines in 2021. . . . They're just going to be exporting masses and masses of vaccine.

—Hilda Bastian, scientist, medical researcher, and health consumer advocate

The first thing to understand is that despite common assumptions Covid-19 vaccines are not vaccines in the way most people think of them, that is, something like the measles vaccine, which prevents all future infection. More properly, they activate the immune system to enable it to better respond to the virus if exposed. For some people this means they will not be infected, while for others it means that they merely have a less severe infection. For others there will be less chance of death if they do become seriously ill. Regrettably, the vaccines that are now in use were designed for the original or wild form; they were not designed for the newer variants. While they, at this point in time, do offer some protection, emerging reports of infection in the vaccinated are becoming common throughout the world.

There are (apparently) some 240 different vaccines in development, none which look likely to provide permanent protection to the coronavirus group. Six vaccines are officially approved/authorized in various countries, 22 are in phase three trials, 23 are in phase two trials, 18 are in phase one. The approved/authorized vaccines are: the Moderna mRNA-1273 vaccine, the Pfizer-BioNTech BNT162b2 vaccine, the AstraZeneca AZD1222 vaccine, the Sinovac Biotech and Sinopharm vaccines from China, the GAM-COVID-Vac (Sputnik V) vaccine, the Novavax NVX-CoV2372 vaccine, and the Johnson & Johnson/Janssen Ad26.COV2.S vaccine.

The Pfizer and Moderna vaccines are made using messenger RNA (mRNA). This delivers a bit of genetic code (the spike protein) into the body, which stimulates the immune system to make antibodies to stop spike attachment. Johnson & Johnson used a different approach, creating what is called a viral vectored vaccine. In this version, a relatively benign adenovirus has been altered to carry the SARS-2 spike protein into the body, which, again, stimulates the immune system to produce antibodies. Pfizer is for people 16 and older, the others for 18 and older.

The Novavax vaccine delivers the actual spike protein (bioengineered via moth cells) into the body to stimulate an immune response.

It also has an additive that "soups up" the immune response—a saponin from the Chilean soapbark tree. (Technically it is considered to be adjuvanted recombinant protein nanoparticles.) The Sinovac Biotech and Sinopharm vaccines use an inactivated virus. This is similar to how the flu vaccine is made. However, in this instance there is some concern about the vaccine's safety, given the speed of the production, in that early trials with the inactivated dengue and respiratory syncytial virus vaccines caused serious side effects in large numbers of people. The Sputnik V and AstraZeneca vaccines are vector vaccines similar to the Johnson & Johnson vaccine. There is also a Sinofi/GlaxoSmithKline vaccine that is apparently somewhat similar to the Johnson & Johnson vaccine and may be available in late 2021 in the United States. And there are many others in use that just don't show up on the media radar. There are five coming out of Cuba, one in Thailand, and another from UNICEF.

Efficacy vs. Effectiveness

As usual, academicians' convoluted language (which the media endlessly repeats) has created confusion about how effective the vaccines are. "Effective" and "efficacy" are not the same thing. Pfizer and BioNTech say their vaccines have a 95 percent *efficacy* rate; Moderna's is 94.5 percent; Sputnik V is over 90 percent; Johnson & Johnson's single shot is (on average) 66 percent (but 85 percent against severe disease); Novavax is 89 percent but only 50 percent against the South African variant. The media have often translated this improperly, as in "Moderna's vaccine is 94.5 percent effective." Most people assume that if a vaccine is 95 percent effective, it means that 5 out of 100 people who receive that vaccine will get sick, and 95 will not. But there is a difference between efficacy and effectiveness, and in the real world it is a big difference.

Using the Pfizer vaccine as an example: There were 43,661 people recruited for the trials. They were split into two groups. One received a placebo, the other the vaccine. Then the researchers waited for 170 of those people to become ill—that is, to show symptoms. (Yes, a strange number; I have not been able to find the rationale for it, it was apparently a suggestion from a statistician as being the lowest meaningful number.) Then they looked at who got sick. Of the 170 people who became ill with Covid-19, 162 had received the placebo, and 8 had received the vaccine.

If people in the placebo and vaccine groups become ill in the same numbers, the efficacy of the vaccine is zero. If no people in the vaccine group get sick, then the efficacy is 100 percent. The 95 percent number represents a relation between the numbers of people who got sick in the placebo and vaccine groups, that is, 8 divided by 162, which gives you 0.05 (close enough), which is 5 percent. In other words, the number of people in the vaccine group who got sick is 5 percent of 162, and therefore the vaccine is 95 percent effective. This makes sense in the world researchers live in, not in the real world. It confuses people, but more concerning is the fact that these figures don't actually have anything to do with the real world.

These trials worked with a very limited number of people. In the real world, hundreds of millions of shots will be given (billions eventually, utilizing a variety of different vaccines). The larger group of people who get the vaccine will be much different in their health and genetic makeup than the volunteers in the trials. In consequence, the efficacy percentage (that is, the effectiveness) will be different in the real world; no one knows what it will actually be. Further, many people who become infected show no symptoms; this was true in the trials as well. But the trials counted only those people with symptoms. This is going to alter both efficacy and effectiveness percentages.

Four further points: 1) The shots only reduce the severity of the disease if you become infected; they do not prevent infections (as, for example, the smallpox vaccine does). They act more as a "dampener on the virus's ability to replicate inside you" (S. Zhang, February 9, 2021). Because the severity is reduced, the assumption is that there will be less chance of dying. (No one knows as yet if this is true; this was not examined in the initial trials.) 2) The shots are into muscle tissue. This stimulates overall immune responses but it doesn't necessarily stimulate immune responses in the nasal tissues, where the virus first takes hold. 3) Most of the vaccines stimulate antibody production. Antibody production falls over time, so that, again, shots may be necessary every 6 months to 1 year. (No one knows about this either.) 4) Unexpected side effects will also appear, and they often will be very different than those found during the trials. (This is true of *all* vaccines.)

Vaccine Side Effects

The major side effects of the vaccines (so far) appear to be pain (sometimes severe) at the site of the injection and overall feelings of having the flu for a few days (chills and fever, nausea, aches and pains, fatigue). People report that these are far more severe after a second injection (with the two-dose vaccines). However, reports are emerging (as of March 2021) that the range of side effects is far broader than was first imagined. Here's a list (which has been compiled from official government and pharmaceutical sources, media reports, and extensive personal communications): mild to severe allergic reactions (anaphylaxis); tremors (both transient and permanent); immune thrombocytopenia (very serious); Bell's palsy; headache (transient and continuing); constipation; diarrhea; jitteriness; odd taste in the mouth; alterations in menstrual flow and texture; dizziness; fatigue; high fever; severe joint pain; severe bruising and swelling at the site of injection; chills; tachycardia; seizures; severe nausea; vomiting; bone pain; slurred speech; facial numbness; pressure behind the eyes; paralysis of the body or limbs (sometimes extreme and long lasting); mental confusion (sometimes severe); hearing loss; extreme abdominal pain; swollen lymph nodes (a.k.a. lymphadenopathy, sometimes severe); loss of sight (temporary); acute appendicitis; passing out; mottling and cyanosis of the extremities; acute pancreatitis; stroke; heart attack; angioedema; neurological deficits (various); hospitalization due to side effects; death. While some side effects present immediately, many physicians are reporting that there is often a delay between dose and side effects of up to 8 days.

Note: The CDC maintains a database of vaccine side effects that have been reported to the government (more are being reported all the time). You can find it by searching online for "Vaccine Adverse Event Reporting System." To access the data for Covid-19 vaccines, in the menu where the system asks you to select vaccine characteristics, select the option for Covid-19 vaccines. You can tailor your search by other factors, such as demographics, vaccine manufacturer, specific symptoms, et cetera, if you like.

Regrettably, government spokespeople, medical researchers, and media articles continue to downplay vaccine risks (when what is true at this point is that no one can know how many there are or how severe they are going to be). As an example: Despite instances of Bell's

palsy onset immediately after injection, some medical reports insist that because the number of palsy events is not larger than the normal percentage in the population, it has nothing to do with the vaccine. This is not doing anything to alleviate concern about side effects; quite the contrary.

It *is* true that only a small percentage of people are experiencing side effects when compared to the total number of doses given, but the fact remains that some people are having very serious reactions, some of them permanent. That it is rare does not appear to help those who become very ill ("Sorry, don't do no good, does it?"). As with all things you put in your body (pharmaceuticals or plants), every person should carefully examine their own state of health, what is known about the substance and its possible side effects, and their intuitive sense of the risks for them as an individual, and then decide for themselves how they wish to proceed.

I think when you have no commercial experience with a vaccine strategy and you're using that as a way to try to stop a new virus, there will be something of a learning curve. . . . I wish there was a little more humility from some of these companies. . . . You're only going to know about rare adverse events once these vaccines are out there, because even in a best-case scenario, they are tested on 20,000 or 30,000 people, not 20 million or 30 million people. So you are only going to know about a rare adverse event post-licensure. . . . When do you know enough to say that a vaccine's benefits outweigh its theoretical risks? You have to also make sure people know what you don't know. You don't know how long protection is going to last. You're only going to know that afterwards. You don't know whether it causes a rare side effect. You're only going to know that afterwards. . . . I just think we have to be honest and transparent about what we know and what we don't know.

—Paul Offit, MD, September 9, 2020

There is no other way to say this . . . what we are facing is serious. If the emerging picture of what the virus is doing is borne out, and if it continues to develop more sophistication with the human body and its immune responses, the human species is going to be in for a very bumpy ride.

There is little reason, at this point, to believe that this virus, as SARS did, will just fade away anytime soon. As Michael Osterholm has commented, "We will be dealing with this forever" (Rosenbaum, 2020). We will only discover what is true over time, perhaps only over several years. But we are, in fact, in a hell of a mess.

A Deeper Look at What the Organism Does in the Body

As time goes on, much more will be learned about the virus, its infection processes, and what it does in the body. Nevertheless, here is a pretty good view of what is happening. Knowing what the virus does and how it does what it does gives a good deal of information about how best to create and utilize sophisticated herbal (or medical) protocols to intervene in the process.

The virus is primarily spread through the air (inhalation) and transfers during touch between the infected and the noninfected. While it was originally assumed that the virus only traveled on large exhaled droplets (those from coughing, for instance), it is now known that it also attaches to tiny aerosol particles that are simply breathed out during respiration. This is why the virus infects certain cellular structures in the nose first—so it can use our breathing to spread from person to person.

Quite often there are no symptoms during the early stages of infection. On average, 30 to 40 percent of those who are infected have no symptoms. (Nevertheless, this is only an average—in a Boston homeless shelter, 147 people were infected but 88 percent of them had no symptoms; a poultry plant in Arkansas reported that 481 people were infected but 95 percent of them had no symptoms.)

Some people may begin to feel unwell within a few days, others can go weeks before they do, and some never feel ill. For most of the infected, there are 1 to 2 days during this nonsymptomatic period when the virus reproduces in tremendous numbers, promiscuously spreading in our exhalations (viral shedding). This ensures that, before symptoms appear, it can quickly spread throughout the population. It's a matter of timing. Someone who, during that particularly infectious period, immerses themself in a crowd can infect scores to hundreds of people in what are called superspreader events. (Restaurants, bars, nightclubs, concerts, convention gatherings, church services, warehouses, packed grocery stores, confined workspaces . . . all of these are perfect venues for superspreader events to occur. The more enclosed the space is, and the more poorly ventilated, the more that viral aerosols will circulate within the crowd.)

Infection can, of course, occur at any time during the course of the disease. (Asymptomatic people tend to shed longer, around 19 days versus 14 for the symptomatic.) It is just that during this early period, the virus sheds in far greater numbers—and infection is transmitted far more easily because of this. (Researchers have found that around 80 percent of infections are from superspreaders.) The more virus a person inhales, the greater their chance of being infected. Because the virus is very stable in tiny aerosol mists it can remain in the air a long time and infect people who are quite far away. This has particular relevance for large gatherings in enclosed spaces.

Concern has been raised about infection occurring from touching virus-contaminated surfaces. However, that doesn't appear to be a serious problem except in small enclosed rooms where those with active infections are sequestered over long periods (e.g., hospital rooms). Viral concentrations ranged from 55 percent (remote controls for televisions) to over 80 percent on ventilation grates in their rooms. No surface was found free of virus particles. The main routes of infection are still considered to be inhalation of virus-infected aerosols and droplets and from droplets or aerosols on the hands that then touch the mouth, nose, or eyes.

Once in the nose the virus looks for ACE-2 receptors. There are about 40 trillion cells in the average human body, a great many of these have ACE-2 receptors, and the nasal passages are no exception. It is here that it begins to reproduce so that it can spread through exhalations and, as well, begin to move deeper into the body. Once the virus enters the body, it has millions more options for attachment.

The "spikes" on the virus (famous from the many media representations of them) are the part of the virus that attaches to the ACE-2 receptors on the cell. To facilitate this, the spikes utilize an enzyme found on our cells—transmembrane protease, serine 2 (TMPRSS2). This enzyme "primes" the virus's spike protein so that it displays itself as a "fusion protein" to the cell via the ACE-2 receptors. This allows viral attachment to the ACE-2 receptor and subsequent entry into the cell. (The primary herb that can be used to protect TMPRSS2 integrity, and stop the priming, is *Salvia miltiorrhiza*.) A number of the more infective influenza viruses also utilize TMPRSS2 in this way. (Another intelligent intervention is interfering with spike attachment to ACE-2; see page 85.)

Generally, the virus first enters the so-called upper respiratory system (beginning with the nose). There it attaches to ACE-2 receptors on certain epithelial cells, specifically goblet secretory cells (which produce mucus) and ciliated cells (which have tiny hairlike extrusions, i.e., cilia, that move mucus and particulate matter up and out of the respiratory system). (*Bidens pilosa* is protective of these cells.) The virus utilizes those cells' TMPRSS2 to prime the spike, allowing entry inside. These particular cells possess a large number of innate immune-associated antiviral genes, which is leading to speculation that the virus may be using its access to these cells to subvert a healthy immune response. (And, indeed, interferon production does seem to be inhibited early in the infection.)

Once it gains entry into the nasal cells, the virus utilizes those cells' structures in order to reproduce, creating more copies of itself. At this point in the infection there are often no symptoms. Then, for a week, sometimes longer, the virus releases copies of itself from the infected cells. (Tests of health care workers without sufficient protective equipment found that their noses and mouths were full of live viruses that they then exhaled onto every new patient they saw.) The viruses travel outward with the breath (and also infect the hands when you rub your nose), enabling them to spread to other people, passing the infection

The main purpose of masks is to protect others if we are infected. It is not primarily to keep us from getting infected—though it does in fact help prevent it. (And yes, if you don't cover your nose with the mask, you are still infecting people.) For those with underlying lung conditions such as COPD or post-coronavirus problems (which often make mask wearing difficult because of the buildup of CO_2 behind the mask), the use of a mask with an external rechargeable air-purifying respirator, about the size of a pack of cigarettes (and which is worn on the arm), is a good idea. A tiny pump sends highly filtered outside air into the mask so that breathing is far less impaired than with a conventional mask. I now use a "4WDKING rechargeable electrical air purifying reusable portable air purifier with HEPA filter." It comes with a substantial number of replacement paper masks (which the pump/filter system connects to). The filter is good for 500 hours before replacement is necessary. The incoming air is *very* well filtered, far more than regular masks, and is cooled as it comes into the mask, which also makes breathing easier.

more widely into the species. And again, exhaled aerosols, not just droplets, can spread the virus, thus extending the range of infection to something like 15 feet, not the 6 feet that is generally suggested for safety.

The viral infection of olfactory sensory neurons (or their underlying cellular substrate), located in a small area of specialized tissue high in the nose, is the reason for the loss or dysregulation of smell that is one of the early signs of infection. The virus disrupts the cilia in the olfactory neurons; they become completely detached. Once this occurs, people lose their ability to detect odors. There is some conflict over which cells are causing the problem, infected neural cells or underlying support cells in the epithelium. (Tempers among researchers tend to run a bit high.) Olfactory neurons in the nose do connect directly to the brain and are, according to some researchers, one avenue the virus uses to infect the brain.

From the nose the organism begins to move deeper into the respiratory system, infecting goblet and ciliated cells in the throat and bronchi. This is the point where the first symptoms generally appear: slight fever, dry cough, sore throat, head and body aches. From there, the virus moves deeper into the lungs (the so-called lower respiratory system), where its preferred cell is type II pneumocytes (a.k.a. type II alveolar cells). (ACE-2 receptors are also strongly present in type I cells, but the virus seems to prefer type II; there doesn't seem to be anything in the literature on type I infection.) These cells are common throughout the lungs' alveoli (along with type I) and exist in scattered pockets in the bronchioles, and as well in the alveolar ducts.

The alveoli are incredibly tiny, microscopic, grapelike sacs at the end of very tiny, also microscopic bronchioles. Air travels through the bronchi, which diverge into smaller and smaller and still smaller air passages, at the end of which are the alveoli. The alveoli have an extremely thin exterior membrane that is covered by a network of incredibly tiny blood vessels. As we breathe in, the alveoli expand much like very tiny balloons, then oxygen (and other gases and volatiles) pass through the thin alveoli membranes into the bloodstream, and carbon dioxide (and other gases and volatiles) pass from the blood into the alveoli and are breathed out. There are around 300 million alveoli, so there are a great many ACE-2 receptors in the lungs for the virus to attach to.

As the infection progresses the immune system responds. White blood cells release activated molecules to fight the virus (cytokine is the

general name for messenger molecules, and chemokines are specialized cytokines that call immune cells to the sites of infection, but I just call them all cytokines). The alveoli fill up with fluid (edema) and dead cells, which makes breathing more difficult (pneumonia). Coughing, fever (often high), and rapid and slow respiration are common. (There are exceptions; some people never show respiratory symptoms.) Blood oxygen percentage falls. Some people experience what is called acute respiratory distress syndrome (ARDS). This is generally accompanied by what is often called a cytokine storm, a massive inflammatory response throughout the body. The oxygen levels in the blood plummet, and the alveoli are filled with pus, mucus, white blood cells, dead viruses, and destroyed lung cells. (These are the people most commonly put on ventilators. However, the majority of ventilated people, around 85 percent on average, die; there is growing recognition that ventilators may not be a proper intervention with this particular infection.) For many people, the damage to the thin cellular barrier between the alveoli and the blood vessels results in scarring, a.k.a. fibrosis. (When the alveoli are damaged, hypoxia, and eventually emphysema, or severe fibrosis can occur. Protecting the cells from this induced hypoxia can help reduce damage in the lungs. Rhodiola, *Rhodiola* spp., is specific for this. It prevents hypoxia-induced oxidative damage, increases intracellular oxygen diffusion, and increases the efficiency of oxygen utilization.)

The scarring that occurs is one cause of what is commonly called COPD (chronic obstructive pulmonary disease) or sometimes idiopathic pulmonary fibrosis. This results in long-term pulmonary problems. (Because of the scarring, the oxygen exchange is impeded so that oxygenization does not occur efficiently—thus during any event that demands the use of the muscles people run out of breath, often quite quickly. The scarring is usually progressive over time.)

It is not known how many people are developing this postinfection complication in the lungs, but it is cause for concern. There is the significant possibility that thousands of people who have apparently cleared the infection are going to experience long-term, debilitating impacts on various organs of their bodies, which will necessitate continued care the rest of their lives. (Speculation is that a decent estimate is 10 percent of the infected, whether symptomatic or not. Since current infection levels are around 30 million in the United States, that would indicate that

there are now 3 million people with post-coronavirus problems, a.k.a. long haulers. There are now internet-based long hauler support groups in many countries around the world. A rough, back-of-the-envelope computation shows current U.S. membership in such groups to already be around 200,000 members.)

One of the more important interventions during Covid-19 infection is keeping the lymph system working well in order for the immune system to work most effectively. *Salvia miltiorrhiza*—and cleavers (*Galium* spp.)—will help both the spleen and appendix (which is not a vestigial organ as reductionists have long insisted but an important part of the lymph system) work at optimum levels as well as keeping the lymph nodes clear of infection debris and thus less swollen. (Given that *Ceanothus* spp., a.k.a. red root, does stimulate clotting—although its actions are offset by other herbs in the protocols—I would avoid its use during Covid-19 infections.)

The less clogged the lymph system is, the more efficiently the body can process the cellular debris that comes from immune activity. This necessity extends to the lungs themselves as they also possess an extensive lymph system with similar nodal structures that are used to regulate interstitial fluid clearance. This is often impaired during Covid-19 infection, in part through the viral damage to lymphatic endothelial cells. (Lymph vessels are lined with endothelial cells just like blood vessels.) During pathological states where the lungs' lymph system is impaired, this loss of lymphatic function itself creates an inflammatory condition in the lungs. Protecting the integrity of the endothelial cells of the lymph vessels in the lungs and stimulating healthy lymph function is important.

In addition to *Salvia miltiorrhiza* and *Galium* spp., another herb of note (for the lungs) is *Eleutherococcus senticosus* (a.k.a. eleuthero or Siberian ginseng). It has, among its many actions, the ability to stabilize lymphatic vessels by protecting and enhancing the endothelial cells of the lymph system. The use of the herb, in clinical trials, has been shown to stimulate lymph drainage to such an extent that edema of the lower limbs was "significantly" attenuated at 2 and 4 hours after ingestion. Other herbs of note are *Scutellaria baicalensis* and *Polygonum cuspidatum*, which are both highly protective of lymphatic endothelial integrity as well as interfering with cellular invasion by pathogens

or the damaging impacts of cytokines. As specifics, both pleurisy root (*Asclepias tuberosa*) and inmortal (*Asclepias asperula*) can help stimulate lymph drainage from the lungs.

During viral infection of the lungs, the microbiome of the lungs is significantly disturbed. This can allow a bloom of what are normally quiescent pathogenic members of the microbiome. This is why during viral pneumonia most physicians will also prescribe broad-spectrum antibiotics in an attempt to ward off pathogenic bacterial overgrowth (a practice that has exacerbated the emergence of resistant bacteria in hospitals). Additionally, because coronavirus commonly infects the lower GI tract, *its* microbiome is also disturbed. Crucially, the lung and GI tract microbiomes are, in essence, a single interconnected system. Of further concern is that pathogenic or antibiotic disturbances of the GI tract microbiome also negatively affect heart function, another organ strongly impacted by the virus. (The daily use of a probiotic is strongly suggested. The cheapest good one is PB8, but those in the $40 to $60 range are better.)

At first it was believed that the virus was a typical, although unique, respiratory pathogen. It isn't. The virus can become systemic, affecting many other organs in the body. This is because ACE-2 is widely distributed throughout the body's tissues. As Hamming et al. (2004) noted in their exploration of SARS and ACE-2:

> Since identifying the possible route of infection has major implications for understanding the pathogenesis and future treatment strategies for SARS, the present study investigated the localization of ACE2 protein in various human organs (oral and nasal mucosa, nasopharynx, lung, stomach, small intestine, colon, skin, lymph nodes, thymus, bone marrow, spleen, liver, kidney, and brain). The most remarkable finding was the surface expression of ACE2 protein on lung alveolar epithelial cells and enterocytes of the small intestine. Furthermore, ACE2 was present in arterial and venous endothelial cells and arterial smooth muscle cells in all organs studied. In conclusion, ACE2 is abundantly present in humans.

Because ACE-2 is present throughout the body, the virus can, theoretically, affect any location in which those receptors exist. Again, at this point in time, the lungs, kidneys, GI tract, heart, blood vessels, skin, eyes, and liver/gallbladder are known to be the most common infection sites.

ACE-2 is ubiquitous in endothelial cells in all large and small arteries and veins in all the tissues of the body. Smooth muscle cells

have them, as do myofibroblasts (their infection is possibly the source of muscle weakness during and after infection) and the membrane of fat cells in the organs and everyplace that fat accumulates. The entire GI tract has large numbers of ACE-2 receptors: the stomach, duodenum, jejunum, ileum, and colon. (Viral infection of the GI tract is the source of the diarrhea that many people experience.) ACE-2 receptors are present in the basal cell layer of the epidermis, in hair follicles (the infection of which causes hair loss in some people), in and around the sebaceous and sweat glands, and in all the blood vessels that lie close to the skin surface. (This is the source of the skin rash that about 20 percent of those infected report.) The brain has ACE-2 receptors, as do the bile ducts, lymph nodes, heart, and kidneys. More troubling, because ACE-2 receptors are common throughout the entire circulatory system in vessel and arterial walls, blood clotting in the circulatory system has become a serious issue.

Blood Clotting

The blood coagulation problems seem to come from two main causes. As the virus spreads through the blood, it has access to a great many ACE-2 receptors on the circulatory system's endothelial cells. Viruses attach to the receptors, enter the cells, reproduce, and blow the cells apart as their offspring exit. In essence, this is no different than a scrape to the surface of the skin. It's a wound. So, the internal version of a scab begins to form. Unfortunately, this is happening not to one cell but to thousands.

Damage to the endothelial cells that line the vessels recruits platelets to that location, where they begin to cluster at the point of damage. The platelets initiate the production of fibrin, which forms a kind of net that traps within it more platelets and red blood cells, essentially plugging the wound (i.e., creating a clot, a.k.a. the internal form of a scab) in the vessel wall. This is the initial, primary cause of the massive clotting in the body—though as mentioned earlier certain inflammatory processes stimulate systemic clotting as well. Because so many cells are affected, there are hundreds to thousands of clots forming throughout the entire circulatory system and, potentially, in every organ of the body.

Given that coagulation (clotting) problems are so extremely common with this infection—as well as the fact that many people with asymptomatic Covid-19 appear well but suddenly experience stroke or heart

attack—the use of anticoagulants is, I think, essential. A number of the herbs suggested for use in treating Covid-19 infections are anticoagulant and very specific for protecting endothelial cells from inflammatory damage and/or stopping clotting—*Salvia miltiorrhiza, Polygonum cuspidatum,* and *Scutellaria baicalensis* are some examples. However, I think the daily use of specific anticlotting agents is warranted for anyone—whether infected or not—for the duration of the pandemic. I suggest either lumbrokinase or nattokinase—and yes, serrapeptase will work, too, but it is very weak compared to the other two. (Lumbrokinase is 30 times stronger than nattokinase and 300 times stronger than serrapep-tase; I would suggest that only lumbrokinase be used given the excessive clotting the virus causes. The worm-derived form is strongest.)

Note: Those already on anticlotting drugs, a.k.a. "blood thinners," should avoid the use of lumbrokinase or, at minimum, approach its use with caution.

Immune Dysregulation

Nasal goblet cells, some of the first cells infected, are involved in initial interferon (IFN) responses to viral infections. But with this virus, there is considerable evidence that IFN responses are dysregulated. During early infection, as soon as the nasal goblet cells are infected, IFN responses are delayed. Later, IFN activity is often overactive, initiating highly inflammatory cytokine cascades. In the latter situation, there is strong evidence that what is called cyclic GMP-AMP synthase (cGAS) and its downstream effector STING (stimulator of interferon genes) become overactive. This same dynamic is the source of an unrelated but difficult and painful autoimmune disease called STING-associated vasculopathy with onset in infancy (SAVI). Unsurprisingly, the symptoms it causes bear a resemblance to some of the symptoms that occur during Covid-19 infections.

STING is an adaptor molecule that links sensing of foreign microbial pathogen DNA to the production of type 1 IFNs during the innate immune response. It is expressed in alveolar macrophages, bronchial epithelium, and type II pneumocytes—all SARS-CoV-2 infection sites. STING has a direct effect on endothelial cells, stimulating inflammation and initiating a coagulation cascade. This is in addition to the dynamics already in play through viral attachment to endothelial ACE-2 receptors.

STING dysregulation, caused by the SARS-CoV-2 virus, is at the root of many of the pulmonary, coagulation, and inflammation problems seen both in Covid-19 infections and SAVI. (Note: This may be related to the condition, termed multisystem inflammatory syndrome in children, or MIC, that is affecting increasing numbers of children, teenagers,

Tiny Rant: A number of people have expressed concern about upregulating and strengthening ACE-2 since the virus attaches to that receptor. Wouldn't it be better, they say, to just inhibit ACE-2 in the body completely? Why don't we just get rid of ACE-2 entirely? Then we won't get infected. Won't upregulating and strengthening ACE-2 lead to more attachment points and more infection? Well, no, it's not that simple. For one thing, there are some 40 trillion cells in the human body, a significant number of which have ACE-2 receptors on them—including fat cells. The more fat you have, the more ACE-2. (Needless to say, Americans have a *lot* of ACE-2 receptors.) Getting rid of ACE-2 receptors is simply not possible, which is a good thing since they are essential for the body to remain healthy. Without them we die. Really really fast.

Secondly and importantly, *herbs are not drugs*. Nor are they even *raw drugs*, which some phyto-semi-rationalists and reductionists erroneously call them. They are plants, which are, at root, only one thing: ecological modulators—of both large systems like the Earth and smaller ones like our bodies. They act to move systems, irrespective of size, back to health, to reestablish homeodynamis—what some people incorrectly call homeostasis (there are no static states in nature, only dynamic ones). And plants are extremely good at their job, which they have refined over several hundred million years or so.

Pharmaceuticals, which are a century old or so, are single molecules that force a change in the body of one sort or another. (They come out of a medical system whose approach to disease is based on cut, kill, or force—and now perhaps, to some extent, and very dangerously, reprogram.) They don't usually perform multiple actions. Herbs often contain hundreds of compounds that act synergistically. *Pueraria lobata* (kudzu) does not simply upregulate ACE-2. It is more accurate to think of its actions with ACE-2 as performing a modulatory and regulatory function as part of a much wider range of actions in the body (such as downregulating overactive cytokines like TNF-α and IL-1β and supporting the health and maturation of dendritic cells). It is *not* a single-action stimulant (such as a pharmaceutical) that forces ACE-2 expression, nor is it a straight suppressant, depressing ACE. You *can* compare apples and telephone poles, it just doesn't make any sense when you do.

and young adults, which was first noted in New York. They get a mild case of Covid-19, then several weeks later develop a serious multiorgan inflammation requiring hospitalization. Most recover; some do not.)

SAVI is accompanied by abnormal inflammation throughout the body, especially in the skin, blood vessels, and lungs—idiopathic pulmonary disease is a common problem for children with SAVI. There are also continual problems with blood vessels (vasculopathy) and damage to the tissues that rely on these vessels for their blood supply. The condition causes a chronic vessel-endothelium inflammation that leads to the vasculitic rash common in SAVI . . . but also seen in Covid-19 infections. This often extends to the toes and fingers, producing a condition that is very similar to what is being called "Covid toe." As with Covid toe, the rash is not limited to the toes but extends to the sole, sides, and top of the foot and is sometimes accompanied by lesions. JNK (c-Jun N-terminal kinase) inhibitors have been found to help quiet the STING-initiated, overactive IFN activity, reducing the systemic inflammation in the body. (Some plants that inhibit JNK are *Ailanthus altissima*, *Andrographis paniculata*, *Aster tataricus*, *Eucommia ulmoides*, *Forsythia suspensa*, *Glycyrrhiza* spp., *Lonicera japonica*, *Magnolia officinalis*, *Paeonia suffruticosa*, *Polygonum cuspidatum*, *Sophora flavescens*—all of which have been found to be useful for treating pulmonary problems similar to those caused by this coronavirus.)

As with Lyme infections, interfering with the production of upstream cytokines during Covid-19 infections can significantly reduce the inflammatory cascades they initiate, thus reducing the damage to the body. Xiaobing Deng, Xiaoyu Yu, and Jianfeng Pai (2020) comment that control of upstream cytokines is a promising strategy in the treatment of Covid-19, with special attention paid to the dysregulation of type 1 IFN that the virus causes early during infection. Stopping the virus-caused abnormal activity of cGAS-STING, which is a main source of cytokine overactivation and inflammation, is one potential upstream point at which to intervene. The plant-derived cyclopeptide astin C is particularly potent in accomplishing this. It's a compound from the plant *Aster tataricus* (a highly underutilized herb in the Western world), which has been used in traditional Chinese medicine for some two thousand years. The root is often used to treat lung and bronchial disease, especially chronic bronchitis and coughing. It is considered antibacterial and antifungal (with a good range of action against a

number of pulmonary pathogens), antitussive (reducing coughs), expectorant (expressing mucus out of the system), and stimulant. It is particularly good for a number of post-coronavirus problems. (This is not an herb that I have previously used or have experience with—though that is changing—but given its history of use and its ability to inhibit JNK and cGAS-STING its use with Covid-19 certainly should be considered.)

As noted, there are ACE-2 receptors on macrophages, monocytes, and lymphocytes, including T cells. This allows the virus entry into those cells, where it can then affect immune responses. There is growing evidence that, like SARS-CoV-1, this virus can also infect dendritic cells and it definitely does interfere with their maturation. By infecting a wide range of immune cells, the virus can lower or inactivate some immune responses and significantly upregulate others. Similarly to the *Borrelia* bacteria that cause Lyme disease, it is very sophisticated in modulating immune responses to infection. During early stages, it shuts down significant parts of a healthy immune response, which allows the virus to spread and infect widely divergent parts of the body more easily. (As an example, during SARS-CoV-2 infection it is common for the body to have very low levels of lymphocytes, a condition called lymphocytopenia. *Houttuynia cordata* is very good at correcting this as well as being a specific antiviral for this particular virus.) Later in the infection, the virus stimulates immune activity, thus causing more inflammation. (Inhibitors for the organism's actions on the immune system is covered a bit later on.) Some people have immune responses that do in fact quite easily stop the infection, while others, apparently very healthy, do not. No one knows why. (Reductionists continually fall back on GENETICS!, which they use about the same way that our ancestors used "the gods did it" or "it's an imbalance in the humors." The truth is they don't know.)

CD147 and Cyclophilin A

The virus has also been found to attach itself to the CD147 receptor that is present on many cells in the body. CD147 is also known as neurothelin, basigin, or, more descriptively, extracellular matrix metalloproteinase inducer (EMMPRIN) since it stimulates fibroblasts to secrete a range of matrix metalloproteinases (MMPs)—themselves a source of inflammation and cellular breakdown. (The plethora of names that all refer to

the same thing that researchers continually come up with are a constant source of irritation to those of us who use language to communicate.)

CD147 is regarded as a novel modulator of inflammatory and immune disorders and its dysregulation has been linked to the pathogenesis of such things as asthma, lung inflammation, hepatitis, myocardial infarction, ischemic stroke, and, importantly, neuroinflammatory diseases—most of which occur during Covid-19 infections.

CD147 receptors are found on olfactory and brain neurons, red blood cells, epithelial cells, endothelial cells, leukocytes, monocytes, lymphocytes, neutrophils, and platelets. It is strongly upregulated on activated immune cells, neutrophils, T and B lymphocytes, monocytes, macrophages, and dendritic cells. While the virus can use this receptor to gain entry to cells (and does sometimes do so), it appears that a more important aspect is the affinity of cyclophilin A (CyPA) for CD147 receptors.

Damaged epithelial and endothelial cells and macrophages tend to upregulate and release CyPA, and CyPA has been found to stimulate CD147 surface expression on cells. CyPA has been shown to facilitate viral replication, including that of SARS-CoV-1. CyPA, when released from cells, strongly binds to the upregulated CD147 receptors. By attaching itself to the CD147 expressed on the surface of cells and simply waiting, the virus gains access to the CyPA, which, when released from damaged endothelial and epithelial cells, seeks out CD147 to bind with. When it does so, the virus can utilize the CyPA to facilitate its reproduction. Viral load then increases substantially.

The cyclophilin inhibitor cyclosporin A has been found to inhibit the replication of coronaviruses. (*Magnolia officinalis* contains magnoloside A, which has also been found to inhibit CyPA. It is a traditional Chinese herb used to treat, among other things, lung infections and inflammation.) As well, anti-CD147 antibodies tend to inhibit the virus from attaching to host cells or using that receptor to gain entry into them. (*Scutellaria baicalensis* accomplishes this as well, in part, by downregulating CD147 expression.) Blocking CD147/CyPA interactions during in vivo studies of induced acute lung inflammation by the use of anti-CD147 mAb led to a 50 percent reduction of neutrophils within the lung tissues and airways accompanied by a similar decrease in tissue damage (Zhu et al., 2014).

CyPA is a potent proinflammatory molecule. The more that is released from damaged cells, the more inflammation that occurs in the system. The binding of CyPA to CD147 activates MAPK pathways, stimulates leukocyte recruitment, and specifically induces MMP-9 expression through ERK and NF-κB pathways, all of which play a role during Covid-19 infections. (Among other actions, *Polygonum cuspidatum* strongly downregulates MMP-9.) CyPA also induces the production of numerous cytokines, e.g., IL-1β, IL-6, and IL-8, in macrophages and monocytes and promotes the proliferation and migration of VSMC (vascular smooth muscle cells). It enhances platelet adhesion and thrombus (clot) formation and activates ERK-1 and ERK-2, NFκB, Akt, JNK, and p38 MAPK, again, all of which play a role in Covid-19 infections.

Inflammatory Cytokines

Once the virus enters the body it initiates a rapid process of replication that causes massive endothelial and epithelial death (apoptosis) and, because of the endothelial cell damage, vascular leakage. This triggers the release of "exhuberant" (as they say) proinflammatory cytokines and chemokines (known hereafter as just plain old cytokines). These include TNF-α, IL-1β, IL-6, IL-8, VEGF (vascular endothelial growth factor), MCP-1, among others. The viral infection of macrophages and lymphocytes can result as well in a type of apoptosis or cell death called pyroptosis that is by its nature highly inflammatory when it occurs. The virus doesn't generally reproduce in white blood cells but it does actively interfere with their ability to fight off the infection. (See the section on smoking, page 90, for a bit more on this.)

In addition to attaching to and infecting ACE-2 receptors, the virus can also downregulate ACE-2 and induce, as they say, the shedding of "catalytically active ACE-2 ectodomain"—these guys are great fun at parties. What this does is initiate the loss of ACE-2 function in the lungs, which tends to create acute lung injury. This loss of ACE-2 function often causes dysfunction of the renin-angiotensin system (RAS) in the body. RAS is intimately involved in modulating a number of systems in the body needed for health. As soon as ACE-2 reduction or loss occurs, general inflammation in the body increases and vascular walls become more permeable. In the lungs, loss of ACE-2 results

in more edema, leaking blood vessels, neutrophil accumulation, and diminished lung function.

Protecting and strengthening ACE-2 receptors is, I think, essential. Herbs that block viral attachment to ACE-2 linkages are *Glycyrrhiza* spp., *Scutellaria baicalensis*, *Sambucus* spp., *Aesculus hippocastanum*, *Polygonum cuspidatum*, *Rheum officinale*, and plants high in procyanidins and lectins (e.g., *Cinnamomum* spp., i.e., cinnamon). Herbs that upregulate ACE-2 are *Pueraria lobata*, *Salvia miltiorrhiza*, and *Ginkgo biloba*. ACE inhibitors (in contrast to ACE-2 upregulators) will increase the presence of ACE-2 and help protect the lungs from injury: *Crataegus* spp. and *Pueraria lobata* are specific for this. (This is part of the reason *Crataegus*, i.e., hawthorn, is good for heart health; it upregulates ACE-2 by downregulating ACE, thus increasing ACE-2 receptors in the heart, thus supporting heart health and vitality.)

To continue . . . the increase of TNF-α and IL-1β in the system stimulates the "shedding" of ACE-2, which results in less membrane-bound ACE-2 on the body's cells. This is pervasive throughout the body—the more inflammation, the more shedding. No matter the organ, when this shedding occurs, organ function decreases. (Plants that can inhibit TNF-α include *Andrographis paniculata*, *Cordyceps* spp., *Eupatorium perfoliatum*, *Glycyrrhiza* spp., *Houttuynia cordata*, *Pueraria lobata*, *Salvia miltiorrhiza*, *Sambucus* spp., *Scutellaria baicalensis*, and melatonin, not a plant but useful in this infection for a variety of reasons. IL-1β inhibitors include *Cordyceps* spp., *Eupatorium perfoliatum*, *Polygonum cuspidatum*, *Pueraria lobata*, *Salvia miltiorrhiza*, *Scutellaria baicalensis*.)

SARS-CoV-2 can, it seems, infect dendritic cells (DCs), both mature and immature. It doesn't kill them (as far as I can find) but merely stops them from maturing and thus initiating an effective adaptive immune response. DCs exist abundantly just under the epithelium layers in the lung tissue. The cytokine upregulation that infection causes makes the endothelium much more porous, allowing the virus to penetrate and infect the DCs. Upregulated IL-6 and IL-8 from epithelial and endothelial cells concentrate around the immature DCs and strongly inhibit their maturation and the priming ability that mature DCs have for the generation of active T cells. This inhibits the production of active T cells, allowing the spread of the infection. Stimulating DC maturation (*Cordyceps* spp., *Pueraria lobata*), along with inhibiting cytokines, can help prevent this.

Dysregulation of the brain ACE-2 and RAS system is intimately related to poorer cardiac function as well as dysregulated hypothalamic function, blood pressure, and autonomic system function. (This is a contributing element to the wide range of neurological effects that are being seen.)

Not to get into it too deeply, ACE-2 (angiotensin converting enzyme 2) antagonizes the actions of angiotensin II (AngII). AngII is involved in modulating immune function. When not controlled by the presence and action of ACE-2, it contributes to general and autoimmune inflammation, hypertension, organ and ventricular hypertrophy, and the decrease of endothelial progenitor cells that are necessary for vascular repair and promotes organ damage and fibrosis in the body. The less ACE-2, the more those effects occur. ACE-2 is *very* important to healthy functioning. ACE-2 is powerfully affected by the virus, so, again, the use of ACE-2 protectants and modulators that normalize function is, I think, crucial.

The extensive cytokine release in the body causes an ongoing inflammation that can attack most organs, eventually leading to organ damage and collapse. Interfering with the generation of the cytokines, which can be accomplished through a variety of herbal interventions, can substantially help the course of infection. For example, some researchers have found that simply reducing IL-6 during a Covid-19 infection will reduce inflammation, making the disease less acute, and enabling a better long-term resolution. That is why the arthritis drug tocilizumab, which inhibits IL-6, has been found of use in treating acute Covid-19 infections.

IL-6 and IL-8 are two of the more important cytokines to inhibit as part of Covid-19 treatment. IL-6 plant inhibitors include *Andrographis paniculata, Isatis* spp., *Pueraria lobata, Salvia miltiorrhiza, Scutellaria baicalensis,* and melatonin (which is often a constituent in many medicinal plants). IL-8 inhibitors include *Cordyceps* spp., *Isatis* spp., *Polygonum cuspidatum.* (And just to note: Melatonin has good application in this disease, not only as an anti-inflammatory but also because, among other things, it helps reduce anxiety and promotes sleep.)

A Further Comment on RAS Dysregulation

When the virus disrupts the RAS system, it also dysregulates the mechanisms for regulating a chemical called bradykinin. Bradykinin levels increase in the body, causing a "bradykinin storm." A number of researchers now feel that some of the more serious effects of a Covid-19 infection come from this. In concert with a cytokine storm, the results can be deadly.

As bradykinin builds up in the body, vascular permeability substantially increases. In other words, the blood vessels become leaky. This causes more fluid to build up in the lungs. In addition, the virus appears to increase production of hyaluronic acid (HLA), which, when combined with the fluid leaking into the lungs, creates a kind of hydrogel. As researcher Daniel Jacobson notes, "It's like trying to breathe through Jell-O" (Thomas Smith, 2020). Because the alveoli are filled with this gel, even ventilators are unable to get more oxygen into the blood.

This increase in bradykinin also affects the heart, causing alterations in blood pressure and heart rate. Brain inflammation increases, producing neurological symptoms. Blood potassium levels increase, cough and fatigue occur, smell and taste are altered or suppressed.

Polygala tenuifolia inhibits bradykinin-mediated effects on the body and reduces levels of bradykinin in the body. It is specific for bradykinin-mediated pain and inflammation. *Sophora japonica* can help protect the brain from bradykinin-mediated inflammation and vascular effects.

Note: During Covid-19 pulmonary infection the use of any *Echinacea* species should be avoided, as continuing, large doses of it will increase HLA levels in the body.

The Heart

As mentioned earlier, the virus does infect cardiac cells via their ACE-2 receptors and thus damages heart tissues, including its muscle tissue. Some people so affected have no respiratory symptoms at all and present at the hospital solely with cardiac problems such as sudden heart attack. At first glance it seemed that perhaps 10 percent of those infected with Covid-19 suffered cardiac complications. This is now uncertain; it may be far greater. Permanent heart damage is apparently occurring in more people than was at first realized. Many asymptomatic people or those

with very few symptoms are finding that even though they are apparently well they now have underlying myocarditis (inflammation of the heart).

Two studies in Germany raise serious issues for long-term heart problems in those who have recovered from Covid-19. In one, MRIs of 100 people who had recovered from the infection were compared with 100 similar people who had not been infected. Seventy-eight of the infected were found to have signs of structural damage to the heart, 60 of them had myocarditis. All of them were relatively young. (A second study on 39 people who had died from the infection found that 24 of them had active virus in their heart tissue.) In essence, many people who recover from the disease are going to have long-term heart problems from the infection. The heart damage can become serious when the body is put under muscular stress. Several athletes have died after returning to their sport simply due to pressure on the heart from extreme exertion. Examinations of others who seemed well have found chronic heart inflammation where there previously was none.

During cardiac infection the initial manifestation is "an increase in high-sensitivity cardiac troponin 1 (hs-cTnl) levels" (Y.-Y. Zheng et al., 2020). As the damage spreads, median creatine kinase levels rise to double the levels of those without cardiac infection. In a more perfect world everyone infected with this virus would be tested for those elevated cardiac biomarkers. (They aren't.)

Herbal interventions are very specific for preventing this kind of damage during infection. In general, during the pandemic it is a good idea to take heart adaptogen and tonic hawthorn (*Crataegus* spp.) as part of a daily preventive regimen. The herb most specific for the damage the virus causes is *Salvia miltiorrhiza*. It is significantly more effective if combined in a one-to-one ratio with *Pueraria lobata* (L. Wu et al., 2007). A combination of *Paeonia suffruticosa* and *Salvia miltiorrhiza* has also been found to be effective (H. Li et al., 2016). *Salvia miltiorrhiza* is a truly important medicinal in the treatment of inflammatory diseases such as Covid-19. It has a long history of use in China for the treatment of systemic disease, including reversing or treating adverse impacts in most organs of the body including the heart. It is effective for inhibiting increases in troponin and creatine kinase—again, not as a suppressor but as a modulator of function. The herb promotes blood circulation, inhibits platelet aggregation, protects endothelial structures, is anticoagulant, antihypertensive, antithrombotic, antiallergenic, and

strongly protective of the kidneys. It is a potent cytokine adaptogen—reducing any cytokine levels that are too high, increasing any levels that are too low—another way to think of it is as an immune-response adaptogen. It is strongly anti-inflammatory, protects Golgi structures, is neuroprotective, restores mucosal integrity in mucosa-infected cells, is highly protective of the spleen—enhancing its immune functions—and has shown remarkable effectiveness in the treatment of lung disease. In short, a truly world-class systemic modulator for inflammatory diseases of any sort.

The world's best herbal monograph (on *any* herb) is the three-volume (1,800 pages total) compilation by Xijun Yan (editor): *Dan Shen (Salvia miltiorrhiza) in Medicine* (2015). It covers every possible use of the herb and looks at both historical use and its outcomes in clinical trials and in laboratory study. It makes any other herbal monograph in existence look paltry and rather shamefaced in comparison.

Scutellarin and baicalin from *Scutellaria baicalensis* are also particularly effective in treating and preventing heart damage from the virus. Scutellarin prevents the increase of cardiac troponin (by correcting or preventing the underlying damage). Baicalin inactivates creatine kinase. Specifically the herb has a broad range of cardiovascular actions: It promotes vasodilation, protects against ischemia/reperfusion, is anti-inflammatory, anticoagulatory, antithrombosic, protects endothelial integrity, protects the myocardia, stops cardiac remodeling, and possesses anti-arrhythmic actions. It is also a strong systemic antiviral herb, specifically so for this particular organism. It has a long use in China for treatment of blood circulatory problems and cerebral insufficiency. (Quercetin and *Polygonum cuspidatum* will also inactivate creatine kinase.)

Additionally, a Chinese blend called QiHong (not findable as a premade formulation on the internet as far as I can determine) is very specific for preventing viral myocarditis. It is a blend of equal parts of *Astragalus* spp., *Rhodiola rosea, Sophora flavescens.*

One final thing: L-malic acid has been found to be extremely low in the infected; levels become progressively lower as severity increases. L-malic acid is an essential amino acid in the body when the immune system is struggling with any type of systemic inflammation. This amino acid is rapidly consumed during inflammatory states in order to provide energy and materials for the proliferation of and phagocytosis capacities of immune cells. Supplementing L-malic acid is strongly

suggested, especially during more serious infections. (It can cause diarrhea in high doses.)

Given that long-term heart damage is likely, an MRI of the heart is indicated for anyone who has been infected and subsequently recovered. Use of heart-supportive herbs is highly suggested.

A Brief Comment on Smoking and Covid-19

Despite a great many media articles early in the pandemic that insisted that smokers who contracted the new coronavirus would suffer worse outcomes than nonsmokers, such has not generally been the case. (Initiate hair pulling by prohibitionists.) As Lippi and Henry (2020) comment: "In conclusion, the results of this preliminary metaanalysis based on Chinese patients suggest that active smoking does not apparently seem to be significantly associated with enhanced risk of progressing towards severe disease in COVID-19." Some researchers are speculating that since smoking reduces macrophage activity it interferes with the systemic inflammatory processes the virus initiates. As Yang and Chen (2018) note:

> A study by Chen et al demonstrated that in smokers' alveolar macrophages, there is a decrease of proinflammatory cytokines including TNF-α, IL-1β, IL-6, IL-8, and reduced TLR2 and TLR4 signaling as a result of impaired activation of NF-κB.

These are in fact some of the most active of the cytokines during Covid-19 infection, which explains why smokers generally have a better outcome *in acute infections* than nonsmokers.

Other researchers speculate that because nicotine has definite effects on the RAS/ACE system (modulating its actions), *that* is the reason for smokers' better outcomes during infection. As well, nicotine actually prevents acute lung injury in animal ARDS models and has immune modulating actions. (To stop the run on nicotine patches the French government prohibited the sale of over-the-counter patches until the pandemic subsides. Nevertheless at least one hospital in the EU issued nicotine patches to all its medical workers . . . always fun to see a prejudice defeated by a deeper prejudice or, in this case, a deeper fear.)

There is, inevitably so, continued conflict on this issue between researchers who have a strong prohibitionist orientation and those who are just looking at the data. Only time will reveal whether smoking

reduces negative outcomes during acute infection but at this point in time, it appears that it does due to its lowering the activity of certain cytokines by inhibiting macrophage activity.

Post-Coronavirus Syndrome

There is a growing recognition that many people who have been infected by the virus continue to report severe symptoms for months (and perhaps much longer) after their initial infection. These are now being referred to (most commonly) as "long haulers," that is, people who "should" have recovered but have not. And most of them are relatively young—three in five are between the ages of 30 and 49. (Some continue to test positive for the virus, others do not—false negatives are common in around 30 percent of those tested; there is, as yet, no truly reliable test.) Somewhere between one in 20 and one in 10 people are reporting a long-term illness. (Newer research is revealing that as many as 30 percent of the asymptomatic may develop long-haul symptoms.) Some have been experiencing debilitating symptoms for as long as 12 months; no one yet knows how long it will be until they resolve . . . if they ever will.

As Jorge Mercado, MD, comments, "Reports on potential for long-term consequences have been broad, from blood clots to heart damage, lung damage, and neurological symptoms. While some conditions may be reversible over time, there is growing evidence that some long term effects from COVID-19 may be irreversible" (Seaton, August 7, 2020).

Margot Gage, an epidemiologist, was infected early in the pandemic, as was her family. Unlike them, she became seriously ill. Five months later, she still has brain fog, seizures, and extreme fatigue. She still can't work. Luckily she found a responsive and knowledgeable physician . . . most have not been so lucky (Seaton, August 5, 2020).

As with Lyme disease, many physicians believed (as some continue to do) that long-haul coronavirus problems do not exist. In other words, that there is no post-coronavirus syndrome. This is, of course, infuriating to those who suffer from it. As Fiona Lowenstein (2020) comments:

> Since contracting Covid-19 in March [2020] and launching a virtual support group for other patients, I have witnessed first hand the limitations of expert advice for a novel pandemic, and the need for patients to become their own experts and advocates. When my own Covid-19 case morphed and dragged on for months, I found no expert advice that applied to my situation. . . . Connecting

to thousands of other patients helped me discover that my symptoms and "long-haul" condition were not unusual.

It wasn't until July 24, 2020, that the CDC finally issued a statement acknowledging that up to a third of those infected with the virus were suffering long-term problems. Prior to this physicians routinely discounted their patients' experiences. (Again, this is an incredibly common experience for many people who enter the medical system, irrespective of the condition they have.)

Carol Holguin, for example, was still experiencing Covid-19 symptoms 130 days after initial infection. Medical providers continually dismissed her when she told them of her condition—even in the early stages of infection.

"'I'm having trouble breathing,' I told the nurse. She inquired about my other symptoms, which included vertigo and light-headedness. But I'd never had a fever above 100.4, so she said I couldn't be tested. Then she told me it sounded like I had anxiety." (Regrettably, it is extremely common for licensed medical technologists with no depth training in psychology to diagnose psychopathology instead of listening to their patients.) As Holguin notes, being refused treatment was a "turning point because I took my health into my own hands." Still, her symptoms continued, often worsening.

Suddenly, months later, she couldn't breathe. Her husband called the EMTs. She told them she was positive for Covid-19 and was sure it was the virus acting up again "but they didn't listen." Later, she comments, "I asked the cardiologist if this could be Covid-19, but he didn't even acknowledge the question" (Holguin, 2020).

As Ed Yong reports in *Atlantic* magazine (June 4, 2020), another patient, Hanna Davis, told of similar dismissive behavior. Yong relates, "Davis described her memory loss and brain fog to a neurologist, who told her she had ADHD. 'You feel really scared: These are people you're trying to get serious help from, and they don't even understand your reality,' she said. Vazquez [another of the infected] said her physicians repeatedly told her she was just having panic attacks. . . . Athena Akrami, a neuroscience professor at University College London, said two doctors suggested she was stressed, while a fellow neuroscientist told her to calm down and take antidepressants." As Yong comments, "Well before the pandemic, the health care profession had a long history of medical

gaslighting—downplaying a patient's physical suffering as being all in their head, or caused by stress or anxiety."

When these kinds of long-term problems occur—which generally points to an ongoing chronic condition—medical practitioners often separate into different cliques, each promoting or defending their favorite explanation or theory. (Extensive name-calling is common.) But those who continue to struggle with debilitating symptoms are the ones who suffer for it. As Clare Rayner, a consultant in occupational medicine in the UK, says, "There's pathology here that's not being investigated." Or as Timothy Nicholson, MD, puts it, "Lots of people feel that their symptoms are not believed" (Gross, 2020). (This is because they are not.) Note: One of the better articles on long haulers (as I was finishing this update) is "What If You Never Get Better from Covid-19?" (Velasquez-Manoff, 2021).

As I mentioned, these kinds of responses have been common in the Lyme community with which I've worked for over 15 years now. Many people in this community have long-term post–Lyme disease symptoms, which are still routinely discounted. (The most common response from physicians is some form of "it is all in your head" and the prescribing of some form of psychotropic drug, usually an antianxiety medication.) Because so few physicians understand or are responsive to their struggles, both the Lyme and Covid-19 community have formed support groups (easily found via the internet and on Facebook). This kind of support can make the journey to health far easier than it would be otherwise. Both groups are focused on taking back control of their health care, the journey to health, and are exploring a great many interventions to reduce or eliminate the symptoms they experience. None of them are willing to accept that there is nothing that can be done.

The most common symptoms that accompany post-coronavirus syndrome are severe fatigue, headaches, trouble breathing, and a recurrent cough. But there are a great many more than that . . . over 80 symptoms have now been reported. Things are quite a bit more complicated than they appeared to be when the virus was first being treated. There are not just the dead, the sick, and the recovered. There are the (potentially) hundreds of thousands, or millions, who are still struggling, some of whom may take years to recover, some of whom may never do so.

Post-Coronavirus Syndrome: The Symptoms

Many of the people with long-term problems have tested negatively for antibodies. (Again, no one knows why . . . maybe they can't make them or maybe the antibodies fade quickly.) One of the main fears for many of them is reinfection, which could make things much worse when added to the problems they still have. (The Covid-19 protocol outlined on page 105 can help prevent, or significantly reduce the intensity of, reinfection.)

The long-term symptoms people experience are often cyclical in nature—they come and go, much like relapsing fever infections such as malaria. (With malaria, the infection recurs as new generations of malarial parasites are born, generally on a very specific schedule.) The recurrence can be mild or strong. And to make things worse, the recurrent symptom may not be the same each time. For some people, the body seems to cycle through a number of symptoms over and over again.

Fatigue is very common. For many it is severe and debilitating, so much so that a month in bed every so often is not uncommon. This is being likened to chronic fatigue syndrome (also known as myalgic encephalomyelitis or ME) though (of course) arguments are occurring over what *real* ME is and is not. (No, no, it is the tiny mark on the corner of the stamp that makes it a true 1942 Eagle, not the smudged ink at the top.) It reminds me of the Greek scholar who spent 40 years proving that *The Odyssey* wasn't written by Homer but by another Greek of the same name. No matter the medical arguments, what people are experiencing is an unremitting fatigue that is indeed chronic. They just want it to go away.

(Supporting mitochondrial health is essential in chronic fatigue-like conditions as they are the source of energy in all our cells. Also important is the use of adaptogenic herbs, which will increase energy and help the body respond to long-term chronic conditions and the stress they bring.)

Shortness of breath is another very common problem. It can be periodic or continual and ranges from feeling slightly out of breath to the breathlessness you feel after you have run a race. But it can also present as intense episodes of "air hunger" where the body just can't seem to get enough oxygen. It feels as if every cell in the body is starving for air simultaneously and is, not surprisingly, accompanied by extreme anxiety and panic.

SYMPTOMS THAT MAY OCCUR DURING INFECTION AND ARE ALSO COMMON FOR THOSE SUFFERING POST-CORONAVIRUS SYNDROME

SYSTEM	SYMPTOMS
Respiratory system—upper	Loss or alteration of smell, nasal congestion, sneezing, sore throat (severe or mild), sinus pain (severe or mild), postnasal drip, sinus infections
Respiratory system—lower	Persistent uncontrollable cough, dry cough, cough with mucus, extreme mucus production by lungs, coughing up blood (hemoptysis), shortness of breath (severe and mild), air hunger, hypoxia, wheezing, rattling breath (a.k.a. crackling or Velcro sounds), lung burn (severe or mild), pneumonitis, pneumonia, cessation of breathing during sleep, tightness in chest (severe or mild), chest pain, fibrosis, emphysema, chronic obstructive pulmonary disease (COPD), idiopathic pulmonary fibrosis, bronchiectasis, recurrent lung infections
Temperature regulation	Repeated shaking with chills, continual or recurring high or low fever, hot flashes, chills, sweats
Ears	Earaches, tinnitus (severe and mild), otitis media, hearing loss, eustachian tube dysfunction
Eyes	Sensitivity to light, eyestrain, blurry vision, floaters, pink (red) eye
Oral/mouth	Loss of taste, rash, tooth loss, tooth fragility/chipping, teeth turning gray, more cavities, sensitive gums, salivary gland ectasia, temporomandibular joint abnormalities, facial pain, masticatory muscle weakness
Liver	Elevated liver enzymes, fibrosis, cholangiopathy (bile duct damage), hepatitis
Reproductive system	Low libido, damage to reproductive system (male and female): menstrual irregularity, severe cramping, infertility, erection problems, bilateral orchiditis, testicle pain, hypogonadism, extended menstrual bleeding, cessation of menstruation, early menopause
Musculoskeletal	Fatigue (severe and mild), muscle pain (myalgia, severe or mild), joint pain (arthralgia, severe or mild), body aches (severe or mild), muscle spasms, body shaking, hand tremors, feeling of electricity zapping through body, tingling/numbness in extremeties, extreme muscle weakness

(Chart continues on the next page)

(Continued from previous page)

SYSTEM	SYMPTOMS
Kidney/urinary	Frequent urination, proteinuria, hematuria, elevated serum creatine, elevated urea nitrogen, extreme thirst, kidney injury (acute or mild), fibrosis
Skin	Dry skin, rash, prominent veins, easy bruising, acne flare-ups, extreme skin sensitivity (to touch of any sort), tingling, red/purple lumps (itching or not)
Heart and blood	Myocarditis (inflammation of heart muscle), blood clotting (often extensive), cardiomyopathy, generalized inflammation throughout system, fibrosis, palpitations, heart attack, low pulse rate, fast pulse rate, elevated heart rate (tachycardia), postural orthostatic tachycardia syndrome (POTS, dizziness or fainting upon standing suddenly)
Gastrointestinal tract	Diarrhea (mild or severe), cramping (mild or severe), loss of appetite, nausea, vomiting, lower esophagus burning
Neurological	Brain fog, trouble concentrating, short-term memory loss, anxiety (severe and mild), panic attacks, depression (severe or mild), headache (severe or mild), malaise, vertigo, dizziness, lucid dreaming, mood swings, seizures, stuttering, various psychiatric disorders, muscle weakness, nerve pain, depressed levels of consciousness, tingling/fizzing sensations throughout body, hair and scalp pain, confusion, a sense of not being one's self, long-term mental impairment, anxiety, insomnia, paralysis, severe psychosis, delirium, hiccups, stroke, encephalopathy (general), parainfectious or septic encephalopathy with psychosis or delirium, meningoencephalitis, limbic encephalitis (in the thalami, medial temporal regions, and pons), microbleeds, acute hemorrhagic leukoencephalitis, postinfectious brain stem and cortical autoimmune encephalitis, parainfectious involvement of central and peripheral nervous system, brachial plexopathy, acute myelitis, quadriparesis, facial paresis, Guillain-Barré syndrome
Miscellaneous	Hair loss, swollen lymph nodes, insomnia (severe or mild), low immune function, Sjögren's syndrome, syncope, compulsive water drinking

You can find the rest of the extended symptom list in the chart that begins on page 95. This is taken from a May 2020 analysis of a survey by the group Patient Led Research for COVID-19, which you can find online (see also Assaf, 2020), and from a number of other sources such as news articles, journal papers, and personal communications. There may be others that I have missed. Treatment suggestions for the majority of these are included in the extended protocol section that follows the main protocols suggested for Covid-19 treatment.

Herbal Interventions for SARS-CoV-2 Prevention and Treatment

Here is the rationale underlying my suggested protocols:

My approach in the treatment of systemic inflammatory infections has always been to find plants that will counteract the actions of the organism involved—in this instance, SARS-CoV-2—then to cross-correlate those with each other to find the plants that are active in the most categories that will inhibit the infection and its cytokine cascades, and that have effectiveness for the symptoms the disease causes, and have a long historical record of use for treating such conditions. To find these I extensively research hundreds, sometimes thousands, of scientific and research journals and articles. I then look at both historical and contemporary use, which also involves researching a great many sources. The herbs chosen also have to be relatively easy to find.

The herbs I suggest here are not the only ones that can help during a Covid-19 infection; there are scores that will do so, many of which are listed in this material. However, that being said, I would strongly suggest that *Salvia miltiorrhiza* ***not*** be eliminated from the protocol under any circumstances (unless you absolutely cannot find a source for it or experience side effects from its use). In my opinion, given its effects in counteracting so many of the actions and impacts the virus has, it is crucial to successful treatment of this pathogen.

As you can tell from the list of herbs that affect various aspects of the virus, its infection, and its inflammation strategy, there are a number that are active in multiple areas, such as *Andrographis paniculata, Houttuynia cordata,* and *Polygonum cuspidatum*. These herbs can be blended in various ways to create your own protocols. Personally, I would always include *Isatis* spp., *Salvia miltiorrhiza, Scutellaria baicalensis,* and *Pueraria lobata*. I feel they are just too important in treating this infection.

Finding the Herbs

Some herbal companies are making the blends I suggest in these protocols; you will just have to look around online or ask to find them. Because several (insert *strong* expletive) herb companies utilized my name (without authorization) and quoted some of my comments on treating coronaviruses with herbs in order to increase their sales, the FDA and FTC began making house calls on them . . . to their dismay. So, despite a senior White House official touting unproven remedies (injecting or drinking bleach?) that are seriously dangerous, the government became quite upset with herbal companies making claims and made it a priority to shut them down. Thus the companies who are still making blends based on these suggestions have become, let us say, shy. The blends are out there, you just might have to ask if it is not listed on their websites.

Sources: You can generally find any herb or tincture you might need online (search for the botanical name and/or common name of the herb), and especially Etsy, which is home to a good number of small herb companies that are selling herbal tinctures you will not find anywhere else. They are often far better (as well as cheaper) than those made by huge corporations. Please see Sources of Supply (page 454) for the herb and tincture suppliers I would recommend.

If you cannot find the protocols preblended, you can blend them yourself. Just buy the individual herbal tinctures and mix them together. (To clarify: if I say three parts of one herb, buy 3 ounces, if I say one part of another herb, buy 1 ounce, then blend them together in that ratio.)

Note: The protocols I suggest for Covid-19 rely heavily on herbal tinctures. Some people tend to avoid tinctures due to their alcohol content. However, I absolutely believe that avoiding the use of tinctures will

not be beneficial with this infection. I have not found glycerin tinctures to be strong enough. *I do not have an alternative protocol for those who wish to avoid alcohol intake.* Please be aware that the amount of alcohol in tinctures is minimal; much of the content is water.

Children's dosages: A child's dose can be found by dividing the child's weight (in U.S. pounds) by 150 or 160. Thus if your child weighs 40 pounds, give them one-fourth the adult dose; if they weigh 50 pounds, give them one-third the adult dose.

And as always: If any adverse symptoms appear or if you feel something is off, *then stop taking the herbs*. Remember: You are the one who lives in your body, so you are the best person to determine whether something is working for you or not and if it feels right to your body when you take it.

You are the only one who truly knows how health feels to you. Always pay attention to that and never settle for less *no matter what any particular health "expert" tells you (including me).*

Overall, in my opinion, the most effective herbal approach to SARS-CoV-2 addresses three different situations: 1) reducing the chance of infection; 2) treatment of active infections; 3) treating post-coronavirus syndrome. Here are my suggestions.

Reducing the Chance of Infection

Besides all the endlessly cited (and now tiresome) admonitions about hand washing, masks, and self-isolation (and repeated ad nauseam by too many medical practitioners of various sorts, including herbalists), actively supporting a strong immune system is the best place to begin. Secondly, I think the daily intake of a systemic anti-inflammatory such as mangiferin or Japanese knotweed root, an anticoagulant and fibrinolytic agent such as lumbrokinase, and L-malic acid will help the system be prepared if an active infection does occur. Thus:

Pre-infection immune tincture formulation: *Eleutherococcus senticosus* (2 parts), *Astragalus* spp. (2 parts), *Cordyceps* spp. (1 part), *Rhodiola* spp. (1 part), *Glycyrrhiza* spp. (1 part). Dosage: 1 teaspoon 3x daily.

Systemic anti-inflammatory: The best I know of is a formulation of *Mangifera indica* standardized to 60 percent mangiferin. Its anti-inflammatory actions are *very* specific for the kinds of inflammation seen in damaged lungs and other organs. There are a great many very good studies on mangiferin and its actions in various organs for the treatment of systemic inflammation. The only good source in the United States at this time (that I know of) is Green Dragon Botanicals (https://greendragonbotanicals.com). Dosage: 200–600 mg 3x daily. Japanese knotweed root is also very good, especially since it stabilizes and protects endothelial structures: 1 teaspoon tincture 3x daily. (This herb is good for many problems that aging bodies experience since it is so high in resveratrol; no matter what is going on with you it is a good food-grade herb to take daily.)

Lumbrokinase: 600,000 IU (a.k.a. 40 mg) in the morning and again in the evening.

Taffix: Taffix is an Israeli-developed nasal spray, a powder, which, upon inhalation, forms a gel that covers the nasal mucosa. It has been found to give 4 to 5 hours of protection from viral infection. In one trial it significantly reduced Covid-19 infections in crowded, unmasked groups of people. It's legal for sale in the EU, with an attached claim that it reduces Covid-19 infections. It is not, as of this writing, available for sale in the United States, though it can be ordered from Israel through eBay. The spray has been found in lab studies to be 99 percent effective in blocking viral attachment to nasal membranes and is apparently effective (though to what extent is not yet known) against coronavirus variants. Given the rapidly emerging number of variants, I think it warrants serious consideration as an adjunct protection, in addition to masking, if you are going to be in crowded situations where participants may be unmasked.

Melatonin: Melatonin has emerged as a potentially crucial adjunct to helping prevent, reduce the severity of, and treat Covid-19 infections. One study showed that intubated patients who were given melatonin had "significantly" higher survival rates. As well, those taking melatonin have been found to have "significantly lower odds" of developing a Covid-19 infection, "much less dying of it" (Hamblin, 2020). Because insomnia is a very common symptom for the infected as well as those suffering post-coronavirus syndrome, the supplement is of additional

benefit; sleep disruption appears to be a consistent problem in all groups. There is also early research showing that the coronavirus can possibly be blocked by melatonin. Healthy sleep is, as well, essential for the body to deal with the infection, especially its damage to the brain. Dosage and form: I prefer liquid melatonin, which I take nightly (30 ml) when I get in bed; it seems to work better for me than tablet or capsule forms. The preeminent researcher on melatonin, Russell Reiter, takes 70 mg daily. Note: Since the supplement is used to promote sleep, taking it at bedtime is highly suggested, rather than earlier in the day.

And yes, you can use nattokinase or serrapeptase in place of lumbrokinase. However . . . lumbrokinase is 30 times stronger than nattokinase and 300 times stronger than serrapeptase. Because the clotting during Covid-19 is so severe, I think lumbrokinase the best approach. Serrapeptase has other functions that make it useful during infection (despite it not being a very good fibrinolytic agent): It modulates temperature fluctuations in the body, relieves sinus pressure and inflammation especially during infection, degrades fibrin (but not very well), breaks down mucus in the lungs, helps break down circulating toxins and cellular debris, is generally anti-inflammatory, and is especially good for helping relieve swelling and minor pains in the body. It is a very useful adjunct for lung conditions such as COPD. Nattokinase is best used for mild hypercoagulation problems and to break down fibrin in the body (including the lungs), while lumbrokinase is best for severe hypercoagulation problems and, as well, breaking down fibrin. Both enhance circulatory health. (Note: Some people think that earthworm-based forms of lumbrokinase are better than its synthetic, chemically produced forms. The best of these seems to be Canada RNA brand but it is very expensive; the dosage is half of the synthetic forms.)

Caution: Nattokinase and lumbrokinase should be used with caution if you are taking pharmaceutical "blood thinners."

Treatment of Active Covid-19 Infection

What is needed are plants that have the following actions:

1. **Specific antiviral action for the SARS-CoV group of viruses.** The strongest found so far are *Scutellaria baicalensis* (root—a potent systemic antiviral herb), *Isatis* spp. (root and leaf), *Houttuynia* spp. (leaf), *Lycoris radiata* (extremely potent but not easy to find), and the essential oil of bay laurel (*Laurus nobilis*—very strong as well). These are followed by *Glycyrrhiza* spp. (root), *Forsythia suspensa* (the fruit), and *Sophora flavescens*. *Lonicera japonica* and *Polygonum cuspidatum* are effective as antivirals for coronaviruses as a whole but have not, to my knowledge, been tested against the SARS group.
2. **ACE-2 interventions,** meaning herbs that:
   - Protect ACE-2 by blocking viral attachment. Specific for this are *Glycyrrhiza* spp., *Scutellaria baicalensis*, *Sambucus* spp., *Aesculus hippocastanum*, *Polygonum cuspidatum*, *Rheum officinale*, plants high in procyanidins and lectins (e.g., *Cinnamomum*) and luteolin.
   - Upregulate and protect ACE-2 expression, increase its activity (especially in the aged), and lower AngII. Herbs specific for this are *Pueraria lobata*, *Salvia miltiorrhiza*, *Ginkgo biloba*.
   - Inhibit ACE (in contrast to upregulating ACE-2) to increase the presence of ACE-2 and help protect the lungs from injury. *Crataegus* spp. and *Pueraria lobata* are specific. Remember: These are not drugs, they are *modulators*, and they do many other things besides this.
3. **Modulation of cytokine responses,** in general (*Salvia miltiorrhiza*—a cytokine adaptogen) and in specific: Plants that can inhibit TNF-α include *Andrographis paniculata*, *Cordyceps* spp., *Eupatorium perfoliatum*, *Glycyrrhiza* spp., *Houttuynia cordata*, *Pueraria lobata*, *Sambucus* spp., *Scutellaria baicalensis*, *Salvia miltiorrhiza*, and melatonin, not a plant but useful in this infection for a variety of reasons. IL-1β inhibitors include *Cordyceps* spp., *Eupatorium perfoliatum*, *Polygonum cuspidatum*, *Pueraria lobata*, *Salvia miltiorrhiza*, *Scutellaria baicalensis*. IL-6 inhibitors include *Andrographis paniculata*, *Isatis* spp., *Pueraria lobata*, *Salvia miltiorrhiza*, *Scutellaria baicalensis*, and melatonin. IL-8 inhibitors include *Cordyceps* spp., *Isatis* spp., *Polygonum cuspidatum*.

4. **Protection for endothelial cells.** *Polygonum cuspidatum, Salvia miltiorrhiza, Scutellaria baicalensis.*
5. **Protection for the spleen and lymph nodes and strengthening effects for the lymph system.** *Bidens pilosa, Galium* spp., *Salvia miltiorrhiza, Scutellaria baicalensis.*
6. **Protection against damage for the lungs, heart, kidneys, and brain.** The herbs already suggested will accomplish this for most of the organs without adding anything else. There will be additional suggestions in the extended protocol (see page 109). However, during active infection, to protect the kidneys, regular consumption of a strong nettle (*Urtica dioica*) infusion (see page 132) along with a tincture of nettle seed is highly recommended. As well, since heart damage is being found in a large number of those infected by the virus (in those with and without symptoms, in the young as well as the old), plants specific for minimizing or preventing that damage—hawthorn (*Crataegus oxyacantha*) and astragalus (*Astragalus* spp.)—are, I think, essential.

During active infection, continual use of standardized *Mangifera indica* at a higher dose, a higher dose of lumbrokinase (or nattokinase), and L-malic acid may also help protect the organs.

The use of both a nebulizer and a steam inhalant is strongly suggested for infection in the lungs as well as the use of plants that can stimulate lymph drainage from that organ (see page 106).

Use of the Protocol during Pregnancy

The majority of herbs in the protocol on the facing page appear safe, and historically have been used, during pregnancy. However, I would not use the protocol during the first trimester. Afterward, the following caveats apply:

Licorice: Can be used but with caution. Heavy exposure during early pregnancy (up to 38 weeks) has been found in a Finnish study to increase the likelihood of early-term birth. There is nothing to suggest its use in late pregnancy in small doses is unwarranted, and nothing I can find indicates that its use as a minor component in a blended formulation is unsound during pregnancy. It is ⅛ of the formulation, a relatively minor and certainly not heavy use of the herb.
Rhodiola: Two mouse studies found it to be very mildly toxic if taken during early pregnancy. Other found it safe for long-term use during pregnancy. I can't find any adverse reports from its traditional use for over 1,000 years; nevertheless, if concern is present, eliminate this herb.
Bidens pilosa: It's a weak uterine stimulant and may be used depending on dosage. However, I would avoid it.
Nettles: There are some mixed concerns regarding nettles, but I am not sure they are warranted. It has been used for centuries during pregnancy for nutritional support, like many greens. However, some recent studies indicate it has uterine activity despite the fact that there have been no reports of adverse events.
Lumbrokinase, nattokinase, or serrapeptase: No data on adverse events, but due to their anti-clotting actions, I would avoid them during pregnancy.
L-malic acid: I would avoid it.
Bay laurel essential oil (used for an active lung infection; see page 106): The literature is confusing. Bay leaf is commonly used in food preparation; there are no contraindications for its use in pregnancy. However, most lay and a few research papers recommend that the essential oil not be used in pregnancy. I can find no definitive reasons for this nor any reasons why inhalation of 1 to 2 drops of the essential oil in boiling water on the stove would be contraindicated. However, there is not enough clear data for me to determine safe use during pregnancy.

Core Protocol for an Active Infection

This protocol is composed of three tincture formulations and some suggested supplements. They should *all* be taken at the first signs of infection, and they should be continued for 2 weeks after the cessation of symptoms; otherwise the symptoms may recur.

1. Antiviral tincture blend formulation: *Scutellaria baicalensis* (3 parts), *Isatis* spp. (2 parts), *Pueraria lobata* (2 parts), *Glycyrrhiza* spp. (1 part). Dosage: 1 teaspoon 3x daily at onset; if infection becomes acute (i.e., more intense), 1 teaspoon 6x daily. The more serious the infection, the higher the dose. Note: *Houttuynia cordata* (2 parts), which is a very good antiviral for this organism, can also be added to the blend if desired or substituted for *Isatis* spp.
2. Immune tincture formulation: *Cordyceps* spp. (3 parts), *Eleutherococcus senticosis* (2 parts), *Rhodiola* spp. (1 part), *Astragalus* spp. (1 part). Dosage: 1 teaspoon 3x daily at onset; if infection becomes acute, 1 teaspoon 6x daily.
3. Cellular protection/cytokine modulation/spleen and lymph support tincture formulation: *Salvia miltiorrhiza* (3 parts), *Galium* spp. (2 parts), *Bidens pilosa* (1 part). Dosage: 1 teaspoon 3x daily at onset; if infection becomes acute, 1 teaspoon 6x daily.
4. *Urtica dioica* (nettle): 1 quart of infusion (see page 132) daily, plus ¼ teaspoon of nettle seed tincture 3x daily. (This is good for you for many reasons but with this infection it will help your kidneys stay healthier than they would without it. It may, under some circumstances, help prevent or allow recovery from dialysis, especially in the very early stages of kidney damage.)
5. *Crataegus oxyacantha* (hawthorn): 1,800 mg 2x daily.
6. *Mangifera indica* capsules, standardized to 60 percent mangiferin: 600–1,000 mg 3x daily (Green Dragon Botanicals brand: https://greendragonbotanicals.com).
7. Lumbrokinase (or nattokinase): 600,000 IU (a.k.a. 40 mg) 2–3x daily (and please use an oximeter to check blood oxygen levels daily).
8. L-malic acid: 600 mg 3x daily.
9. Probiotic: 1 capsule daily.
10. Vitamin D_3: May possibly be of use in reducing the severity of infection, especially for those over 55. Dosage: 10,000 IU daily.

To Address Active Lung Infection

In addition to the core protocol, I suggest three things to treat Covid-19 lung infection: bay laurel (*Laurus nobilis*) essential oil as a steam inhalant; the use of a nebulizer as outlined below; and guaifenesin (Mucinex or its equivalent).

BAY LAUREL ESSENTIAL OIL

This essential oil is potently antiviral for SARS viruses, and it can be used as an adjunct to kill the organism in the lungs.

Bay laurel essential oil as inhalant steam: Add 1 or 2 drops to a pot of boiling water on the stove. Turn the stove off, remove the pot from the stove and set it on a table, sit down, cover both your head and the pot with a towel, and breathe in for a while. (More than 2 drops will probably be too strong.) People have reported good success with bay laurel essential oil in reducing the impact of the infection on the lungs—some have said it eliminated the infection entirely. (And no, they probably won't let you do this in the hospital or a nursing home for either yourself or your loved ones.)

Note: Bay laurel essential oil can also be dabbed or misted on masks or gloves to kill any virus that lands on their surfaces.

NEBULIZER

The use of a nebulizer will help a lot. These are available inexpensively through pharmacies and many online outlets.

The nebulizer cups that come with these machines are often not very good. You will need to get a different one. Philips Respironics is the brand I suggest. You can get the cups on the internet but not from the company that makes them without a prescription—which I find rather idiotic. They make two kinds (also idiotic). One is very cheap and is listed as disposable (don't get it), and the other is listed as reusable. *Get the reusable one.* Just wash it out after use with very hot water and liquid dish soap. The reusable one will stand up to essential oils if washed well after use. Mine lasts months before I can find any evidence of degradation of the plastic. The disposable ones will begin to degrade from the essential oils within a few days and you will start inhaling microparticles of plastic. Very much *not* a good idea.

You will need saline solution for the nebulizer. I use Modudose saline solution for inhalation, one 5 ml container per session.

You'll also need effervescent glutathione capsules. Glutathione is a potent antioxident, normally present in the surfactant liquid in the lungs. People with severe lung infections and chronic conditions tend to have low levels of all antioxidants including glutathione in their lungs. Using this in the nebulizer (see the instructions below) will help reduce inflammation in the lungs. I think Theranaturals Reduced L-Glutathione Plus (enhanced absorption, ultra purity grade) is the best one to use.

Essential oils are also very useful. I use peppermint (*Mentha piperita*) and eucalyptus (*Eucalyptus* spp.). The peppermint is strongly antispasmodic (helping coughing); both of the essential oils will help thin and liquify mucus and help it move up and out of the lungs, thus enhancing breathing and oxygen exchange. (Note: A drop of oregano essential oil can help reduce or stop the development of a lung infection . . . though not always. Nevertheless it is a good thing to try at early onset. Other suggestions are in the extended protocol that follows [see section 2.0, on the respiratory system, beginning on page 111]. And if essential oils are too strong for you, eliminate them.)

To set up the nebulizer: Pour 5 ml of saline solution into the nebulizer cup. Add the contents of a single 200 mg capsule of glutathione and let it dissolve. (It will fizz and foam when you first put it in the liquid . . . after you have finally gotten the capsule apart, that is.) Then, just before using the nebulizer, add 1 or 2 drops of peppermint essential oil and 1 or 2 drops of eucalyptus essential oil.

GUAIFENESIN

An over-the-counter guaifenesin tablet, such as Mucinex, will, along with the rest of the nebulizer protocol, thin and help move mucus up and out of the lungs. Dosage: 1 tablet (600 mg) in the morning and another in the evening. (I prefer extended-release tablets, but there are also non-extended-release forms, and they all work fine.)

To Protect the Kidneys

Again, nettle infusion daily along with nettle seed tincture. (See the core protocol on page 105.)

To Protect the Heart

Researchers are finding that heart damage (especially myocarditis) is a regular occurrence in people infected by this virus. For some people it will not resolve after infection. It is often not apparent until the person is under physical stress. Many of the people who have prolonged heart damage did not show any symptoms of Covid-19 infection at all; most of them are young, not old. Given this, it is essential that heart-protective herbs be taken if the virus is endemic in your area, if people around you are being infected, or if you yourself become infected. I'd suggest the following:

- ***Crataegus oxyacantha* (hawthorn),** 900 mg 2x daily as a protective measure; for an active infection, 1,800 mg 2x daily, as noted in the core protocol on page 105.
- ***Astragalus* spp.,** 1,000 mg 3x daily.

To Address GI Tract Exacerbations

To help with the symptoms of GI tract infection, e.g., cramping and diarrhea, I have found the following to be very helpful.

For cramping: *Viburnum prunifolium* (a.k.a. cramp bark) and other related species. Dosage: 30 to 90 drops of tincture up to 6x daily. This can take anywhere from a few minutes to a few days to kick in but it does help when it does. *And/or:* Peppermint—either capsules that include the essential oil *or* those really tiny coffee mints that are incredibly strong . . . just swallow 3 or 4 of them as needed.

For diarrhea: *Rubus villosus* (a.k.a. blackberry) root. Take it as a strong decoction: Put 1 to 2 ounces of the root in 2 quarts of water. Bring to boil and simmer until the liquid is reduced by half. Cool and then consume during the day. Repeat every day until the diarrhea is under control. Note: This should also help control any bleeding that is occurring. *Please* do not buy blackberry root tea bags, they are useless. Do not try to substitute raspberry root either, it is not nearly as good and you will probably end up with the leaves anyway, which are not nearly as strong. If you have trouble finding blackberry root (for some reason very few herbal companies carry this herb), try the herb shops on Etsy.

Also: *Ailanthus altissima* (tree of heaven) is another powerful (and underutilized) herb that is very good for a number of problems that

occur during Covid-19 infections, including diarrhea. It is the inner bark that is used (that is, the white bark that peels off easily, which is located just under the very thin green outer bark). This is an invasive botanical throughout the United States and much of the EU and thus pretty easy to find. *Other actions of the herb:* bronchial dilator, anti-inflammatory (especially for the lungs), antifibrotic (especially in the lungs), antiasthmatic, strong antioxident, antiviral, antimicrobial, antimycotic, antimalarial.

Note: Tincture of goldenseal (*Hydrastis canadensis*) or any of the other berberine-containing plants may be of use; they can sometimes initiate strong healing of the GI tract.

Extended Symptom-Specific Protocol for Treatment of Covid-19 and Post-Coronavirus Syndrome

1.0 Fatigue

A. Acute fatigue

1. *Eleutherococcus senticosus* tincture, in a 1:1 or 2:1 formulation (made by Herb Pharm and some others), taken as directed on the product label. Stop every 10 days for a few days, then begin again. When the fatigue becomes less severe, switch to the 1:5 formulation.
2. Glutrasol IE (a commercial supplement), taken as directed on the product label.
3. D-ribose powder, taken as directed on the product label.

B. Chronic fatigue

1. *Eleutherococcus senticosus* tincture, in a 1:5 formulation as a tonic, ½–1 teaspoon 3–6x daily, and/or . . .
2. Chronic fatigue formula (see 1.1, page 110): ¼ cup of the powder, blended in juice or water, in the morning and again just before bed, and/or . . .
3. D-ribose, in capsule form, up to 3,400 mg in the morning and at noonish, or 1 scoop of powder in liquid in the morning and at noonish, and/or . . .
4. Glutrasol IE (a commercial supplement): 1 scoop of powder in liquid daily.

C. For adrenal fatigue, add:

1. *Pinus* spp. (pine) pollen tincture, ¼–½ teaspoon 3x daily (take by mouth, let sit a minute, then swallow; do not dilute in water), and/or . . .
2. *Glycyrrhiza* spp. (licorice) tincture, ¼ teaspoon 3x daily (not to exceed 30 days), and/or . . .
3. *Lepidium meyenii* (maca) powder, 1 teaspoon 2–3x daily, and/or . . .
4. *Rhodiola* spp. tincture, 10–40 drops 2–4x daily, and/or . . .
5. *Codonopsis pilosula* tincture, ¼ teaspoon 4x daily.

D. For thyroid fatigue, add:

1. *Juglans nigra* (black walnut) hull tincture, 5–10 drops 2x daily, and/or . . .
2. Selenium, 200 mcg daily, and/or . . .
3. Kelp, 500 mg every other day, and/or . . .
4. *Rhodiola* spp. tincture, 10–40 drops 2–4x daily.

E. For mitochondrial fatigue, add:

1. *Leonurus cardiaca* (motherwort) fresh plant tincture, ½–1 teaspoon 3x daily, and/or . . .
2. Nicotinamide riboside (an NADH precursor), 900 mg daily during acute fatigue, 300 mg daily for mild, and/or . . .
3. NADH (nicotinamide adenine dinucleotide hydride, or NAD+), 10–20 mg 2x daily, and/or . . .
4. D-ribose, in capsule form, up to 3,400 mg in the morning and at noonish, or 1 scoop of powder in liquid in the morning and at noonish, and/or . . .
5. L-arginine, 1,000 mg 3x daily, and/or . . .
6. L-carnitine (500 mg 3x daily), alpha-lipoic acid (200–600 mg daily), coenzyme Q10 (60–150 mg daily).

1.1 Chronic Fatigue Formula

This is *very* specific for reversing fatigue, especially if it is chronic. Note: All the herbs must be *powdered*. (You can find this formula, preblended, from various sources if you do a quick search online. Etsy is a good place to look.)

You'll need:

- 2 parts (for example, 4 ounces) *each* of astragalus, dandelion root, licorice, milk thistle seed, nettle leaf, spirulina, and turmeric
- 1 part (for example, 2 ounces) *each* of ashwagandha, bladder wrack, burdock root, chlorella, eleuthero, and dried wheat grass juice powder

To make: Blend all the powdered herbs well in a *very* large bowl.

Dosage: In cases of very severe, acute fatigue (e.g., mono), I normally suggest ¼ cup of the powder, blended in a blender in water or juice, in the morning and evening just before bed. For ongoing, continual fatigue (where you are not bedridden), I suggest taking it only before bed. (Some people report being kept awake on this, and if so, take it before dinner; it doesn't bother me.) The dose can be adjusted up or down as necessary. Note: These are food-grade herbs, just like broccoli . . . well, okay broccoli is not actually edible . . . like red chard then.

2.0 Respiratory System

Covid-19's involvement with the respiratory system, during infection and in long haulers, can be broken down into two categories: upper and lower.

2.1 Upper Respiratory

A. Sinus problems

1. Sinusitis, ongoing (including infection):

- **a.** Cold Snap, taken as directed on the product label.
- **b.** Black seed (*Nigella sativa*) oil, 2,000 mg 1–3x daily.
- **c.** Xlear Nasal Spray, as needed.
- **d.** Caprylic acid, taken as directed on the product label.

2. Sinus pain: serrapeptase, 120,000 SPU 2–3x daily.

3. Congestion:

- **a.** Myrtol (a.k.a. GeloMyrtol Forte), taken as directed on the product label.
- **b.** *Monarda* spp. (bee balm) tincture, 20–30 drops as needed.
- **c.** Xlear Nasal Spray, as needed.

4. Burning:
   a. Homeopathic Cantharis 30C, taken as directed on the product label.
   b. Homeopathic Gelsemium 30C, taken as directed on the product label.
5. Sneezing (with drippiness): homeopathic Sulphur 30C, taken as directed on the product label.
6. Postnasal drip:
   a. Myrtol (a.k.a. GeloMyrtol Forte), taken as directed on the product label.
   b. Homeopathic Sulphur 30C, taken as directed on the product label.
   c. Xlear Nasal Spray, as needed.

B. Sore throat: *Echinacea angustifolia* tincture (do not use *E. purpurea*), half a dropper or so of tincture in the mouth, hold until saliva is stimulated, then let the tincture dribble slowly down the back of the throat. Repeat as needed.

C. Loss of smell: If the core protocol (page 105) does not restore function, try these. They work best with smell, and to some extent with taste. The first two are the most reliable (so far). (About two-thirds of people recover their sense of smell after the first 2 days. Another 20 percent recover their sense of taste after using the aspirin/ivermectin combination. Nearly all people recover some or all of their sense of smell after the final aspirin/ivermectin/L-lysine combination.)

1. Zinc/quercetin combination. Dosage: zinc, 50 mg daily; quercetin, 200–1,000 mg daily (800 mg suggested).
2. Ivermectin/aspirin combination treatment. (This is complicated, as all forebrain people make things, but it does seem to work.) To begin, reduce arginine-rich foods in the diet, including coffee, soft drinks, and all citrus fruits. Then:
   a. Ivermectin, 0.2 mg per kg of body weight daily for 2 days, taken after dinner. If not resolved by second day, then switch to . . .
   b. Aspirin, 100 mg after breakfast and 100 mg after dinner for 5 days (from day 3 to 7). On days 5 and 6, take ivermectin again, now at 0.4 mg per kg of body weight daily; take half the dose after lunch, the rest after dinner. If not resolved by the morning of day 8, then . . .

   c. Continue taking aspirin, same dosage and times, and start taking L-lysine, 500 mg daily. Every 3 days, increase the L-lysine dosage by 500 mg until you are at 2,000 mg per day. On the eighth day after beginning the L-lysine, start ivermectin again, 0.4 mg per kg daily in two divided doses for 3 days. Then stop the protocol. If it has not worked by now, it probably will not.

3. Olfactory training. The process, which uses essential oils to retrain the sense of smell, takes several months but seems to be effective for most people if diligently done. Do an internet search for "olfactory training"; there are several companies that specialize in it (one by someone who suffered loss of smell), their directions are easy to understand, and they seem very responsive to inquiries. Google Scholar will lead you to a number of studies on its effectiveness.

4. Acupuncture and traditional Chinese medicine. A number of studies found that acupuncture with or without traditional Chinese medicine is effective in restoring a sense of smell and, to some extent, taste.

5. Chamomile nasal irrigation. Make a decoction of chamomile (bring to boil, reduce heat, and let simmer until water is decreased by half, then strain very well). Use it with a neti pot for nasal irrigation daily for a week (ick), or put the decoction into a nasal sprayer and use 3x daily for a week.

6. Lion's mane (*Hericium erinaceus*). This herb is fairly good at restoring damaged neural structures in the brain and body. There are some reports of it helping restore sense of smell. Dosage: tincture, ½ to 1 teaspoon 3x daily.

D. Loss of taste

1. The above protocols for loss of smell (especially 1 and 2) can help with this.

2. Monosodium glutamate (MSG), used as directed on the product label. Though MSG has a bad reputation, more recent research has found that unwarranted. MSG enhances the sensitivity of the taste buds to the taste of food. It has, for some people, helped resensitize the sense of taste after Covid-19 infection.

2.2 Lower Respiratory

A. General: Continue with the nebulizer protocol (page 106); this will help clear mucus and help breathing and oxygen intake.

B. Persistent, uncontrollable cough

1. Oxygen generator/concentrator, used as needed. Most of the standing machines have settings that go to 5 (a few go to 10). Use setting 2, at most 3.
2. Tramadol (an opiate, by prescription, and due to the current opiate hysteria, hard to get). Some people prefer to use 50 mg twice daily; I have found the 100 mg extended-release capsule once daily preferable, but this is simply a matter of individual taste and response.
3. Herbs that stimulate the vomiting reflex are sometimes specific for severe uncontrollable cough. (The point is to *stimulate* the reflex, not activate it.) Ipecac, once a staple in every medicine cabinet in the United States, is a case in point but is now, thanks to the FDA, impossible to get. However . . .
   a. *Sambucus* spp., fresh (that is, *nondecocted*) leaf tincture, up to 30 drops as needed. Note: Decocted tinctures (which deactivate the compounds that cause nausea and/or vomiting) are available but for this the nondecocted is more effective (if it is going to work for you at all). Extended use may cause watery diarrhea.
   b. *Lobelia inflata*, fresh or dried leaf tincture, 5–20 drops as needed, or the dried seed tincture, 3–10 drops as needed. The dried leaf is far more nausea inducing than the fresh leaf or seed and may be more useful for stopping severe coughing. For some people the plant is also a strong emetic, I, however, have not found it so. Note: This tincture can also help move mucus up and out of the lungs; see section D (facing page).
4. *Echinacea angustifolia* root tincture can anesthesize the back of the throat and to some extent the bronchi if a half dropper of tincture is taken, held in the mouth until saliva is stimulated, and then slowly dribbled down the back of the throat. Repeat as needed.
5. Western skunk cabbage (*Lysichiton americanus*) can sometimes help; see section D (facing page).
6. Other herbs and herbal combinations might be useful as well; please see section C (facing page).

C. Cough, general

1. Nebulizer with peppermint essential oil can help, sometimes significantly. Peppermint is specific for spasming. It works just as well in the lungs as the GI tract.
2. *Desmodium* spp. leaf tincture (1:5 formulation, 50% alcohol), 1 teaspoon up to 6x daily. It may also be used in cough syrups; it is an excellent underused herb.
3. *Aster tataricus* root tincture (1:5 formulation, 50% alcohol), ½–1 teaspoon up to 6x daily; another excellent underused herb.
4. *Pelargonium sidoides* (umckaloabo) tincture (1:5 formulation, 50% alcohol), 30 drops up to 6x daily.
5. Myrtol (a.k.a. GeloMyrtol Forte), taken as directed on the product label.
6. *Hedera helix* (English ivy), as tea, often combined with . . .
7. *Thymus vulgaris*, as tea with or without ivy, or combined in cough syrup.

D. Mucus, excessive, in the lungs (with cough or not): Mucus buildup is often a problem during lung infections and in damaged lungs. The buildup of mucus in the lungs is bad for a number of reasons: It inhibits depth of breathing thus lowering blood O_2, is a fertile ground for pathogenic organisms, and by itself stimulates the cough reflex . . . which will not stop until you get it out. If you are suffering post-coronavirus syndrome and you have persistent mucus buildup in your lungs you will need to develop a daily regimen to get the mucus out. There are a number of things that can help. Use as many of them daily as you can. (Xlear Nasal Spray, as needed, will also help.)

1. *Lysichiton americanus* (western skunk cabbage) *freshly* dried root tincture (the well-dried root is far less effective). Dosage: As desired or needed—normally, what I use for myself is around 30 drops whenever I want or feel like I need some.
2. Guaifenesin tablets, 600 mg daily. Mucinex is a good one but others work well. Guaifenesin is a compound isolated from plants in the *Guaiacum* genus. It will thin and help stimulate the expectoration of mucus from the lungs. It is only minimally a cough suppressant and only then because there is less mucus in the lungs.
3. Nebulizer with essential oils of peppermint and eucalyptus daily.

4. Fresh ginger juice tea (see page 39), 3–6x daily. Extremely good for thinning mucus.
5. Other mucus-thinning herbs of note: *Desmodium* spp., *Aster tataricus*, fennel, fenugreek, yerba santa (*Eriodictyon* spp.), thyme, English ivy, coltsfoot, cayenne, osha, *Pelargonium sidoides*, Myrtol (a.k.a. GeloMyrtol Forte), and so on.
6. Flutter device. Smiths Medical acapella is a decent one. This will help break up mucus in the lungs and stimulate expectoration. Many people use them. They work better if you are also using herbs that thin the mucus.
7. Inversion table. If you have an inversion table, lying on one daily can help the mucus flow upward, stimulate cough, and help it move out of the system, especially if you are taking herbs that thin the mucus. (I didn't find it particularly helpful but many people do.)

E. Hemoptysis (coughing up blood)

1. Yin Qiao San (a traditional Chinese medicine, or TCM, formulation), taken as directed on the product label. Note: You can find this formula by searching online.
2. Ke Xue Fang (another TCM formulation), taken as directed on the product label. Note: This formulation is generally available only to licensed practitioners; if you're not a practitioner, you could try Green Dragon Botanicals (https://greendragonbotanicals.com).
3. Huai Jiao Wan (one good brand-name version is called Sophora Support), taken as directed on the product label. Note: You can find this formula by searching online.
4. *Desmodium* spp. leaf tincture (1:5 formulation, 50% alcohol), 1 teaspoon up to 6x daily.
5. *Cinnamonum* spp. (cinnamon) tincture (60% alcohol, 5% glycerin), 20–50 drops 4x daily.
6. Combination tincture formula: equal parts of *Polygonum cuspidatum, Echinacea angustifolia,* and *Salvia miltiorrhiza* tinctures, ½–1 teaspoon up to 6x daily.

F. Shortness of breath (dyspnea)

1. Severe:
   a. Liquid chlorophyll (the ChlorOxygen brand is often used), 1 tablespoon in 20 ounces water, drink throughout the day, and/or . . .
   b. *Ailanthus altissima* tincture, 10 drops–½ teaspoon 4x daily, and/or . . .
   c. *Lysichiton americanum* (western skunk cabbage) freshly dried root tincture, 30 drops as needed or desired.
2. Mild: same as above, plus . . .
   a. *Cordyceps* spp. tincture, 1 teaspoon 3x daily, and/or . . .
   b. *Polygonum cuspidatum* (Japanese knotweed) root tincture, ½ teaspoon 3–6x daily, and/or . . .
   c. *Astragalus* spp., 1,000–4,000 mg 3–4x daily.

G. Hypoxia

1. *Lysichiton americanus* (western skunk cabbage) freshly dried root tincture, 30 drops as needed or desired.
2. *Rhodiola* spp. tincture, 10–40 drops 2–4x daily.

H. Wheezing

1. *Ammi visnaga* (khella) tincture (1:5 formulation, 60% alcohol), 60–120 drops up to 4x daily. Capsules are also helpful.
2. *Datura* spp. fresh leaf tincture, 5–10 drops as needed.
3. *Lysichiton americanus* (western skunk cabbage) freshly dried root tincture, 30 drops as needed or desired.

I. Rattling breath (i.e., crackling or Velcro sounds): This is common in chronic lung conditions. It is caused by mucus building up in the bronchioles in the lungs. When you breathe in or out, the air has to move through the mucus, which makes the sound. Clearing the mucus will help. See section D (page 115).

J. Lung burn

1. Xie Bai San (TCM formulation), taken as directed on the product label. Note: This formulation is generally available only to licensed practitioners; if you're not a practitioner, you could try Green Dragon Botanicals (https://greendragonbotanicals.com).

2. Ma Xing Gan Shi Tang (TCM formulation; again, generally available only to practitioners), taken as directed on the product label.
3. Combination tincture formula: 1 part goji berry (*Lycium chinense*) tincture, 1 part white mulberry (*Morus alba*) tincture, 1 part white peony root (*Paeonia lactiflora*) tincture, ½ part licorice (*Glycyrrhiza* spp.) tincture. Dosage: 1 teaspoon in the liquid of your choice 3–6x daily depending on the intensity of symptom. (Note: The first three herb tinctures can be a little tricky to find. The best place to look is Etsy. And again, in my experience, glycerites are *not* strong enough.)

K. Chest pain (including tightness in the chest)

1. Pea protein, taken as directed on the product label, 1–2x daily. (Note: I use the Jarrow Formulas brand.)
2. *Piper methysticum* (kava) 10:1 extract, as an instant powder, in a cup of hot water (with honey and cream), 1–3 cups daily as needed.
3. Combination tincture formula: equal parts of motherwort (*Leonurus cardiaca*) and *Pedicularis bracteosa* (i.e., lousewort, which I prefer, for taste reasons, over *Pedicularis groenlandica*, i.e., elephant head, though either works fine). Dosage: 1–2 tablespoons as needed, in liquid.
4. *Pulsatilla patens* (pasque flower) fresh flower tincture, 5–10 drops as needed.

L. Pleurisy (inflammation of the pleural sac)

1. *Asclepias tuberosa* (pleurisy root) tincture, 30–90 drops 3x daily. (Note: All *Asclepias* species are useful for this.)
2. *Mangifera indica* capsules, standardized to 60 percent mangiferin, 200–600 mg 3x daily (Green Dragon Botanicals brand: https://greendragonbotanicals.com).

M. Chronic bronchitis

1. Si Ni Tang (TCM formulation of aconite, ginger, licorice), 4 capsules 3x daily. Note: Best if used with ephedra (and yes, I still sometimes order ephedra from China irrespective of what the FDA thinks I should do; it's a good herb but meth heads ruined it for the rest of us).
2. *Desmodium* spp. leaf tincture (1:5 formulation, 50% alcohol), 1 teaspoon up to 6x daily.
3. *Aster tataricus* root tincture (1:5 formulation, 50% alcohol), ½–1 teaspoon up to 6x daily.

4. *Pelargonium sidoides* (umckaloabo) tincture (1:5 formulation, 50% alcohol), 30 drops up to 6x daily.

N. Recurrent lung infections

1. Combination tincture formula: 2 parts *Lomatium* spp., 2 parts *Echinacea angustifolia*, 2 parts *Glycyrrhiza* spp. (licorice), 2 parts *Ceanothus* spp. (red root), 2 parts *Bursera microphylla* (elephant tree), 1 part decocted *Sambucus* spp. (elder) leaf or bark, 1 part *Asclepias asperula* (inmortal; pleurisy root will do but is not as good), 1 part *Ligusticum porteri* (osha), 1 part *Inula helenium* (elecampane), 1 part *Isatis* spp. (root or leaf or combination of the two), 1 part *Eriodictyon* spp. (yerba santa). Dosage: 30–60 drops each hour until the infection resolves.
2. Gan Mao Ling, 5–6 tablets 6x daily during active infection. Use this formula in concert with the lomatium combination tincture formula above (since lung infection in those with compromised lungs is a serious issue, such infections need to be reduced as rapidly as possible).

O. To inhibit, reduce, or repair fibrosis (scarring) of the lung

1. Combination tincture formula: equal parts of *Angelica sinensis, Salvia miltiorrhiza, Lonicera japonica, Polygonum cuspidatum, Cordyceps* spp. Dosage: 1 teaspoon 3–6x daily depending on the severity of fibrosis, and . . .
2. Lumbrokinase or nattokinase, 600,000 IU (a.k.a. 40 mg) 2–3x daily. (Note: If you are already taking anticoagulants, caution is warranted in adding either of these.)

Western Skunk Cabbage

This herb (*Lysichiton americanus*) has a great deal of usefulness in chronic lung conditions; it increases O_2 levels in the blood, lowers cough levels (even when intense), and, *importantly*, liquifies and then stimulates expectoration of mucus from the lungs, copiously. Note: I have not used the eastern variety and I am not sure it will do the same thing (though I have been told it will). The western variety is a bit hard to find.

3.0 Neurological/Brain Problems

A. Specific

1. *Uncaria rhynchophylla* tincture, ½–1 teaspoon 3–6x daily, depending on the severity of the brain infection.
2. Tryptophan, 1,500 mg 3x daily. (Note: Will lower brain inflammation and decrease a number of psychological/physiological symptoms.)

B. With severe brain/central nervous system involvement, add:

1. *Scutellaria baicalensis* tincture, increase the current dose, plus . . .
2. *Chelidonium majus* (greater celandine) tincture, ¼ teaspoon 3x daily, plus . . .
3. *Pueraria lobata* (kudzu) root tincture, ¼ teaspoon 3–4x daily.
4. N-acetylcysteine, 2,000 mg 2x daily, may also help, as will . . .
5. *Leonurus cardiaca* (motherwort) fresh plant tincture, ¼–½ teaspoon up to 6x daily.

C. To reduce neurotoxins in the brain (e.g., quinolinic acid), add:

1. *Sida cordifolia* tincture, 5–40 drops up to 3x daily, and/or . . .
2. *Angelica sinensis* tincture, ¼–½ teaspoon 3x daily, and/or . . .
3. Melatonin, 3–9 mg daily.

D. When the brain "feels toxic," add *Centella asiatica*, 500 mg or ¼ teaspoon tincture 2x daily. (Note: May cause headaches.)

E. With low brain energy, add acetyl-L-carnitine, 500 mg 2x daily. (Note: Contraindicated if seizures are present.)

F. With brain "pressure," add *Pueraria lobata* (kudzu) tincture, ¼–½ teaspoon 3x daily.

G. With hand or body tremors, add:

1. *Sida acuta* (or equivalent species) tincture, 5–40 drops 3x daily, and/or . . .
2. *Scutellaria baicalensis* tincture, ½ teaspoon 3x daily, and/or . . .
3. *Mucuna pruriens* (an L-dopa precursor), 500 mg 1x daily in morning.

H. With brain fog, memory issues, cognitive dysfunction, or trouble finding words, add:

1. Phosphatidylserine, 100 mg 3x daily, and/or . . .
2. *Ginkgo biloba*, as standardized capsules, 150 mg 2x daily, and/or . . .

3. *Centella asiatica* (gotu kola), 500 mg or ¼ teaspoon tincture 2x daily (may cause headaches), and/or . . .
4. Taurine, 125 mg 3x daily.
5. Some of the following may also be of use:

   - Phosphatidylcholine, 500 mg 3x daily.
   - *Cordyceps* spp. powder, 1 teaspoon–1 tablespoon 3x daily, or tincture, 1 teaspoon 3x daily.
   - *Pueraria lobata* (kudzu root), 500–1,000 mg or ¼–½ teaspoon tincture 3x daily.
   - *Polygala tenuifolia* (Chinese senega root) tincture, 30 drops 3x daily.
   - *Hericium erinaceus* (lion's mane), 1 teaspoon powder or ¼–½ teaspoon tincture 3x daily.
   - Quercetin, 1,200 mg daily.
   - Pycnogenol (from French maritime pine bark only), 100 mg 1x daily.
   - Vitamin D_3, 5,000–10,000 IU daily.
   - *Bacopa monnieri* (especially for short-term memory help), 500 mg 2x daily.
   - Homeopathic Kali Phos 30C, 4 pellets 3x daily.

I. With hypoperfusion of the brain, add *Ginkgo biloba*, as a standardized tincture, ¼ teaspoon 3x daily, or as standardized capsules, 125 mg 3x daily.

J. With neural pain, add:

1. *Chelidonium majus* (greater celandine) tincture, ¼ teaspoon 3x daily, and/or . . .
2. *Pueraria lobata* (kudzu) root tincture, ½ teaspoon 3–4x daily, and/or . . .
3. *Melissa officinalis* (lemon balm) tincture, ½ teaspoon 3–4x daily, and/or . . .
4. Homeopathic Kali Phos 30C, 4 pellets 4x daily.

K. With a "buzzing" or "electric feeling" in the nerves, add:

1. *Sida acuta* (or equivalent species) tincture, 5–40 drops 3x daily.
2. *Pulsatilla patens* (pasque flower) tincture, 5–10 drops as needed.
3. *Piper methysticum* (kava) 10:1 extract, as an instant powder, in a cup of hot water (with honey and cream), as needed or desired.
4. Vitamin B_{12}, 1,000 mcg sublingually.
5. *Cannabis indica*, smoked or as an edible (5 mg).

L. With epilepsy/seizures, add:

1. *Uncaria rhynchophylla,* in an increased dose of up to 1 tablespoon 6x daily depending on the severity of the seizures, and also take . . .
2. *Gastrodia elata* tincture, ¼–½ teaspoon 3–6x daily.
3. *Salvia miltiorrhiza* may also be of help; it's already part of the core protocol for active infection, and here you can increase the dose to 1 tablespoon 3–6x daily, depending on the severity of the seizures, and/or . . .
4. *Cannabis* spp. oil or equivalent, variable dosages, and/or . . .
5. *Cryptolepis sanguinolenta* tincture, ½ teaspoon 3–6x daily, and/or . . .
6. Taurine (which sometimes helps), 125 mg 3x daily.
7. Frankincense essential oil, applied topically, daily, to the temples and base of skull may help alleviate severity of seizures.

M. With left temporal strokes, add:

1. *Salvia miltiorrhiza,* in an increased dose of up to 1 teaspoon 6x daily, and/or . . .
2. *Uncaria rhynchophylla,* in an increased dose of up to 1 teaspoon 6x daily, and/or . . .
3. *Ginkgo biloba,* as a standardized tincture, 1 teaspoon 3–6x daily, or as standardized capsules, 600 mg 3x daily.

N. With subarachnoid hemorrhage, add melatonin, 3–9 mg daily.

O. With bouts of unrestrained rage, add:

1. *Uncaria rhynchophylla,* in an increased dose of up to 1 teaspoon 6x daily, and/or
2. *Cryptolepis sanguinolenta* tincture, ½ teaspoon 3–6x daily, and/or . . .
3. Tryptophan, 1,000–1,500 mg 3x daily.

P. With a feeling of the brain being "on fire," add homeopathic Gelsemium 30C, 4 pellets 4x daily.

Q. To restore neuronal structures, add neural regrowth stimulants:

1. *Polygala tenuifolia* (Chinese senega root) tincture, 30 drops 3x daily, and/or . . .
2. *Hericium erinaceus* (lion's mane) powder, 3–8 grams per day, or 1 teaspoon tincture 3–4x daily.

R. When the limbs feel heavy, add *Centella asiatica* (gotu kola), 500 mg or ¼ teaspoon tincture 2x daily.

3.1 Muscle Twitches, Tingling/Crawling Sensations/ Numbness in the Extremities

A. General

1. Vitamin B_{12}, 1,000 mcg daily (lower the dose to 500 mcg as symptoms resolve), and/or . . .
2. Vitamin B_6, 100 mg 2x daily (lower the dose to 50 mg as symptoms resolve), and/or . . .
3. Folic acid, 400 mcg daily, and/or . . .
4. Magnesium, 200–400 mg up to 3x daily, and/or . . .
5. *Sida acuta* tincture, 5–40 drops 3x daily.

B. With numbness, add:

1. *Polygonum cuspidatum* (Japanese knotweed) root tincture, ½ teaspoon 6–10x daily. (Note: Especially useful for carpal tunnel—and lateral epicondylitis—type problems.)
2. *Ginkgo biloba,* as a standardized tincture, 1 teaspoon 3–6x daily, or as standardized capsules 600 mg 3x daily.
3. Fresh ginger juice tea (see page 39), 3–4 cups daily.

3.2 Anxiety/Hysteria/Extreme Fear/Panic Attacks

A. General

1. *Pulsatilla patens* (pasque flower) tincture, 10 drops each hour for as long as necessary, and/or . . .
2. *Leonurus cardiaca* (motherwort) fresh plant tincture, ¼–½ teaspoon up to 6x daily, and/or . . .
3. *Corallorhiza maculata* (coral root), or equivalent species, tincture, 30 drops (full dropper) up to 6x daily, and/or . . .
4. Homeopathic Gelsemium 30C, 4 pellets 4x daily, and/or . . .
5. *Scutellaria baicalensis* tincture, ¼–½ teaspoon 3x daily, and/or . . .
6. *Verbena officinalis* (vervain) tincture, 30 drops up to 6x daily, and/or . . .
7. *Uncaria rhynchophylla* tincture, 30 drops up to 6x daily, and/or . . .
8. Tryptophan, 1,000–1,500 mg 3x daily.

B. With inconsolable anxiety, add homeopathic Aconite 30C, 4 pellets dissoved in ½ cup water, sipped throughout the day.

3.3 Sleep Disturbance/Insomnia

A. General

1. Melatonin liquid, taken as directed on the product label, an hour before bed, and/or . . .
2. *Withania somnifera* (ashwagandha) tincture, ½ teaspoon an hour before bed, or powder or capsules, 1 gram an hour before bed, and/or . . .
3. *Scutellaria baicalensis* tincture, ½–1 teaspoon 3x daily, and/or . . .
4. *Leonurus cardiaca* (motherwort) fresh plant tincture, ¼ *ounce* (yes, that is right) in liquid just before bed (if the melatonin does not help), and/or . . .
5. Suan Zao Ren Tang tablets/pellets (look for the Plum Flower brand of this TCM formula, which you can find by searching online), 5 tablets just before bed, and/or . . .
6. Te Xiao Zao Ren An Mian Pian (look for Sleepeace, a version of this TCM formula from the manufacturer Guang Ci Tang), 5 tablets just before bed, and/or . . .
7. Glycine, 125–375 mg daily, and/or . . .
8. Tryptophan, 1,000 mg just before bed, and/or . . .
9. *Cannabis indica* gummies, 5 mg just at bedtime.

B. For bolting awake in middle of night, add:

1. Phosphatidylserine, 100 mg 3x daily, and/or . . .
2. *Withania somnifera* (ashwagandha), ½ teaspoon of tincture or 1 gram powdered or in capsules an hour before bed, and/or . . .
3. *Schisandra chinensis* tincture, ½ teaspoon just before bed, and/or . . .
4. *Cannabis indica*, various formulations.

3.4 Depression

A. General

1. *Eleutherococcus senticosus* tincture, in a 1:1 formulation, ¼–½ teaspoon 3x daily (with a break every 10 days), and/or . . .
2. Melatonin, 3–9 mg daily, and/or . . .
3. *Mucuna pruriens*, 500 mg 1x daily in the morning, and/or . . .
4. *Leonurus cardiaca* (motherwort) fresh plant tincture, ¼–1 teaspoon as often as needed, and/or . . .
5. *Corallorhiza maculata* (coral root), or equivalent species, tincture, ½–1 teaspoon up to 6x daily, and/or . . .

6. SAMe, 200 mg 1–2x daily, and/or . . .
7. Tryptophan, 1,000–1,500 mg 3x daily, and/or . . .
8. *Mitragyna speciosa* (kratom) powder, ½ teaspoon mixed in warm water, 1–3x daily (may cause jitteriness—or nausea at higher doses).

3.5 Headaches

A. Migraine-like

1. *Verbena officinalis* (vervain) tincture, ¼–1 teaspoon as needed, and/or . . .
2. *Cannabis* spp. or cannabidiol (CBD), variable dosages, and/or . . .
3. *Pueraria lobata* (kudzu), ½ teaspoon 3–4x daily (will also help prevent), and/or . . .
4. *Scutellaria baicalensis* tincture, ½ teaspoon 6x daily (in addition to the core protocol dose), and/or . . .
5. *Piper methysticum* (kava), 10:1 extract, instant powder, in a cup of hot water (with honey and cream), as needed or desired, and/or . . .
6. Lithium orotate, 5–20 mg daily.

B. Headache at the back of the head: *Verbena officinalis* (vervain) tincture, ¼–1 teaspoon as needed.

C. Headache at the front of the head

1. *Silybum marianum* (milk thistle) seed, standardized, 1,200 mg every 3 hours, and/or . . .
2. *Rumex crispus* (yellow dock) root tincture, 1 teaspoon in water at bedtime.

4.0 Cardiovascular System

A. Cardiomyopathy, general: *Crataegus oxyacantha* (hawthorn), 120–900 mg 3x daily.

B. Blood clotting (thick blood): Lumbrokinase, 600,000 IU (a.k.a. 40 mg) 2–3x daily. (Note: Nattokinase will also work, but serrapeptase is too weak. If you are already taking anticoagulants, caution is warranted in adding either lumbrokinase or nattokinase.)

C. Elevated heart rate (hypertension/tachycardia)

1. Specific: *Uncaria rhynchophylla* tincture, ½ teaspoon up to 6x daily.
2. *Crataegus oxyacantha* (hawthorn), 120–900 mg 3x daily, and/or . . .

3. *Leonurus cardiaca* (motherwort), 30 drops–1 teaspoon up to 6x daily, and/or . . .
4. *Mimosa pudica* tincture, 20–60 drops daily. (Note: May also be of benefit if accompanied by depression, anxiety, headaches, and damaged nervous structures.)

D. Low pulse rate (hypotension)

1. *Glycyrrhiza* spp. (licorice) root tincture, 1 teaspoon up to 6x daily depending on the severity of the condition (note: do not take for more than 60 days in this form), and/or . . .
2. Caffeine, variable dosing (try rocket juice chai: a strong infusion of 2 heaping tablespoons black tea chai, 1 heaping tablespoon yaupon, 1 heaping tablespoon yerba mate, 1 heaping tablespoon kola nut, 1 heaping tablespoon guarana, all in a French press, let steep 1 hour, add honey and heavy cream to taste), or . . .
3. If nothing else works, try yohimbine as a supplement. Begin with the dosing recommendations on the product label and increase as needed. (Please note the warnings on the label and use caution.)

E. Palpitations (specific and immediate options)

1. *Scutellaria lateriflora*, 20–60 drops as needed or desired.
2. *Passiflora incarnata*, ½ –1½ teaspoons as needed or desired.
3. *Piper methysticum* (kava), 10:1 extract, instant powder, in a cup of hot water (with honey and cream), as needed or desired.

F. Angina

1. *Terminalia arjuna* (a.k.a. arjuna), 500 mg 3x daily.
2. Hartone capsules (an Ayurvedic remedy from Swadeshi Pharmaceuticals), 1–2 capsules 3x daily.
3. *Ammi visnaga* (khella), 250–300 mg daily, and/or . . .
4. L-carnitine, 500 mg 3x daily, and/or . . .
5. *Crataegus oxyacantha* (hawthorn), 120–900 mg 3x daily, and/or . . .
6. *Salvia miltiorrhiza* tincture, ½ teaspoon 3–6x daily, and/or . . .
7. *Astragalus membranaceus*, 1,000–4,000 mg 3–4x daily.

G. Myocarditis

1. *Mangifera indica*, standardized to 60 percent mangiferin, 1,000 mg 3x daily (Green Dragon Botanicals brand: https://greendragonbotanicals.com), plus . . .

2. *Crataegus oxyacantha* (hawthorn), 120–900 mg 3x daily, and . . .
3. *Astragalus membranaceus*, 1,000 mg 3x daily.

H. Cardiac fibrosis

1. *Terminalia arjuna* (a.k.a. arjuna), 500 mg 3x daily.
2. *Curcuma longa* (turmeric), 750 mg 3x daily.
3. *Polygonum cuspidatum* (Japanese knotweed) root tincture, 1 teaspoon 3x daily.
4. *Salvia miltiorrhiza* tincture, 1 teaspoon 3x daily.

I. Arrhythmia

1. *Stephania tetrandra* or *S. cepharantha* tincture, ½ teaspoon 3x daily, and/or . . .
2. *Crataegus oxyacantha* (hawthorn), 120–900 mg 3x daily, and/or . . .
3. Taurine, 125–375 mg 3x daily, and/or . . .
4. *Leonurus cardiaca* (motherwort) fresh plant tincture, ¼ teaspoon 4x daily.

J. Shortness of breath

1. *Polygonum cuspidatum* (Japanese knotweed) root tincture, ½ teaspoon 3–6x daily, and/or . . .
2. *Astragalus membranaceus*, 1,000–4,000 mg 3–4x daily, and/or . . .
3. Liquid chlorophyll, 1 tablespoon in 20 ounces of water once a day, and/or . . .
4. *Cordyceps* spp. powder, 1 teaspoon–1 tablespoon 3x daily, and/or . . .
5. *Ailanthus altissima* tincture, 10 drops–½ teaspoon 4x daily.

K. Poor circulation (cold extremities): Fresh ginger juice tea (see page 39), 3–4 cups daily.

5.0 Gastrointestinal Tract

A. Loss of taste: See 2.1, section D (page 113).

B. Loss of appetite: *Cannabis* spp.

C. Nausea

1. Homeopathic Nux vomica 30C, 4 pellets every hour, and/or . . .
2. *Mentha piperita* (peppermint) essential oil, 1 drop only, on the tongue, followed by 6 ounces of water.
3. *Moringa oleifera*, 1 teaspoon powder in water 3x daily.

D. Vomiting: Homeopathic Arsenicum album 200C, immediately, at the first feeling of possible vomiting, as directed on the product label.

E. Cramping

1. *Viburnum* spp. (cramp bark), 30–90 drops up to 4x daily.
2. *Pulsatilla patens* (pasque flower), 10 drops as needed, usually no more often than once per hour.

F. Diarrhea

1. Blackberry root, as a strong infusion: ¼–1 ounce herb in 1 quart of hot water, cover and steep overnight, strain, and drink throughout the day. Or prepare the root as a decoction (see page 108) for acute episodes. Note: Do *not* use blackberry tea bags and do not substitute raspberry (unless you must). Oak has minimal effectiveness. Blackberry root is the way to go. It is rare to find any herbal company selling it; try Etsy, it is always there.
2. *Ailanthus altissima* tincture, 1 teaspoon 3x daily or as needed.

G. Gastric reflux (ranging from lower esophageal burning to heartburn to GERD)

1. For lower esophageal burning: *Heracleum maximum* (cow parsnip) seed tincture, 1–2 drops. Use at the onset of burning, as needed. Note: This is a *very* strong, resinous tincture that is also excellent for hiatus hernia.
2. For mild to moderate GERD, as well as lower esophageal burning:
   a. Iberogast (an over-the-counter herbal formulation; you can find it by searching online), as directed on the product label.
   b. Wu Zhu Yu Tang (sometimes called Evodia Formula), as a liquid, as directed on the product label. (Note: Evodia Formula can be made by combining 2 parts ginger root, 1 part evodia fruit, 1 part Asian ginseng root, and ½ part jujube.)
3. For severe GERD: Sini Zuojin combination formula as a decoction or powder. This traditional Chinese formula combines Sini powder extract and Zuojin pill as a treatment for severe GERD.

H. Leaky gut

1. *Salvia miltiorrhiza* tincture, 1 teaspoon 3x daily.
2. *Althaea officinalis* (marshmallow) root powder, 1 teaspoon–1 tablespoon in liquid 3x daily.

3. Turmeric milk, 3x daily.
4. Glutamine, 500 mg 2x daily.

I. Ulceration/damage to bowel wall and epithelia

1. Fresh juice formula: 1 wedge of green cabbage the size of a medium carrot (the core of the protocol—lowers inflammation, heals ulceration/mucosa), 3–4 fresh plantain (*Plantago* spp.) leaves (if you can find them—look in the yard, the plant really does help heal the mucosa and lower inflammation), 1 medium beet, 4 stalks celery, 3 carrots. Drink the blend twice daily, in the morning and just before bed.
2. The chronic fatigue formula (see 1.1, page 110) will help heal the bowel wall as well as lower bowel inflammation and help normalize cytokines.
3. Although most herbalists no longer recommend it, I still use and am a fan of comfrey root powder for healing bowel ulceration, mucosa, and inflammation. I add 1 tablespoon to the chronic fatigue formula (see 1.1, page 110), or else I simply make up a separate blend of 1 tablespoon comfrey root powder, 1 tablespoon licorice root powder, and 1 tablespoon marshmallow root powder. Limit the intake to 30 days.

6.0 Liver, Elevated Enzymes/Inflammation

A. General: *Silybum marianum* (milk thistle) seed, standardized, 1,200 mg 3x daily.

B. Liver pain, just under rib cage

1. *Salvia miltiorrhiza* tincture, 1 teaspoon 3x daily, and/or . . .
2. *Ceanothus* spp. (red root) tincture, ¼–1 teaspoon 3x daily, and/or . . .
3. *Schisandra chinensis* tincture, ¼–½ teaspoon 3x daily.

Concerns about Comfrey

The reason most people are skittish about comfrey is due to concerns about the pyrrolizidine alkaloids (PAs) in the plant. I don't consider PAs a problem for short-term use and have never seen negative impacts from them in 35 years of practice when used short term. But if you have concerns about PA impacts on the liver, take comfrey with standardized milk thistle seed (1,200 mg three times daily). I have never found anything better for healing damage to the intestinal tract, even in cases where surgeons were prepared to remove large sections of the stomach or bowel due to ulceration.

C. Fibrosis in organs: See 13.0 (page 133).

D. Cholangiopathy (i.e., primary sclerosing cholangitis, bile duct damage)

1. *Silybum marianum* (milk thistle) seed, standardized, 1,200 mg 3x daily.
2. *Salvia miltiorrhiza* tincture, 1 teaspoon 3x daily.
3. *Curcuma longa* (turmeric), 750 mg 3x daily.

E. Hepatitis (inflammation of liver): *Silybum marianum* (milk thistle) seed, standardized, 1,200 mg 3x daily.

7.0 Fever

A. General

1. *Eupatorium perfoliatum* (boneset), as a hot tea, as often as needed.
2. *Sambucus* spp. (elder) flower, as a hot tea, as often as needed.
3. *Mentha piperita* (peppermint), as a hot tea, as often as needed.
4. *Corallorhiza maculata* (coral root), or equivalent species, tincture, 30 drops (full dropper) each hour depending on the severity, and/or . . .
5. *Achillea millefolium* (yarrow), as a hot tea, as often as needed, or as a tincture, 10–30 drops as often as needed.
6. *Cryptolepis sanguinolenta* tincture, ½–1 teaspoon 3–4x daily.

B. If severe, add:

1. Wash with cool cloth or soak in tub until fever lowers, and/or . . .
2. Dosages of the above herbs may be increased if the fever is very severe.

C. For relapsing/recurrent fever (shaking chills/alternating sweats): *Eupatorium perfoliatum* (boneset) tea, 3–6 cups daily.

D. For temperature fluctuations in the body: serrapeptase, 120,000 SPU 3x daily.

8.0 Eye Problems

A. Specific for infected conjunctiva: *Isatis* spp. infusion eyewash (prepared as with nettles; see page 132), 1–2 drops in each eye 3x daily. Keep the infusion refrigerated; it will last a week. (If necessary you can also prepare isatis as a decoction for a stronger antiviral effect.)

B. Supportive (and for blurry vision)

1. Vitamin C, 1,000 mg, 3x daily, and . . .
2. Zinc, 25–50 mg once daily, and . . .
3. Lutein, 50 mg 3x daily, and . . .
4. Bilberry (*Vaccinium myrtillus*), 500 mg 2x daily.

C. Floaters

1. *Stephania tetrandra* tincture, ½ teaspoon 3x daily, and/or . . .
2. Chlorella, 1 tablespoon 3x daily, and/or . . .
3. Zeolite, 15 drops liquid 3–4x daily, or 2 heaping teaspoons powder daily, or 3 capsules daily.

D. Photosensitivity

1. Melatonin, 3–9 mg daily, and . . .
2. *Leonurus cardiaca* (motherwort) tincture, ½–1 teaspoon 3–6x daily, and/or . . .
3. *Hericium erinaceus* (lion's mane) tincture, ¼–½ teaspoon 3x daily, and/or . . .
4. Lichi berries, eaten throughout the day.

9.0 Pain

A. General

1. Pea protein, one scoop every 8 hours (I use the Jarrow Formulas brand), and/or . . .
2. Homeopathic Bryonia 30C 4 pellets 4x daily, and/or. . .
3. Homeopathic Arnica 30C, 4 pellets 4x daily, and/or . . .
4. Homeopathic Hypericum, 4 pellets 4x daily, and/or . . .
5. *Corydalis* spp. tincture, ⅛–¼ teaspoon 3–4x daily (contraindicated in liver disease), and/or . . .
6. *Monotropa uniflora* (Indian pipe) tincture, ¼–½ teaspoon hourly or as needed, and/or . . .
7. *Corallorhiza maculata* (coral root), or equivalent species, tincture, ½–1 teaspoon up to 6x daily, and/or . . .
8. *Verbena officinalis* (vervain) tincture, ¼–1 teaspoon as needed, and/or . . .
9. *Leonurus cardiaca* (motherwort) fresh plant tincture, 1 teaspoon–½ ounce (yes, ounce) in water as needed, and/or . . .

10. *Pedicularis bracteosa* (lousewort) tincture, 1 teaspoon–½ ounce (yes, ounce) in water as needed.

10.0 Muscle Weakness

A. General

1. Combination tincture formula: equal parts of *Pinus* spp. (pine) pollen, *Aralia nudicaulis* (or equivalent species), and *Panax quinquefolius* (American ginseng). Dosage: 30 drops (full dropper) of the tincture formula 3x daily for 6 months (take by mouth, let sit a minute, then swallow; do not dilute in water), and/or . . .
2. L-carnitine, 1,000 mg 3x daily, and/or . . .
3. Taurine, 500–1000 mg 3x daily, and/or . . .
4. Homeopathic Lycopodium 30C, 4 pellets 4x daily.

11.0 Swollen Lymph Nodes/Sluggish Lymph

A. General

1. *Salvia miltiorrhiza* tincture, 1 teaspoon 3x daily, and/or . . .
2. *Phytolacca americana* (poke) root tincture, 5–10 drops 2x daily, and/or . . .
3. *Galium aparine* (cleavers) tincture (especially for nodules and cysts), ½ teaspoon 3x daily.

12.0 Kidneys

A. To repair or inhibit further damage: *Urtica dioica* (nettles). Make 1 quart of nettle infusion (see below) and drink throughout the day, every day. As well, take ¼ teaspoon nettle seed tincture 3x daily, every day.

Nettle Infusion

To make:

Add 1–2 ounces of dried nettle leaf to a quart mason jar. Fill the jar with hot water, let steep overnight, strain, and drink throughout the next day. (Some people think the herb can be used again at least once more.) Note: For years I was curmudgeonly in response to (i.e., highly suspicious of) occasional claims I heard about nettles being able to heal kidney

damage. However, my partner Julie McIntyre has been suggesting it in practice for some time and has reported significant healing of damaged kidneys, in one instance so much so that dialysis was avoided. I rather shamefacedly stand corrected.

B. Frequent urination

1. *Verbascum thapsus* root tincture, 10–30 drops up to 6x daily. Will help restore bladder tone over time. Best, in this instance, if used with an endothelial normalizer and protectant such as *Salvia miltiorrhiza* or *Polygonum cuspidatum* tincture daily.
2. Ba Wei Di Huang Wan (TCM formulation), as directed on the product label (try Acupuncture Atlanta as a source).
3. Caprylic acid, taken as directed on the product label.

C. Proteinuria: Combination tincture formula of equal parts of *Salvia miltiorrhiza, Astragalus membranaceus,* and *Angelica sinensis*. Dosage: 1 teaspoon 3x daily.

D. Hematuria: same as section C above.

E. Elevated serum creatine: Avarai kudineer (an Ayurvedic blend; you can find it by searching online). Put 100 grams in 30 ounces of water, bring to a boil, reduce to a simmer, and simmer until reduced by two-thirds (note: boiling down is not a perfect calculation; all of us guess). Let cool, then press the liquid out of the herbs and refrigerate. Dosage: 1 tablespoon 3x daily until gone (approximately 10 days).

F. Elevated urea nitrogen: same as section E above.

G. Extreme thirst: Ba Wei Di Huang Wan (TCM formulation), as directed on the product label (try Acupuncture Atlanta as a source).

H. Fibrosis: see 13.0 below.

13.0 Fibrosis in Organs

A. To inhibit, reduce, or repair fibrosis in organs: Combination tincture formula of equal parts of *Angelica sinensis, Salvia miltiorrhiza, Lonicera japonica, Polygonum cuspidatum, Cordyceps* spp. Dosage: 1 teaspoon 3–6x daily depending on severity of fibrosis.

14.0 Reproductive System

A. Male

1. Hypogonadism (low testosterone)
   a. Pine pollen tincture, ¼ teaspoon held on the tongue for 1 minute, then swallowed, at least 3x daily.
   b. *Eurycoma longifolia* (tongkat ali) capsules, 1,000 mg daily.
2. Low libido: Pine pollen tincture, as above.
3. Erection problems
   a. Pine pollen tincture, as above.
   b. *Eurycoma longifolia* capsules, as above.
4. Orchitis: Long Dan Xie Gan Tang (TCM formulation), taken as directed on the product label.
5. Infertility: Combination protocol of the following:
   a. *Cornus officinalis* (Chinese dogwood) tea, daily.
   b. *Tribulus terrestris*, 250 mg, 3x daily.
   c. Speman (a traditional Ayurvedic formulation), 2 tablets 3x daily for 3 months.
   d. L-carnitine, 500–1,000 mg daily.
   e. L-arginine, 500–3,000 mg daily.
   f. Zinc, 25 mg daily.

B. Female

1. Severe cramping
   a. *Viburnum opulus* or *V. prunifolium* (cramp bark, black haw) tincture,½–1 teaspoon 6x daily.
   b. *Actaea racemosa* (a.k.a. *Cimifuga racemosa*, black cohosh), 30–40 drops of the tincture up to 2x daily or 300–1,000 mg daily.
   c. *Zingiber officinale* (ginger) capsules, 1,500 mg daily.
   d. *Leonurus cardiaca* (motherwort) tincture, ¼–1 teaspoon as needed.
   e. *Vitex agnus-castus* (chasteberry), 400–1,000 mg or 60–120 drops of the tincture daily. A tonic; builds in effectiveness over several months.

2. Menstrual irregularity
   a. *Vitex agnus-castus* (chasteberry), 400–1,000 mg or 60–120 drops of the tincture daily.
   b. *Alchemilla vulgaris* (lady's mantle) tincture, ½–1 teaspoon 3x daily.
   c. *Angelica sinensis* (dong quai), 500–1,500 mg 3x daily or ¼–½ teaspoon of the tincture 3x daily.
3. Infertility: *Vitex agnus-castus* (chasteberry), 400–1,000 mg or 60–120 drops of the tincture daily.

15.0 Musculoskeletal Problems

A. Myalgia (muscle pain)

1. Pea protein, one scoop every 8 hours or as needed. (Note: I use the Jarrow Formulas brand.)
2. *Piper methysticum* (kava) 10:1 extract, instant powder, in a cup of hot water (with honey and cream), as needed or desired.
3. Cannabidiol (CBD) or *Cannabis* spp., as needed.

B. Arthralgia (joint pain)

1. *Boswellia carteri*, 1,000 mg 2x daily, or in acute cases, 2–3x daily. I prefer the Superior Labs formulation (which you can find by searching online), which contains 500 mg boswellia, 100 mg L-leucine, and 7.5 mg of a piperine extract. The essential oil of boswellia (frankincense) applied topically may also help.
2. Bromelain, 500 mg daily or, in acute cases, 3x daily. I prefer the Toniiq brand (again, you can find it online).
3. *Piper methysticum* (kava) 10:1 extract, instant powder, in a cup of hot water (with honey and cream), as needed or desired.
4. Cannabidiol (CBD) or *Cannabis* spp., as needed.

C. Tremors and shaking: see 3.0, section G (page 120).

16.0 Skin Problems

A. Dry skin

1. Increase intake of fats and oils, specifically avocado, olive oil, coconut oil.
2. Nettle leaf tea, daily, in quantity.
3. Burdock root tea, daily, in quantity. Or capsules, 900 mg 3x daily.

B. Rash

1. General:
   a. *Galium aparine* (cleavers) tea, daily, in quantity. Or fresh plant tincture, ½–1 teaspoon 3–6x daily.
   b. Any of the following homeopathics can help, sometimes very much so: Apis, Hepar sulphur (Hep), Caladium (especially in asthma or lung disease), Histaminum, Arsenicum. All 30C, taken as directed on the bottle.
2. With itching: homeopathic Sulphur or homeopathic Psorinum, both 30C.

C. Easy bruising: Endothelium-protective and regenerative herbs will help (*Polygonum cuspidatum* and *Salvia miltiorrhiza*, especially), but also:

1. Hesperidin, 100 mg 2x daily.
2. Rutin, 50–100 mg 2x daily.
3. Diosmin, 900 mg daily.
4. Pycnogenol, taken as directed on the product label.

D. Extreme skin sensitivity

1. *Leonurus cardiaca* (motherwort) tincture, 1 teaspoon 3–6x daily, and/or . . .
2. *Corallorhiza maculata* (coral root), or equivalent species, tincture, ½–1 teaspoon up to 6x daily, and/or . . .
3. *Pulsatilla patens* (pasque flower) tincture, 10 drops as needed, usually no more often than once per hour, and/or . . .
4. *Gastrodia elata* tincture, ¼–½ teaspoon 3–6x daily, and/or . . .
5. *Piper methysticum* (kava) 10:1 extract, instant powder, in a cup of hot water (with honey and cream), as needed or desired.

E. Hair loss

1. Nettle leaf tea, in quantity, daily.
2. Pea protein powder, taken as directed on the product label. (Note: I use the Jarrow Formulas brand.)
3. Nutritional yeast (*not* enriched or fortified), 1 tablespoon daily.

17.0 To Lower Histamines/Stabilize Mast Cells

A. General

1. *Petasites hybridus* (butterbur), 50 mg 3x daily, and/or . . .
2. Inositol, 600 mg 2x daily.

B. Specific: *Stachys palustris* (marsh woundwort) tincture, 1 teaspoon 3x daily; excellent but hard to get.

C. NasalCrom inhaler, as needed.

18.0 Generalized inflammation and pain

A. General: Low-dose naltrexone (LDN). Note: Many integrative physicians are using LDN for the treatment of chronic fatigue syndrome with accompanying myalgia and systemic inflammation. It does seem to work well for some people. I order mine from China; some very good supply companies can be found online. It is best, I think, to begin with 1 mg capsules for several weeks, then go to 2.5 mg for a few weeks, then to 4 mg. You can find more information on patient experiences online.

19.0 Postural orthostatic tachycardia syndrome (POTS)

A. General

1. *Leonurus cardiaca* (motherwort), ½–1 teaspoon up to 6x daily.
2. Micronized purified flavonoid fraction (MPFF), 500 mg, 1–3x daily.
3. Diosmin/hesperidin blends, taken as directed on the product label.
4. *Polygonum cuspidatum* (Japanese knotweed) root tincture, ½ teaspoon up to 6x daily.
5. *Scutellaria lateriflora* leaf tincture, ½–1 teaspoon up to 6x daily.
6. Low-dose naltrexone (see 18.0 above). Best if started low and then increased: 1 mg for 2 weeks, 2.5 mg for 2 weeks, then 4 mg.

20.0 Dysautonomia

A. Adaptogenic herbs

1. *Eleutherococcus senticosus* tincture, in a 1:5 formulation (not the stronger 1:1 or 2:1), ½–1 teaspoon 3–6x daily, or capsules, taken as directed on the product label.
2. *Rhodiola* spp. tincture, ½ teaspoon 3x daily (may cause dizziness at higher doses).
3. *Schisandra chinensis* tincture, ½ teaspoon 3–6x daily, or capsules, taken as directed on the product label.
4. *Withania somnifera* (ashwagandha) tincture, ½–1 teaspoon 2x daily, or capsules, taken as directed on the product label. May cause drowsiness; best taken in the late afternoon and at night before bed.

21.0 Thermoregulation dysfunction

A. General: Microdosed psilocybin.

B. Specific: Serrapeptase, 120,000 SPU 3x daily.

C. Female: *Vitex agnus-castus* (chasteberry), 400–1,000 mg daily, or 60–120 drops of tincture daily. A tonic; builds in effectiveness over several months.

Final Comment: Breathing Exercises

Retraining your breathing pattern can reduce many of the symptoms of post-coronavirus syndrome. After Covid-19 damage to the lungs, deep belly breathing has been found to substantially increase oxygen levels. (Despite medical practitioners now taking credit for this in the treatment of long haulers, many psychotherapeutic and bodywork disciplines in the United States developed belly breathing for use in their practices for the treatment of a wide range of conditions in the 1970s; yoga and qigong practitioners did so in the East centuries earlier.) As earlier practitioners realized, this also helps reduce or reverse fatigue, shortness of breath, lymphatic problems, tachycardia, dizziness, anxiety, and brain fog.

To begin, you need to understand the difference between chest and belly breathing. Lie down and place your hand on your belly. Breathe in and out, slowly and deeply, until you feel your hand rise up and down with your breath. When you breathe into your chest only, the belly does not move. Especially if you are severely fatigued, start doing this lying down. After a while, do it sitting up. Then practice it standing, then walking.

Every morning: Inhale through the nose for four counts, exhale through your nose for six. Do this five to ten times.

Every evening: Inhale for four counts, hold for four, exhale for four. Do this five to ten times. (You can increase the number of times you do it as you gain experience.)

Note: If you cannot do these breathing exercises five to ten times, just do them as many times as you can. Don't push yourself. You are just retraining your body's breathing patterns. Some people find that inhaling for a two count, then breathing out through pursed lips for a four count works better for them. The point is to train your body to engage in belly (a.k.a. diaphragmatic) breathing as a normal behavior.

4

VIRAL ENCEPHALITIS INFECTIONS AND THEIR TREATMENT

Emerging RNA viral pathogens such as West Nile virus (WNV), Japanese encephalitis virus (JEV), Australian bat Lyssavirus, retroviruses, and Nipah virus have become increasingly important causes of encephalitis. . . . Worldwide, the flavivirus JEV is the most common cause of arthropod-borne encephalitis with over 50,000 cases reported per year in China, Southeast Asia, and India. Epidemics due to this arbovirus result in a mortality rate that ranges from 30 to 50% with death usually occurring within the first week, and the development of sustained neurological deficits in approximately half of the survivors. Another flavivirus, WNV, is also transmitted by Culex *spp. mosquitoes and can cause fatal encephalitis or long-term neurological sequelae. Once inside the CNS, JEV and WNV infect neurons leading to neuronal apoptosis and causing severe immunopathology.*

—Samantha Furr and Ian Marriott, "Viral CNS Infections"

West Nile virus (WNV) has expanded in the last 12 years worldwide, and particularly in the Americas, where it first occurred in 1999. It has extended throughout the Americas relentlessly since then, causing a severe epidemic of disastrous consequences for public health, wildlife, and livestock.

—M. A. Jiminez-Clavero, "Animal Viral Diseases and Global Change"

***Encephalitis* sounds like a technical term** but it isn't really. It simply means inflammation (the "itis" part) of the brain ("encephal," meaning brain, from an ancient Greek root). As with hepatitis, whose name merely means inflammation of the liver, there are many things that can cause such inflammations; the term refers not to a disease per se but merely to a primary symptom that occurs during the disease process.

There are a range of viruses that can cause encephalitis; many of them are known by that particular symptom—West Nile encephalitis and Japanese viral encephalitis are some examples. The viruses that cause these conditions have an affinity for the brain; it's their preferred habitat. Many of them, such as the Japanese encephalitis virus (JEV), have a tropism for neurons in the brain; they reproduce most easily inside them. So once they infect a person, that is where they like to go. Once established in the brain or central nervous system (CNS), they begin to release (or stimulate the release of) cytokines. Cytokines cause particular cells in the brain to swell and burst apart (the inflammation aspect of things). This gives the viruses the nutrients they need and facilitates their spread. (If the meninges, the sheath covering the brain and spinal cord, also inflames, which it sometimes does, the infection is called meningitis.) Unfortunately, the damage to the brain neurons causes a number of problems, some serious, in whomever the virus has infected. One of the most difficult is the inflammation itself. The brain is encased in a solid shell that, itself, cannot expand. So, if the inflammation is such that the brain is forced to swell beyond the size of its container, well . . . things get dicey.

Technological medicine has a limited capacity to respond to this kind of infection. During a minor infection, the usual response is to tell the person to stay at home and rest in bed. If the condition is severe, as it becomes for many of the viral encephalitides, there is no accepted pharmaceutical treatment. In some cases, but not most, antivirals will help. In others, the treatments are new and rather experimental, and only offer limited help. If the swelling becomes threatening, intravenous steroids to reduce the inflammation may be used, though there are a number of very serious side effects to their use if they are needed long term.

Plant medicines, on the other hand, offer a much more elegant intervention, especially for this kind of condition. With plant medicines, each aspect of the disease can be addressed. Antivirals can be used to kill the

organism (or limit its ability to infect the body's cells). Anti-inflammatories, specific for the brain, can be used to reduce inflammation. Neural protectors can be used to safeguard brain structures from damage (or to restore damaged structures). Immune facilitators (adaptogens) can be used to enhance immune function so the body is more able to fight the infection on its own. And the unique symptom picture that emerges for the infected person can be addressed through the use of plant symptomatics. Too, most of these plant medicines tend to be synergistic with one another (as many of the individual plant compounds are with one another) and, together, produce outcomes that can be very potent. With time, the ability to respond to viral infections can become tremendously sophisticated.

This is an initial exploration of how such an approach can be applied to viral encephalitis.

The Encephalitis Viruses

There are some viruses that specifically infect the brain and cause encephalitis (West Nile) and there are others that sometimes get into the brain and cause encephalitis (herpes simplex). Many of them are spread by insect vectors (mosquitoes and ticks). For some of them, there are vaccines (tick-borne encephalitis—TBE). Others will sometimes respond fairly well to pharmaceuticals (acyclovir for instance for nonresistant herpes encephalitis). Most of the viruses that cause encephalitis show similar initial symptoms; all of them can be treated with plant medicines. (And yes, pharmaceuticals can be used along with plant-based protocols. The plant protocol tends to *increase* the effectiveness of the pharmaceutical protocol while at the same time significantly relieving the symptom picture.)

Here is a look at the most common encephalitis viruses and their geographical distribution:

- **Togaviruses, the alphavirus complex:** eastern equine encephalitis (eastern and Gulf coasts of the United States, Caribbean, South America), western equine encephalitis (western United States and Canada), Venezuelan equine encephalitis (South and Central America, Florida, southwest United States). All are spread by mosquitoes.

- **Flaviviruses, the West Nile complex:** St. Louis encephalitis (United States), Japanese encephalitis (Japan, China, southeast Asia, India, southeast Russia, northern Australia, New Guinea), Murray Valley encephalitis (Australia and New Guinea), West Nile (United States, Africa, Europe, Middle East, Asia), Ilhéus (South and Central America), Rocio (Brazil). All are spread by mosquitoes.
- **Flaviviruses, the tick-borne complex:** Far Eastern tick-borne encephalitis (eastern Russia), central European encephalitis (central Europe), Kyasanur forest disease (India), louping ill (England, Scotland, northern Ireland), Powassan (Canada and northern United States), Negishi (Japan). All spread by ticks. In general, this group is usually just referred to as TBE or tick-borne encephalitis virus and treated accordingly.
- **Bunyaviruses, the bunyavirus complex:** California encephalitis (western United States), La Crosse encephalitis (mid- and eastern United States), Jamestown Canyon (United States, including Alaska), snowshoe hare (Canada, Alaska, northern United States), Tahyna (Czech Republic, Slovakia, former Yugoslavian states, Italy, southern France), Inkoo (Finland). All spread by mosquitoes.
- **Phlebovirus:** Rift Valley fever (eastern Africa). Spread by mosquitoes.
- **Reovirus, orbivirus:** Colorado tick fever (Rocky Mountains, western United States and Canada). Spread by ticks.

The most common in the United States are West Nile, St. Louis encephalitis, La Crosse encephalitis, and California encephalitis. Japanese encephalitis virus (JEV) is the most common in Asia (estimates are that in some areas the infection rate is 100 percent). From all reports, it is the main member of the common viral encephalitides that causes the most damage. The most common encephalitis viruses in Europe and Russia are West Nile and TBE; West Nile is, in fact, becoming a worldwide phenomenon and is present in most of the world. Most of the other encephalitis viruses are somewhat uncommon in people. Vaccines have been created for both Japanese viral encephalitis and TBE but they can't be used on those younger than 15 to 17 years of age.

In spite of the existence of vaccines, there are large outbreaks of both Japanese viral encephalitis and TBE every year, especially in the

young. The vaccine needs to be repeated every year (Japanese) or every 3 years (TBE). For many cultures, the cost is often prohibitive and many people simply never get the vaccines. There are also certain groups of people for whom it is considered risky to give the vaccine, primarily the old and the young. Specifically: 1) those over 60 years of age; 2) those allergic to formaldehyde, neomycin, gentamycin, protamine sulfate, latex, eggs, or chickens; 3) those with a brain or central nervous system problem; 4) those with high temperatures; 5) those with weakened immune systems; 6) those with autoimmune conditions; 7) women who are pregnant; 8) those under 15 years of age (17 for the Japanese viral encephalitis vaccine). There remain a large number of people who can't be vaccinated, many of whom succumb to the infections each year.

Symptoms

Nearly all, 90 percent, of people infected by an encephalitis virus have flu-like symptoms: fever, sore throat, cough, general ill feeling, aches and pains, weakness, and malaise. About 10 percent have no symptoms at all. And for most people the infection passes, just like the flu; they really have no idea that they were infected with viral encephalitis.

If the infection is severe, the symptom picture alters accordingly: Headache, confusion, disorientation, photophobia, agitation, optic neuritis, myelitis, personality alterations, seizures, coma, and death can all occur, depending on the severity of the infection. Depending on which part of the brain is affected, language skills may be impaired and voluntary movement affected, and severe muscle weakness, uncontrollable tremors, partial paralysis, involuntary movements, and loss of ability to regulate body temperature may occur.

Medical Treatment

Most neural encephalitis infections such as Japanese viral encephalitis and West Nile encephalitis have no established pharmaceutical treatment approaches—though there are a few treatments that are showing promise. Minocycline does work to some extent in Japanese viral encephalitis. It modulates the degree of infection and the persistence

of the virus, reduces the inflammatory cytokines in the brain, and is neuroprotective. It also reduces viral replication. Although not yet in common use, it is probably the best treatment approach for severe JEV infections.

Immunoglobulin therapy has helped with a number of different viral encephalitides including West Nile. Interferon-alpha and ribavirin have also been used, though with mixed to moderate success for West Nile. Minocycline has been found effective in West Nile encephalitis and I think it a good treatment approach if the disease becomes serious. Erythromycin can significantly reduce the endothelial hyperplasia that sometimes occurs during West Nile encephalitis.

Anticonvulsant medications are sometimes used to treat seizures. Steroid medications, IV, are used for inflammation in severe cases.

Some of the nonspecific viruses that can sometimes infect the brain, such as herpes, do respond to pharmaceuticals. The usual treatment for them is antivirals: acyclovir (for herpes simplex, Epstein-Barr, and varicella zoster encephalitis) and ganciclovir (for cytomegalovirus and herpes simplex 1 encephalitis).

Mechanisms of Viral Infection

Japanese encephalitis virus (JEV), West Nile virus (WNV), tick-borne encephalitis virus (TBE), St. Louis encephalitis virus, and dengue virus are all flaviviruses, they are closely related, and they have very similar impacts in the body. In other words, with slight differences, what they do once they are in the body, how they affect brain structures, and the kinds of cytokines they stimulate are all very similar. (The treatment protocol for each, in consequence, is also very similar.) I will look in the most depth at JEV and WNV, then just a bit at TBE (which, being tick spread, has some unique features) and a bit at La Crosse, the most common non-flavivirus encephalitis virus. I take a brief look at dengue in general in the next chapter with just a touch on its encephalitis aspects in this one.

Japanese Encephalitis Virus: Initial Infection

Japanese encephalitis virus (JEV), sometimes called encephalitis B, is the best studied of these viruses. At the time of this writing there

are over 4,300 journal articles about it in the online medical database PubMed and another 1,600 at the fledgling Chinese database CNKI. It has been rather intensively studied since the 1930s. It's spread, as most viral encephalitides are, by mosquitoes.

Once the mosquito bites a person, the virus enters the body while the mosquito feeds, then travels to the liver, spleen, and lymph system over the next 5 to 15 days (the incubation period). As with tick-borne infections, the virus uses the saliva of the arthropod vector to facilitate its entry into the body. The chemicals in mosquito saliva interfere with host immune responses, downregulating interferon responses and T cell activation and immune cell numbers. This allows the virus to bypass the immune system, get into the lymph system, and spread throughout the body.

Once inside the spleen it begins infecting dendritic cells and monocytes/macrophages. It is able to replicate, slowly, inside these cells even though they are not its preferred habitat. It uses the cells primarily to foster its invasion of the body.

JEV uses the dendritic cells to begin its subversion of the host immune system, essentially by inhibiting the ability of the dendritic cells to mature. Dendritic cells mature or develop in specific ways when they detect infections in the body. In this particular instance the lack of maturity stops dendritic cell activation of T cells; activated T cells and immunoglobulin M (IgM) play a major role in relieving the body of viral infection and reducing neuronal damage. The most active T cells for this kind of viral infection are those that express CD8+ proteins on their surface. These cytotoxic T cells, or killer lymphocytes, are a type of white blood cell that is very good at killing cells infected with viruses.

While inhibiting the activation of these T cells, the virus begins to infect monocytes/macrophages. These essentially act as transport mechanisms for the virus, taking it into the brain, where it can more easily spread. JEV releases a number of specific compounds to increase the permeability of the blood-brain barrier, thus easing its passage into the brain. Once inside the brain it moves outside the macrophage/monocyte cells and begins to attach itself to brain neurons. JEV has the strongest tropism for neurons in the thalamus, cortex, striatum, hippocampus, and midbrain, which are the parts of the brain impacted most

severely by the infection. The virus reaches peak levels in the brain on the sixth day (11 to 20 days postinfection).

Once attached to the surface of a neuronal cell, the viruses stimulate endocytosis—the process by which cells take molecules into themselves (engulfment). Once inside the neurons, the viruses begin to replicate. The viral particles also begin attaching themselves to microglia, the resident macrophages of the brain and spinal cord. These are the primary immune defensive forces of the CNS. Microglia, like most macrophages, release a number of chemicals during infection that are designed to kill invading organisms. But many microbes have learned to use the host defense system for their own purposes. These kinds of encephalitis viruses are no exception. JEV attaches to the surface of the microglia, stimulating them to release cytokines and chemokines. The virus uses the microglia primarily as a source of inflammation *and* as a reservoir site in the brain.

The astroglia (astrocytes) are often infected as well. These cells are tremendously abundant in the brain and do a number of things, among them keeping the blood-brain barrier intact, providing nutrients to neurons, maintaining ion balance, and, most importantly, repairing damaged neurons. JEV infection of the astroglia inhibits their repair functions and stimulates them, as well, to release a number of cytokines: IL-6, RANTES, and MCP-1. The primary purpose of their infection appears to be inhibition of their maintenance and repair functions. However, more sophisticated research has found that there is considerable virus-stimulated synergy between the cytokine cascades from the astrocytes and microglia. A sort of cross-talk occurs that promotes what researchers call a "fulminate inflammation," which the virus uses to its advantage.

NEURONAL DAMAGE

After JEV is established in the brain, it begins to cause the death of neurons. This is the main source of the symptoms that occur from infection and it results from two factors: 1) direct infection of neurons by the virus, and 2) the overactive immune responses initiated by the virus after it attaches to the microglia.

Once attached to a neural cell, the virus stimulates the production of sphingomyelinase. This is an enzyme that breaks down sphingomyelin,

a kind of lipid that makes up some of the structural elements of cells, especially the myelin sheath that surrounds nerve cells. This causes a variety of neurological problems; for example myelin sheath degradation is common in diseases such as Parkinson's disease. Sphingomyelinase inhibitors (such as cordyceps, which is a very potent sphingomyelinase inhibitor) can reduce or eliminate the damage. As the sphingomyelin is broken apart the cellular membrane becomes more porous but, as well, one of sphingomyelin's major consituents, ceramide, is released.

> A defining feature of viral CNS infection is the rapid onset of severe neuroinflammation and overzealous glial responses. [These] are associated with significant neurological damage or even death. . . . [There is] the possibility that cross-talk exists between these disparate viral sensors and their signaling pathways [and that they can] act in a cooperative manner to promote the fulminate inflammation associated with acute neurotropic viral infection.
>
> —Samantha Furr and Ian Marriott, "Viral CNS Infections"

Ceramide has a number of functions in the cell and body. Its release is intimately involved in apoptosis, or cellular death. It also is deeply involved in the creation of lipid rafts. These rafts can cross the entire lipid bilayer of cells, allowing things from outside the cell (usually molecules) to enter into the cell. The virus attaches itself to the lipid rafts and rides them into the cell.

Once inside the cell, the virus begins reproducing in the cytoplasm, the space inside the cell. The rough endoplasmic reticulum (RER), which covers the outer surface of the nucleus, is used as a sort of uterus for the newly developing viruses; they bud from its membranes as they develop. The RER is also stimulated to proliferate and the resulting hypertrophy makes more space for the viruses to create offspring. Most of the viruses (about 60 percent) remain in this location, while another 20 percent invade the Golgi apparatus that lies between the inner side of the cell and the nucleus. The Golgi apparatus's primary function is to modify proteins delivered from the RER. It generates glycosaminoglycans and proteoglycans. These are forms of sugar molecules that are used structurally in the body. The Golgi apparatus also generates and stores Bcl-2.

The remaining 20 percent of the viruses invade the nucleus of the cell, specifically the nucleolus. In this location, they scavenge RNA particles from the nucleus to facilitate their reproduction. There is a constant movement between the viral particles in the nucleus and those in the cytoplasm where the scavenged RNA is exchanged and used to form new viral particles.

All the infected cell's functions are tightly controlled by the virus. It controls cell life and cell death. Initially it slows the ability of the infected cell to die but once enough virus particles have been reproduced, it stimulates cellular death. The cell bursts apart and a swarm of viral particles is released and the process begins again. The virus uses a highly elegant control of cellular signaling molecules to accomplish this.

The infected cell is stimulated to begin a signaling cascade of highly bioactive molecules: p38 MAPK and a number of caspases are activated. These stimulate the production of CHOP (C/EBP homologous protein), which ultimately is used by the virus to cause cell death. The death of these neuronal cells is the main cause of the damage the disease does and the source of many of the symptoms. (The use of p38 MAPK inhibitors—Chinese skullcap, houttuynia, cordyceps—significantly reduces neuronal cell death, slows the proliferation of new viral particles in the brain, and reduces the damage the disease does.)

The virus specifically downregulates Bcl-2, inhibiting its release by the Golgi apparatus. This stimulates the release of cytochrome c from the mitochondria, initiating a mitochondrial-controlled cellular death process that is tightly controlled by the virus. Bcl-2 downregulation causes the subsequent activation of caspase-8 and caspase-9 (caspases are sometimes referred to as the "central executioners" of cell death), a burst of free radicals, and significant depletion of intracellular glutathione in the cell. (Upregulation of Bcl-2, by such plants as Chinese skullcap, licorice, and rhodiola, can inhibit the process.) All this is part of the sudden cell death the virus causes once its numbers have reproduced sufficiently. (The use of mitochondrial protectors, such as kudzu root, can also interfere with Bcl-2 downregulation, subsequent cytochrome c release, activation of caspase-8 and caspase-9, the intracellular depletion of glutathione, and free radical production.)

MICROGLIAL CYTOKINE CASCADES

Once the virus reaches the brain, microglia are strongly stimulated by virus attachment and tremendously overactivated. The microglia begin producing an overabundance of IL-6, TNF-α, monocyte chemoattractant protein-1 (MCP-1), and RANTES (CCL5). This causes a massive leukocyte migration and infiltration into the brain. (The activated astrocytes produce upregulated levels of IFN-γ and RANTES, also contributing to the problem.) Manganese superoxide dismutase (SOD), IL-8, IL-12, IL-1ß, p38 MAPK, iNOS (inducible nitric oxide synthase), COX-2 (cyclooxygenase-2), IFN-γ, and NF-κB are also upregulated, making the problem worse. Levels of IL-10 and IL-4 (cytokine regulators) are initially high (which reduces the normal, initial antiviral response) and then are inhibited (to keep their balancing effect on inflammatory cytokine upregulation in check) and decline over the course of the illness.

The virus interferes with interferon signaling during infection, reducing the impacts of immune interferons alpha and beta on the infection. The virus also stimulates the production and activity of tyrosine kinase, which is one of the factors behind the production of TNF-α and IL-1ß cytokines.

The greater the levels of induced cytokines in the brain, the worse the outcome. The degree of long-term damage and incidence of mortality are directly proportional to the cytokine levels induced by the virus. Additionally, the cytokine cascade, especially NF-κB, TNF-α, IL-1ß, and IL-6, can stimulate the release of HMGB1 protein (see page 33). The damage to neuronal cells by the virus also causes HMGB1 release into the brain. This induces the release of inflammatory cytokines and excitatory amino acids such as glutamate, stimulates fever, and exacerbates ischemic injury. (The use of HMGB1 inhibitors such as licorice, *Angelica sinensis*, and *Salvia miltiorrhiza* is essential to stop the disease's progression to a generalized sepsis.)

Reducing the cytokine and chemokine cascades has been found to inhibit the course of the disease and reduce neural damage. Studies have shown that ERK and glutaminase inhibition helps prevent cytokine damage in the brain. And rosmarinic acid from *Rosmarinus officinalis*, which has been studied in some depth, has been found to be highly effective in reducing major elements of the cytokine cascade (IL-12, IL-6, MCP-1, IFN-γ, and IFN-α), thus protecting the brain and reducing the mortality levels in mice.

Comment: Rosmarinic acid is not very systemic; it is usually injected to get this impact in the CNS. As such systemic inhibitors of the cytokines are much preferred since they don't need to be injected. These include inhibitors of ERK (kudzu, Chinese skullcap, cordyceps); MCP-1 (houttuynia); RANTES (licorice, isatis); IL-6 (Chinese skullcap, astragalus, cordyceps); TNF-α (kudzu, Chinese senega root, Chinese skullcap, elder, ginger, houttuynia, licorice, boneset, cordyceps); NF-κB (Chinese senega root, Chinese skullcap, ginger, houttuynia, kudzu, licorice, boneset, astragalus); IL-1ß (Japanese knotweed, Chinese senega root, Chinese skullcap, cordyceps, kudzu, boneset); and IL-8 (cordyceps, isatis, Japanese knotweed).

In essence, flooding the CNS with inhibitors of the stimulated cytokines stops the progression of the disease and significantly reduces long-term impacts on the brain. Many of these herbs are known for their brain protective effects; they are systemically disseminated, cross the blood-brain barrier, and are specifically protective, in many instances, for brain neurons. Many of them also possess antiviral actions. Because they hit in so many areas of activity, they are the specifics to use for these kinds of viral diseases.

Of additional use are tyrosine kinase inhibitors (such as cordyceps and Japanese knotweed), which have been found to attenuate JEV-induced neurotoxicity. This does not stop JEV replication in neuronal cells but does stop the neuronal damage caused by cytokines. Sphingomyelinase inhibitors (such as cordyceps) slow the movement of viral particles into cells. Alpha-glucosidase inhibitors of the endoplasmic reticulum (strongly present in such plants as Chinese skullcap and rhodiola) have been found to stop the replication of the virus. And this is, in fact, one of the approaches the body itself uses eventually.

The body, in challenging the virus, ultimately does activate T cells in the spleen. Once activated, the T cells begin production of a T-cell-specific alpha-glucosidase inhibitor. This actively suppresses the replication of the virus but also tends to produce a lingering hypoglycemia during the postinfection stage of Japanese viral encephalitis.

Higher IL-4 and IL-10 levels (Chinese skullcap, elder, houttuynia, licorice, cordyceps) and splenic activation of T cells (red root, poke root, Chinese skullcap, houttuynia, astragalus, cordyceps) have both been found to produce milder infections. IL-10 has neuroprotective effects and helps reduce inflammatory damage.

POSTINFECTION DAMAGE

Survivors of severe Japanese viral encephalitis often have long-term neurological damage. This can include major cognitive impairment and motor and behavioral problems. Part of this comes from the damage to neural structures in the brain, but the virus also significantly reduces the levels of neural progenitor cells in the body. This significantly impairs recovery after the disease since the brain cannot use the progenitor cells to repair the viral damage. The virus initiates the activation of phosphoinositide 3-kinase/Akt signaling during the early stages of infection, which leads, in part, to this effect on progenitor cells. Inhibitors of this (cordyceps) can help alleviate the emergence of this long-term problem. The use of herbs that stimulate neural growth and contain nerve growth factor (Chinese senega root, lion's mane, *Knema laurina*) will help stimulate the production of new neural structures in the brain and significantly reduce postinfection damage.

West Nile Encephalitis

West Nile, in contrast to Japanese encephalitis, and in spite of the concern about it, has been much less explored by researchers. Although discovered in 1937 the first large outbreak did not occur until the 1990s; research did not begin in earnest until the latter part of that decade—after outbreaks occurred in the United States. Thirty-two hundred of the 3,600 studies on the virus have occurred since then; the Chinese database CNKI has a mere 74 journal articles on the virus. Oddly enough, in contrast to the Japanese encephalitis virus, there has been much less work on the cytokine cascade caused by West Nile virus (WNV). The virus is much more poorly understood and its exact dynamics inside the body and brain are less clear.

Nevertheless, WNV follows almost the same process during infection as JEV though it is not nearly so dangerous. Four in five of those infected have few or no symptoms at all. One in five experience a flu-like febrile illness of limited duration. One in 150 develops encephalitis or meningitis or both. In contrast to JEV, which is most active in the young, WNV has the most damaging effects in the aged. The older a person is, the worse the outcome and the greater likelihood they will be seriously ill. The virus seems to specialize in attacking the aging brain (JEV, in contrast, has a preference for immature and developing neural

structures). The younger the person, the younger the neural structures, the less likely the disease is to be severe, or even to be symptomatic. Children and young adults tend to be asymptomatic. The median age of those who present with just fever and flu-like symptoms is 43; the median age of those who develop severe symptoms is 59. The old are also especially at risk because the aging immune system is less strong (immune senescence) and, at the same time, they tend to have preexisting conditions that affect brain function (such as higher reactive oxygen species and other cytokines already active in the brain from Alzheimer's and similar conditions). The incubation period is about the same as for JEV, 3 to 11 days, with the average at 5 days.

Most of the people who experience severe infection have similar symptoms: muscle weakness, gastrointestinal symptoms, fever, headache, alterations in mental status, and movement disorders such as tremors, myoclonus, Parkinsonism. A smaller proportion have an erythematous macular rash of one sort or another that can appear in various places on the body. Meningoencephalitis is common. A polio-like paralysis appears in a small subset of those infected. If meningitis occurs, neck stiffness, headache, hypothermia, photophobia, and/or phonophobia are common. With encephalitis, especially severe, depressed or altered consciousness, lethargy, personality alterations, fever, and hypothermia are common.

Ribavirin and interferon-a2b are the most common pharmaceuticals used, but they show a very mixed success rate. The main intervention is hospitalization ("supportive" treatment, as most journal articles refer to it) and observation; there is little medical intervention. Mostly the people just get well on their own . . . or don't.

INFECTION PROGRESSION

After infection (by mosquito bite) nothing appears amiss for about 5 days or so but on day 6 or 7 lethargy sets in, food intake slows, weakness begins to develop. By days 7 to 10 (in severe cases) tremors and difficulty walking, thinking, and communicating start to occur.

As with Japanese encephalitis, the virus travels first to the spleen and thence to the CNS. The blood-brain barrier is made more permeable (as with JEV) and once into the brain the virus begins to seek out its preferred areas. Those are the cerebral cortex, cerebellar cortex,

subcortical gray matter, hippocampus, and basal ganglia. As the infection progresses the cerebral cortex and the hippocampus are more heavily affected; severe degeneration begins in the neurons in those areas, then escalates as the virus reproduces. The virus then spreads to the brain stem and spinal cord (which is when a general paralysis can occur).

There is direct infection of neurons and an inflammatory stimulation of the host immune system, which the virus uses during the disease process. In essence, it stimulates an autoimmune response to break down cellular structures in the CNS. The microglia are infected, and microglial nodules form, filled with lymphocytic infiltrate, the nodules surrounded by degenerated (exploded) neurons. As the disease progresses, the brain stem is more deeply affected and begins showing tremendous neuronal degeneration and the inflammation in the spinal cord is much more developed. The lumbar region of the spinal cord can become severely affected, with inflammatory infiltrates along the entire length of the cord. The brain stem, temporal lobes, and basal ganglia are, generally, the most severely affected regions of the brain.

In those who survive the illness, persistent neurological problems can remain. The virus can, in some, develop into a long-term, chronic infection that does not heal. Long-term chronic infection with West Nile is almost totally dependent on the failure of CD8+ T cells to become active or highly present in the body. As with Japanese viral encephalitis, the higher the CD8+ T cell activity prior to infection, the less chance there is for infection, especially serious infection, to occur.

THE CYTOKINE/CHEMOKINE CASCADE

The cytokine and chemokine profiles in the CNS are similar to those of Japanese viral encephalitis but there are differences. The cytokines TNF-α, IL-1ß, IL-5, IL-6, IL-8, IL-13, interferon-inducible protein-10 (IP-10), matrix metalloproteinase-1 (MMP-1), MMP-3, MMP-9, NF-κB, COX-2, and the chemokine MCP-1 are all increased. Interferon-gamma levels are strongly increased, as are the levels of IDO (indoleamine 2,3-dioxygenase), ICAM-1 (intercellular adhesion molecule-1), VCAM-1 (vascular cell adhesion molecule-1), E-selectin, p38 MAPK, RANTES, and PGE2 (prostaglandin E2).

Researchers have looked intensively at the presence of a number of chemokines in West Nile encephalitis (not explored in the development

of Japanese viral encephalitis); CCR1, CCR2, CCR5, CCL2, CCL3, and CCL4 all showed increases. IL-10 levels are initially upregulated by the virus upon infection in order to keep the innate immune response as low as possible. Once the virus is established in the body and cytokine levels begin to rise, the levels of IL-4 and IL-10 are inhibited in order to stop their cytokine regulatory actions. The longer the infection, the lower the levels become. Perforin levels are reduced as well. Perforin is a protein in cytotoxic T cells that creates pores in target cells that allows the T cells to enter the infected cells and kill them.

All in all, the infection cytokine dynamic is very similar to that of Japanese viral encephalitis. Three of the main differences, however, are the generation of matrix metalloproteinases (MMPs), cellular adhesion molecules, and IDO.

The virus stimulates the production of MMPs (MMP-1, MMP-3, and MMP-9) once it enters the spleen. The MMPs are a kind of enzyme that especially degrade extracellular matrix proteins. These proteins make up much of the connective tissue in animals, including crucial elements of the blood-brain barrier. In essence, once MMPs are activated in a location, the connective tissue in that location begins to degrade. West Nile uses these to degrade the blood-brain barrier enough that it can gain entry to the brain and spinal cord. The tight junction protein bonds are weakened, making the barrier more permeable. Once inside the brain the virus attaches much more strongly to astrocytes than JEV does and stimulates them to continue releasing MMPs. Astrocytes are, in fact, the main source of these compounds in the brain. (The endothelial cells of the brain's microvascular are also activated to produce MMPs.) This makes the blood-brain barrier significantly more porous, and keeps it that way. This creates an enhanced infiltration of immune cells into the brain, and they cluster around the sites of viral infection and contribute to the inflammation in those locations. The cellular structures are weakened, helping the virus gain entry to their host's cells. In other words, the virus makes the blood-brain barrier porous and actively damages neural structures, which stimulates the production of cytokines and chemokines, which then call to themselves a variety of immune cells at the specific neural locations the virus wants them. The use of MMP inhibitors (such as Japanese knotweed root, Chinese skullcap, cordyceps) has been found to stop the process by inhibiting astrocyte production of MMPs and restoring blood-brain barrier integrity.

WNV also manipulates cholesterol content in cellular membranes. It upregulates host cell cholesterol biosynthesis and redistributes the produced cholesterol into viral replication membranes.

The cytokines stimulated by the virus (specifically TNF-α and IL-1ß) in turn stimulate the production of ICAM-1, VCAM-1, and E-selectin by both astrocytes and endothelial cells in the brain. These are chemotactic for immune infiltrates that form the microglial nodules. Basically, they act like magnets, calling the immune cells to them. The leukocytes that are called to those locations bind to cells and then transmigrate into tissues, in this instance making the cells more accessible to the viral particles. PGE2, which is stimulated by COX-2, is strongly involved in the inflammation process and the aggregation of platelets; it has a number of potent impacts on spinal neurons and is a stimulant of IDO in the body. TNF-α inhibitors (kudzu, Chinese senega root, Chinese skullcap, elder, ginger, houttuynia, licorice, boneset, cordyceps) and IL-1ß inhibitors (Japanese knotweed, Chinese senega root, Chinese skullcap, cordyceps, kudzu, boneset) have all been found to stop this process. Inhibitors of ICAM-1 (Japanese knotweed, elder, cordyceps), VCAM-1 (Japanese knotweed, kudzu, elder, licorice), and E-selectin (Japanese knotweed, kudzu, licorice) can significantly reduce leukocyte infiltration and congregation.

The stimulation of the endothelial cells in the brain's microvascular network also causes a hypertrophy of those cells, interfering with blood flow to the brain. A central nervous system vasculitis can occur that can, sometimes, lead to stroke or other complications. Plants such as Japanese knotweed (or compounds such as EGCG and resveratrol), which are specific inhibitors of endothelial hypertrophy, can not only reduce MMP production but also inhibit this process. (Erythromycin is also specifically indicated for this.) Cerebral vasculitis from West Nile infection is somewhat more common than understood. In severe cases endothelial cell normalizers (Japanese knotweed root, EGCG, hawthorn berry, *Salvia miltiorrhiza*, licorice) really should be used. COX-2 (and PGE2) inhibitors are also helpful, especially because they help reduce IDO levels (Japanese knotweed, kudzu, Chinese skullcap, Chinese senega root, elder, ginger, houttuynia, isatis, cordyceps). COX-2 inhibitors also specifically block the production of WNV-induced cytokines in astrocytes (essentially the same herbs).

One of the first things that the virus does is to, in a specific process, increase levels of interferon-gamma (and TNF-α) in order to stimulate the production of indoleamine 2,3-dioxygenase (IDO). During WNV infection, high levels of IFN-γ, TNF-α, IL-1ß, and IL-2 are present in the brain. (TNF-α is synergistic with IFN-γ in stimulating IDO production.) Additionally, once the virus enters the spleen and infects dendritic cells it begins stimulating them to produce IDO (which reduces T cell proliferation, thus protecting the virus from the most effective immune response). This is one of the main differences between this virus and JEV. IDO is an enzyme that breaks apart (catalyzes) the amino acid L-tryptophan. There are a number of wide-ranging effects from this, all of which have deleterious effects in the body.

Normally, when infection occurs, the body creates IFN-γ to catalyze tryptophan because tryptophan degradation limits protein biosynthesis by depriving cells of an essential amino acid. But West Nile doesn't need tryptophan. It, instead, uses this natural immune response to limit T cell proliferation and to create specific, and very potent, inflammations in the brain. IDO is a potent inhibitor of T cells (it also induces apoptosis of competent T cells) and through its degradation of tryptophan creates powerful inflammatory molecules in the brain. The degree of tryptophan degradation is a specific indicator of the progression, and seriousness, of the infection.

L-tryptophan is normally degraded into L-kynurenine and then into three intermediary compounds that are highly neuroactive in the brain: 3-hydroxykynurenine (3-HK), quinolinic acid (QUIN), and kynurenic acid (KYNA).

High QUIN levels in the brain will cause an overstimulation of neurons, excitotoxic lesions, degradation of brain tissue, high levels of reactive oxygen species in the brain, and convulsions. The precursor to QUIN, 3-HK, is highly neurodestructive, causing cellular disintegration, primarily through the generation of free radicals. Neurons are particularly vulnerable to its actions. KYNA, on the other hand, is neuroprotective, and ameliorates the impacts of QUIN and 3-HK. Unfortunately, the amount of KYNA depends on healthy neuron function, which in WNV infection is inhibited severely. Compromised cellular energy metabolism (i.e., mitochondrial function) will also significantly reduce KYNA levels. In WNV infection 3-HK and QUIN levels are very high,

and KYNA levels are very low. Upon microglial and astrocyte activation 3-HK and QUIN levels can increase 100 to 1,000 times; this is especially true if there is the kind of macrophage infiltration that occurs during West Nile encephalitis. The number and seriousness of seizures, convulsions, and paralysis that people with West Nile experience are directly proportional to the level of IDO generated in the brain (and its subsequent generation of QUIN and 3-HK).

IDO stimulation also has the regrettable ability to significantly inhibit serotonin and melatonin production in the brain. Melatonin, besides its potent sleep regulatory actions, is also an incredibly strong antioxidant, more powerful than most. It very specifically deactivates the oxidants that 3-HK and QUIN create. During WNV infection, however, melatonin levels fall very low in the brain. (Keeping melatonin levels high will reduce, or eliminate, the ability of West Nile—and other encephalitis viruses—to cause infections. Increasing melatonin levels, by using plants such as Chinese skullcap that are very high in melatonin, will reduce vulnerability to infection, symptom picture, and mortality. It specifically reduces paralysis and convulsions and is highly protective of the brain's neural structures.)

IDO inhibitors (and exogenous melatonin) can stop this process (*and* reduce IFN-γ and TNF-α levels), thus protecting brain tissues and reducing symptom severity, postinfection complications, and mortality. The most potent inhibitors of IDO are Chinese skullcap, Japanese knotweed, isatis, and, most especially, *Crinum latifolium*.

The high levels of IDO also create tryptophan depletion in the body. Tryptophan is an essential amino acid that cannot be synthesized by the body; it has to come from outside sources. During severe West Nile encephalitis, tryptophan supplementation should occur. (This will *not* feed the viruses, making the disease worse, but will, in fact, help repair CNS damage.) Studies have found that exogenous tryptophan can help restore healthy T cell function and responses.

There are several other interventions that have been found to reduce WNV impacts in the body. The inhibition of p38 MAPK (Chinese skullcap, houttuynia, cordyceps) significantly decreases chemokine production in response to WNV. CD8+ T cells require perforin to clear West Nile virus from infected neurons. Low perforin levels correlate to a much higher viral load, worse symptom picture, and increased

mortality. Increasing perforin levels (by using plants such as astragalus) stimulates clearance of the virus and reduces symptoms.

Tick-Borne Encephalitis

TBE is very similar to the other members of this group. It has a higher affinity for the lymph system, with subsequent involvement of the nodes. And this is where it first replicates before traveling to the brain (via the spleen and lymph system). The spleen is particularly infected (making the use of spleen herbs essential) and the virus can remain in the body, including the spleen, for months, perhaps years. Acute symptoms (beyond the initial flu symptoms) are hyperemia, petechial hemorrhages, inflammatory infiltration, necrosis of microglial cells, hyperplastic and hypertrophic glial nodules. Lesions are most common in the periventricular regions of the brain stem, including the cerebellum and reticular formation. The cerebellar cortex and basilar portions of the pons, thalamus, and olivary nuclei can also be affected. Perivascular inflammation around the blood vessels and cerebral interstitial edema are common. Laminar necrosis can occur. The cranial nerve and cervical anterior horn motor neuron cells are commonly affected during deeper progressions of the disease. Meningitis is common.

The cytokine/chemokine profiles are similar to those of other encephalitides: IL-6, TNF-α, IL-1ß, IL-1α are all high; IL-10 is initially high, then decreases substantially—as usual with these kinds of viruses. One difference is that if the pH of the blood moves from its normal alkaline state (around 7.3) to an acidic state, it strongly facilitates the ability of TBE virus to infect the body. Sodium bicarbonate, calcium citrate, and magnesium citrate (and isatis) supplementation will reliably increase pH and reduce the ability of the virus to fuse with host cells.

Presenting symptoms in serious cases are high fever, headache, vomiting, and vertigo. Then tremors, profuse sweating, paresis, delirium, psychosis, coma, and death. There is no pharmaceutical treatment for TBE though tetracycline hydrochloride has been found to significantly reduce the inflammatory cytokines during infection. Corticosteroids have as well been found to inhibit inflammation, decreasing the severity of symptoms.

There are two specific physiological differences during TBE (when compared to West Nile and Japanese viral encephalitis): edema is often pronounced in TBE and the microglia are often necrotic.

La Crosse Encephalitis

La Crosse encephalitis virus (LCEV) infections decrease with the age of the host; the younger the person, the more susceptible they are to infection, and the more likely the disease is to be severe. Those under age 8 are the most affected.

There are about 300,000 new LCEV infections each year in the United States. As with most encephalitis infections, many cases are asymptomatic or present as a mild to moderate flu that passes without serious effects. In severe cases, the same kinds of inflammatory lesions that are seen in TBE are found, either diffuse or as microglial nodules. The cerebral cortex is most adversely affected, including the frontal, parietal, and temporal lobes. Inflammatory foci are also present in the pons and basal ganglia. Unlike in TBE the spinal cord is rarely affected, but if the infection travels down the spinal cord from the brain it will cause perivascular cuffs of lymphocytes and nerve degeneration.

There has been, oddly, little study of the cytokine profile of the disease. Though what has been done shows that it is similar to most viral encephalitis infections. LCEV stimulates the production of IL-1ß, TNF-α, and IL-6. Bcl-2 is downregulated as well.

Symptoms during severe infection are meningoencephalitis, high fever, seizures, vomiting, photophobia, stiff neck, confusion, paresis, aphasia, chorea, dysarthria, ataxia, and coma. Fever, headache, behavioral changes, and vomiting are the most common symptoms. Those who recover often show lower IQ scores and a high proportion show ADHD behaviors. Some continue to experience seizures.

The virus is spread by mosquitoes. Once in the body the virus begins replicating in the muscle tissue and spreads to the blood, lymph, and spleen and thence to the brain, where it begins to infect neurons, its preferred replication site. It, like the other encephalitides, is present in high quantities in both the spleen and lymph. There are sometimes areas of necrosis in the spleen. Unlike other such viruses, it also infects the respiratory passages.

Although there is no accepted pharmaceutical treatment, ribavirin and recombinant glycoproteins have been found to help. Corticosteroids are sometimes used to reduce the inflammation.

The infection is considered to most closely resemble herpes simplex encephalitis, with which it is often confused. However, I think the infection dynamics are very similar to those of Japanese encephalitis and natural treatment should follow that protocol with one addition: Endothelial swelling can commonly occur with LCEV; Japanese knotweed (or an equivalent) should be added to the protocol to help alleviate it.

Natural Treatment for Encephalitis

This treatment protocol is specific for all viral encephalitis infections. Some adjustment is needed for West Nile, TBE, and dengue, and those adjustments are detailed at the end of the protocol.

If seizures occur, Chinese skullcap and lion's mane should be used, in large doses. If cerebral edema, ischemia, or hypoxia are present, *Ligusticum wallichii* (or the extract ligustrazine) is specific. Rhodiola is also specific for hypoxia.

Note: Chondroitin sulfate has been found to enhance the infection of brain cells by JEV and the damage it causes in the brain. This supplement should be avoided during active infection.

The treatment protocol is followed by a brief look at some of the more important herbs that are neuroprotective and neuroregenerative: Chinese senega root, Japanese knotweed root, kudzu root, lion's mane, and *Crinum latifolium*.

The Protocol Overview

Treatment approaches that can reduce the impacts of the encephalitis infections consist of the following steps:

1. Antiviral systemics
2. Stimulation of the spleen and lymph system to actively attack the infection, thus reducing movement into the brain by the virus and promoting the induction of splenic T cell activation
3. Reduction of cytokine/chemokine cascades through the use of specific inhibitors

4. Protection of neural cells and neural mitochondria
5. Inhibition of viral infection of neural cells
6. Regeneration of damaged neural structures
7. Enhancement of healthy immune system responses through the use of adaptogenic agents
8. Inhibition of HMGB1 if sepsis appears likely

Protocol Specifics

The protocol contains elements to address all these aspects of the infection.

1. ANTIVIRAL SYSTEMICS

Specific antivirals for encephalitis viruses are licorice, Chinese skullcap, isatis, houttuynia. I personally also use lomatium for West Nile and consider it specific.

Formulation: Equal parts of the tinctures of each herb, combined. Dosage: Depending on the severity of condition, from ¼ teaspoon–1 tablespoon of the combination tincture up to 6x daily. My normal use for mild to moderate West Nile, for example, is 1 teaspoon 4–6x daily for 2–4 weeks. Note: The dosage needs to be moderated for children, depending on their weight.

2. STIMULATION OF THE SPLEEN AND LYMPH SYSTEM

Red root (or poke if you can't find red root) will stimulate spleen function, reduce inflammation in the spleen, and enhance T cell counts.

Formulation: Red root tincture. Dosage: ¼–1 teaspoon up to 6x daily depending on the severity of the condition.

3. REDUCTION OF CYTOKINE CASCADES

The herbs specific for this are Chinese skullcap, licorice, isatis, houttuynia, cordyceps, rhodiola, astragalus. They're all included in antiviral and immune formulations (steps 1 and 7, respectively).

4. PROTECTION OF NEURAL CELLS AND NEURAL MITOCHONDRIA

The main herbs for this are Chinese skullcap, cordyceps, Japanese knotweed, rhodiola, kudzu, olive oil (oleuropein). Most of these are included in the antiviral and immune formulations (steps 1 and 7, respectively).

Of the other herbs just named, the most important is kudzu; dosage: ¼–½ teaspoon of the tincture 3–6x daily.

5. INHIBITION OF VIRAL INFECTION OF NEURAL CELLS

The main herbs for this are cordyceps, licorice, Chinese skullcap. They're included in the antiviral and immune formulations (steps 1 and 7, respectively).

6. REGENERATION OF DAMAGED NEURAL STRUCTURES

The best herbs are Chinese senega root (*Polygala tenuifolia*) and lion's mane (*Hericium erinaceus*). Both contain substantial amounts of nerve growth factor.

- **Chinese senega root:** 30 drops of the tincture 3x daily for 30 days, and . . .
- **Lion's mane:** 3–8 grams per day or 1 teaspoon of the tincture 2x daily.

7. IMMUNE HERBS/ADAPTOGENS

The best for this are astragalus, cordyceps, rhodiola.

Formulation: Tincture combination of all three herbs, with 1 part astragalus, 1 part rhodiola, and 2 parts cordyceps. Dosage: 1 teaspoon of the combination tincture 6x daily during active infections.

Note: Cordyceps is very specific for the kinds of neuroinflammation that occur in the brain during encephalitis infections. In severe cases the dosage of cordyceps can be substantially increased. There is no toxic upper range to the herb.

8. INHIBITION OF HMGB1

The best herbs for this are licorice, *Angelica sinensis*, *Salvia miltiorrhiza*.

Formulation: Equal parts of the tinctures of *Angelica sinensis* and *Salvia miltiorrhiza*, combined. Dosage: 1 teaspoon–1 tablespoon of the combination tincture up to 10x daily (or more) depending on the degree of inflammation occurring.

Strong infusions of these herbs will also work just fine. Formulation: 8 ounces of herb per gallon of water; pour boiling water over the herb, cover, and let stand for 4 hours. Strain and use. Dosage: An entire gallon should be consumed daily as the primary liquid source.

Note: There is no upper dosage range for these herbs. The inhibition is dose dependent.

For West Nile Encephalitis

Add the following to the basic protocol described above:

- **Astragalus:** Increase the dosage of astragalus by taking 3,000 mg daily or ½ teaspoon of the tincture 3–6x daily.
- **Japanese knotweed (*Polygonum cuspidatum*):** Tincture, ½ teaspoon 3–6x daily (or resveratrol, from knotweed root, 4,000 mg daily).
- ***Crinum latifolium* leaf:** Tea, 4–6 cups daily, or, if you wish, capsules, 8,000 mg daily. (Aqueous extracts are best.)

For Tick-Borne Encephalitis

Add the following to the basic protocol described above:

- **Greater celandine (*Chelidonium majus*) tincture:** ¼ teaspoon 3x daily (not to exceed 30 days).
- **Motherwort (*Leonurus cardiaca*) tincture:** ¼–½ teaspoon up to 6x daily.
- ***Ligusticum wallichii* tincture (for edema):** ¼–½ teaspoon up to 6x daily, or a strong infusion 6x daily. (This herb will also reduce many of the cytokines TBE induces; the related herbs *Ligusticum porteri* and *Lomatium dissectum* may possibly be legitimate substitutes.)
- **Reduce blood acidity:** Reduce purine intake in the diet, explore acid/alkaline diet modification, supplement with beta-alanine (750–2,250 mg daily).

For Dengue Encephalitis

To the basic protocol described above, add velvet leaf (*Cissampelos pareira*), which is active against all four serotypes of the virus (see the discussion of dengue on page 167 for more). Sea buckthorn leaf (*Hippophae rhamnoides*), boneset, and cat's claw (*Uncaria tomentosa*) are all antivirals specific for dengue as well. Cat's claw is also a decent immune-enhancing herb. The specifics:

- **Velvet leaf tea:** A strong decoction of velvet leaf. Take 2 ounces 4x daily.
- **Tea from sea buckthorn and boneset leaves:** Add 1 ounce each of sea buckthorn leaf and boneset leaf to a quart jar (that can take heat), add boiling water, cover, and let steep 1 hour. Drink throughout the day, every day, for 2 weeks.
- **Sea buckthorn oil:** ½–1 teaspoon 3x daily.
- **Cat's claw:** Tincture, 1 teaspoon 3–6x daily, or capsules, 1,200 mg 3–6x daily, for 2 weeks.

For Some Other, Rarer Encephalitis Infections

Aqueous extracts of *Achyrocline satureioides* are specific for western equine encephalitis virus. *Nyctanthese arbor-tristis* is specific for Semliki Forest virus and encephalomyocarditis virus.

A Brief Look at Some Neural Herbs for Encephalitis Treatment

There are a few herbs that are very specific for protecting or stimulating the regrowth of the neural structures of the brain. They are Chinese senega root, Japanese knotweed, kudzu root, lion's mane, and pink-striped trumpet lily.

Chinese Senega Root (*Polygala tenuifolia*)

This is one of the 50 fundamental herbs of Chinese medicine and is known there as *yuan zhi*. It is used in China primarily as an expectorant and to help lung inflammations. But it also has strong impacts on cognitive function, enhancing memory, alleviating neurotoxicity, and producing positive impacts in the treatment of Alzheimer's, dementia, depression, and degenerative diseases. It is a very good anti-inflammatory and inhibits NF-κB, nitric oxide, iNOS, COX-2, PGE2, TNF-α, and IL-1ß in microglial cells. One of the more important activities of the herb is that it enhances the secretion of nerve growth factor (NGF) in the brain, spinal cord, and peripheral nervous systems. NGF is a small protein that is crucial for the growth, maintenance, and survival of neurons. When neuronal damage occurs, higher levels of NGF are

essential in stimulating axonal regeneration. This is perhaps the best plant for stimulation of NGF.

PREPARATION AND USE

Take 30 drops of the tincture 3x daily for 30 days. It is contraindicated in those with ulcers or gastritis.

Japanese Knotweed (*Polygonum cuspidatum*)

Japanese knotweed is a world-class invasive originally native to Japan, north China, Taiwan, and Korea that is specific for emerging infections. The root is the part of the plant used for medicine.

The herb has a wide range of actions; among other things, it is an antiviral, antimicrobial (widely), antifungal, immunomodulant, anti-inflammatory, angiogenesis modulator, central nervous system relaxant, central nervous system (brain and spinal cord) protectant and anti-inflammatory, antioxidant, inhibitor of platelet aggregation, inhibitor of eicosanoid synthesis, antithrombotic, tyrosine kinase inhibitor, anti-pyretic, cardioprotective, analgesic, hemostatic, and astringent.

A broadly systemic plant, Japanese knotweed modulates immune function, reducing and balancing cytokine cascades; is anti-inflammatory for arthritic, viral, and bacterial inflammations; protects the body against endotoxin damage; and is a potently strong angio-genesis modulator, highly protective of the endothelia of the body. It is specifically inhibitive of endothelial hyperplasia. It crosses the blood-brain barrier and is potently anti-inflammatory in the brain and CNS. It is highly specific for all CNS infections.

Knotweed is a very strong inhibitor of cytokine cascades initiated by pathogens, especially MMP-1, MMP-3, and MMP-9. These can be stimulated through various pathways but knotweed is highly specific for reducing MMPs *and* mitogen-activated protein kinases (MAPKs), specifically c-Jun N-terminal kinase (JNK), p38 mitogen-activated protein kinase (p38 MAPK), and extracellular-signal-regulated kinase 1 and 2 (ERK-1 and ERK-2). Resveratrol (one of the plant's constituents) is also directly active in reducing MMP-9 levels through both the JNK and protein kinase C-delta pathways; it has been found to specifically inhibit MMP-9 gene transcription. The plant root is so high in resvera-trols that it is the main source of the supplement throughout the world.

Another component of the plant, rhein, inhibits the JNK pathway for MMP-1, MMP-3, and MMP-9 expression. The plant is also high in emodin, a constituent that has been found specifically active against a range of encephalitis viruses. Knotweed is a formidable inhibitor of NF-κB, TNF-α, ICAM-1, VCAM-1, IL-1ß, IL-6, COX-2, IL-8, PI3K (phosphoinositide 3-kinase), E-selectin, IDO, and VEGF (vascular endothelial growth factor).

Polygonum cuspidatum's constituents cross the blood-brain barrier, where they act on the central nervous system as antimicrobials, anti-inflammatories, protectants against oxidative and microbial damage, and calming agents. The herb specifically protects the brain from inflammatory damage, microbial endotoxins, and bacterial infections. The compounds easily move across the gastrointestinal mucosa and circulate in the bloodstream.

The whole herb and its constituent, resveratrol, are both strong antioxidants. People who consume the herb (or constituent) have been found to have significantly increased antioxidant activity in their blood. This potent antioxidant action has been detected throughout the body. Interestingly, resveratrol seems to be an antioxidant modulator in that it will increase antioxidant action when needed (most of the time) but will lower it in instances where necessary, e.g., in leukemia cells.

Another component of knotweed, emodin, is highly protective of brain neurons and reduces pain by inhibiting the activation of the P2X7 receptors in the brain. It reduces impacts on the P2X2/3 receptors as well.

The herb, in fact, is a potent immunomodulator. It normalizes immune response, especially in diseases where autoimmune reactions are stimulated (such as Lyme disease and lupus). It seems able to bring up immune function when necessary and reduce its local manifestations when overstimulated, e.g., in rheumatoid arthritis. This is a very strong aspect of the plant's actions.

The herb and its constituent have also been found to enhance and potentiate the action of other drugs and herbs when taken with them.

Japanese knotweed and its constituents possess strong actions in the central nervous system and brain, and this range of activity is where a great deal of interest in the plant is being generated.

Knotweed and the constituents trans-resveratrol and resveratrol have been found to be strongly neuroprotective through a variety of actions in numerous studies. One of the herb's mechanisms of action

in this regard is as an antioxidant. Resveratrol and trans-resveratrol have been found to protect rat embryonic mesencephalic cells from a powerful pro-oxidant, tert-butyl hydroperoxide. Another study found that regular use of trans-resveratrol prevented streptozotocin-induced cognitive impairment and oxidative stress in rats (an Alzheimer's-like condition). And yet another found that trans-resveratrol protected and reversed many of the impacts of induced stroke in rats.

While the herb's antioxidant actions are important, transresveratrol, for example, has been found in a number of studies to strongly protect neuronal structures from damage through mechanisms other than antioxidant activity alone. Resveratrol has been found specific for protecting the brain from neurotoxic substances such as the beta-amyloid peptides, which are associated with Alzheimer's disease.

Resveratrol and trans-resveratrol are specific for reducing inflammation in the brain and central nervous system. In spinal cord injuries resveratrol "remarkably" reduced secondary spinal cord edema, significantly suppressed the activity of lactate dehydrogenase, reduced malondialdehyde content in the injured spinal cord tissue, and markedly improved NA+/K+-ATPase activities. It immediately stimulated microcirculation to the injured tissues.

Low-level, chronic inflammation in the brain and central nervous system plays a major role in many neurodegenerative conditions. Both the herb and its constituents are specific for such inflammations. They have been found active for such things as amyotrophic lateral sclerosis and other motor neuron diseases, Parkinson's disease, Alzheimer's disease, bulbar atrophy, dementia, Huntington's disease, myasthenia gravis, stroke, multiple sclerosis, frontotemporal dementia, encephalomyelitis, traumatic brain injury, cerebral ischemia, and so on. The resveratrols specifically protect brain cells from assault, whether chemical or microbial in origin. The herb and its constituents, as well, stimulate microcirculation in the brain.

PREPARATION AND USE

Capsules or tincture can be used. Capsules of pure knotweed root can be had from Green Dragon Botanicals (https://greendragonbotanicals.com) or you can buy "resveratrol," which is in fact only a standardized knotweed root formulation. That is, it is knotweed root standardized for the

presence of a certain percentage of resveratrols. Most of the brands on the market are usable. Just make sure they are made from knotweed root (it will say on the package someplace in tiny print) and not grapes. As for dosage:

- **Capsules:** 3 or 4 capsules 3 or 4x daily (or 1 tablespoon of the powdered root in juice 3 or 4x daily).
- **Tincture:** ¼–½ teaspoon 3–6x daily.

The major side effect, in about 0.5 percent of those who use it, is loss of taste. This corrects upon discontinuance of the herb (in a week or so). Metallic or odd taste is moderately common. GI tract disturbances are rare but do occur. Note: Some people have reported rather strong negative physical responses to the Source Naturals resveratrol. I am not sure why this is; however, those who do, having switched to another product by Paradise Herbs, report a better outcome. Just an FYI on that one.

Japanese knotweed should not be used with blood-thinning agents. Discontinue use of the herb 10 days prior to any surgery. The plant is a synergist and may potentiate the effects of pharmaceuticals and other herbs.

Kudzu Root (*Pueraria lobata*)

Kudzu root and its major constituents (puerarin and kakkalide and irisolidone—a metabolite of kakkalide produced by intestinal microflora that is more potent than kakkalide) inhibit TNF-α, IL-1ß, NF-κB, ERK, iNOS, PGE2, COX-2, AP-1 (activator protein-1), ICAM-1, VCAM-1, E-selectin, C-reactive protein (CRP), and the phosphorylation of the 1κB-α protein. It has targeted and potent effects on cytokine-activated microglial cells and will reduce damage by cytokines in the CNS.

Kudzu (and puerarin) are strongly protective of the brain and CNS, especially in ischemia/reperfusion injury. They have a strong protective effect against beta-amyloid-induced neurotoxicity in hippocampal neurons, protect mitochondria from reactive oxygen species (ROS), and stimulate peripheral nerve regeneration. Neuron pain receptors P2X3 and P2X²⁄₃, similar to P2X7, in the brain are inhibited by puerarin, making this a very good companion herb to use with greater celandine. The root is, in fact, strongly anti-inflammatory in the brain and CNS. It significantly inhibits neutrophil respiratory bursts, reducing auto-

immune dynamics in the brain. It modulates Bax/Bcl-2 actions in the mitochondria in the brain and inhibits caspase-3 and iNOS expressions. It is strongly neuroprotective during inflammation disturbances in the brain and CNS.

It is a good herb for viral encephalitis types of cytokine cascades, especially so since it is invasive and there is no dearth of supply. *Anywhere* encephalitis occurs and kudzu grows, use kudzu.

PREPARATION AND USE

In traditional Chinese medicine the usual dose is 6–12 grams per day of the powdered root. Normally dosing in the West is 1 gram per day. The tincture dosage in encephalitis infection is ½ teaspoon 3 or 4x daily.

Lion's Mane (*Hericium erinaceus*)

Lion's mane is a highly edible mushroom, a member of the tooth fungus group of mushrooms. (It should look sort of like a waterfall of beautiful white threads. If it does not, be suspicious.) The mushroom has a long history of use in Asia. Like most mushrooms, it is high in polysaccharides and possesses strong immunomodulating and immune-activating properties. It strongly activates T cells and strongly downregulates iNOS, nitric oxide, ROS, PGE2, COX-2, NF-κB, and JNK during inflammatory overreactions.

However, its strongest actions are in its effects on nerve cells and brain function. The herb stimulates peripheral and CNS nerve regeneration after damage (specifically by inducing nerve growth factor in the body), increases remyelination of nerve sheaths, improves cognitive impairment after neural damage (including amyloid peptide-induced), and significantly alleviates depression and anxiety.

PREPARATION AND USE

The herb can be used as either an aqueous or alcohol preparation; I tend to prefer the alcohol tincture made from the fresh mushroom. Dosage for encephalitis is ¼–½ teaspoon 3–6x daily, depending on the severity of the condition. I think the herb highly indicated to alleviate or in the treatment of postencephalitis conditions.

Pink-Striped Trumpet Lily (*Crinum latifolium*)

Pink-striped trumpet lily is a typical-looking lily, a perennial that grows from a bulb. The herb is commonly used in Asia as a medicinal for inflammatory problems of various sorts, especially rheumatoid complaints. It is most commonly used in the West now to reduce prostate enlargement, either benign prostatic hyperplasia or prostatitis.

The herb is a potent inhibitor, perhaps the best currently known, of indoleamine 2,3-dioxygenase (IDO). In consequence it is a potent stimulant of T cell activation in cases where IDO has been overexpressed by pathogens. It is strongly inhibitive of IFN-γ, stopping the particular cytokine cascade that it starts during infections. It also inhibits NF-κB. The plant is a pretty good free radical scavenger, reducing ROS in the body and CNS. The herb is antiangiogenic, protecting the endothelial cells from inflammation, hyperplasia, and overstimulation by cytokines. It has been found to possess antiviral activity against some vaccinia viruses. And it is active, in vitro and in vivo, against a variety of cancers, including prostate.

PREPARATION AND USE

The leaf, prepared as a tea (aqueous extracts are best), 4–6 cups daily, or, if you wish, in capsules, 8,000 mg daily. The bulbs are easy to find and the herb grows easily. You can find the tea most easily on eBay.

5

A BRIEF LOOK AT SOME OTHER VIRUSES

From Cytomegalovirus and Dengue to Shingles and Their Treatment Protocols

The underlying causes for the emergence of infectious diseases are anthropogenic social and environmental changes. These result from the combined weight of human numbers and their consumption patterns that are overloading the planet's biophysical and ecological capacity. . . . If the human impact on the ecosphere continues to escalate, the rate of emergence of infectious diseases will only increase in the future.

—Thijs Kuiken et al., "Emerging Viral Infections in a Rapidly Changing World"

There is currently a large and ever-expanding global population base that prefers the use of natural products in treating and preventing medical problems.

—R. Hafidh et al., "Asia Is the Mine of Natural Antiviral Products for Public Health"

There are many other viruses that cause human illness that either are becoming resistant or are emerging more strongly into the world's population of people. In this section I will look at cytomegalovirus, dengue virus, enterovirus 71, Epstein-Barr virus, herpes simplex viruses, varicella zoster (shingles), and a few gastrointestinal viruses such as

rotavirus and norovirus. I am not going to explore the Coxsackie or ECHO viruses in this book at this time, nor am I going to look at the five hepatitis viruses or HIV. Viral hepatitis and HIV need, for the most part, highly involved treatment protocols that are beyond the scope of this book; they each need in-depth books of their own.

Cytomegalovirus

Cytomegalovirus (CMV) is a member of the Herpesviridae family of viruses, a very large group of DNA viruses that also includes the herpes simplex viruses, Epstein-Barr virus, and varicella zoster virus (chicken pox/shingles). It is sometimes referred to as human herpesvirus 5 (HHV-5). It usually causes minor problems in people with healthy immune systems but if immune function drops (especially in those with AIDS, those taking immunosuppressive drugs, or those whose immune function is simply becoming weak from either age or illness) or if the immune system has not yet developed fully (babies, in and out of utero) it can cause severe problems up to and including death.

Infection rates are high. In the United States somewhere between 50 and 80 percent of the adult population are asymptomatically infected. When people first become infected the usual signs are fever, fatigue, muscle tenderness, and tender and enlarged lymph nodes. Just like the flu. Then it passes, you feel fine, and you are an asymptomatic carrier.

As the years go by the virus periodically blooms, sending viruses to body fluids: saliva, tears, urine, blood, semen, vaginal secretions, and breast milk. From there it spreads to new hosts. It is a pretty good parasitic virus, and it usually doesn't cause much trouble. However, in people whose immune function is impaired, the virus can cause a wide range of symptoms: encephalitis, myelitis, seizures, coma, psychosis, dysphagia, weakness, numbness in the legs, retinitis, visual impairments including blurred vision or blindness, pneumonia, gastritis, enteritis, colitis, diarrhea, ulcers in the GI tract, and hepatitis.

In babies, CMV is the most common congenital infection in the United States. For most it will not be a problem but for some it can cause severe and long-term difficulties: neurological problems, hearing impairment or loss, visual problems, seizures, or mental and physical disabilities. The worst problems occur in infants whose mothers become infected while the infants are in utero.

CMV is normally treated with the intravenous antibiotic ganciclovir (or valganciclovir), usually along with CMV-specific immune globulin. Treatment usually lasts 2 to 4 weeks, followed by a transition to oral valganciclovir. Unfortunately the virus is becoming fairly resistant to ganciclovir. Generally, this drug will not cure the infection but only lessen its effects. Again, the search is for a vaccine; however, early trials show that the vaccines in development only tend to lessen the effects of the disease, not eliminate it, for the majority of people using them.

Dengue Fever

Dengue viruses (DENVs) are single-stranded RNA viruses in the Flaviviridae family, specifically in the *Flavivirus* genus. This genus includes many of the encephalitis viruses such as West Nile, tick-borne encephalitis, Japanese encephalitis, Murray Valley encephalitis, St. Louis encephalitis, and so on. Yellow fever virus is also in this group. There are various serotypes of dengue (1, 2, 3, and 4). Each one produces a slightly different spectrum of symptoms. DENV-3 produces more musculoskeletal and gastrointestinal symptoms, DENV-4 has more respiratory and cutaneous symptoms.

Dengue fever, a.k.a. breakbone fever, is a mosquito-transmitted disease and is not infectious except through that route. There are about 100 million people infected with dengue each year; several hundred thousand develop dengue hemorrhagic fever, which is very serious, and about 22,000 die.

The disease is endemic pretty much everywhere, including the southern United States. Outbreaks have occurred in recent years throughout the Caribbean including Puerto Rico, the U.S. Virgin Islands, and Cuba. The disease is common in Tahiti, Singapore, the South Pacific, Southeast Asia, the West Indies, India, and the Middle East as well as throughout South America. The incidence of the disease is growing each year.

Once a mosquito bites a person, the virus enters the body and begins seeking out monocytes and macrophages, the primary parts of the body it infects. The symptoms develop in 3 to 15 days (normally 5 to 8). The symptoms are sudden-onset fever, headache, pain on eye movement, and low backache. Muscle and joint pain (which is often very severe—

it's called "breakbone" fever for a reason) occurs soon after, a fever, often as high as 104°F (40°C), develops, heart rate and blood pressure drop. The eyes redden, a rash may occur on the face, lymph nodes often swell.

The fever tends to last several days, then a sudden drop in body temperature occurs accompanied by extreme sweating. This passes, you feel fine, then it starts all over again. This time the rash covers the whole body except for the face.

Dengue hemorrhagic fever (DHF) usually affects children under 10 years of age. It causes severe abdominal pain, hemorrhage, and shock. Sore throat, cough, nausea, vomiting all occur. Somewhere from 2 to 6 days after symptoms begin there is sudden collapse, with cool, clammy extremities, weak pulse, and cyanosis around the mouth. The skin bruises easily; there is bleeding, spitting up of blood, blood in the stool, bleeding gums, nosebleeds. Pneumonia is common; inflammation of the heart may occur. The disease is associated with high levels of circulating von Willebrand factor. Normal treatment for DHF is transfusions, oxygen, and fluid replacement.

There are no pharmaceutical treatments for either form of dengue fever. There is no vaccine. However, recent research has shown that the antibiotic Geneticin is active against the virus. Its effectiveness in practice is unknown.

Chikungunya and Zika

In less than 10 years, CHIKV has spread from the coast of Kenya throughout the Indian Ocean, Pacific, and Caribbean regions, causing millions of cases of disease in over 50 countries. In other words, CHIKV has reemerged as a true global pathogen.

—Thomas E. Morrison, "Reemergence of Chikungunya Virus"

With respect to treatment, the arbovirus pandemics suggest that the one-bug–one-drug approach is inadequate; broad-spectrum antiviral drugs effective against whole classes of viruses are urgently needed.

—Anthony S. Fauci and David M. Morens,
"Zika Virus in the Americas—Yet Another Arborvirus Threat"

In 2012, just as I began the first edition of this book, I came across reports of a virus I had not heard of before. The stories were compelling; they offered a perfect example of an emerging, and unexpected, viral infection moving through the world's communities. So I used that

virus as a metaphor for how previously unknown viral infections can suddenly emerge and devastate communities. Little did I know that the chikungunya virus (CHIKV) would soon present itself as a permanent and medically difficult-to-treat problem throughout the Caribbean as well as in parts of South and North America. (These infections are now becoming endemic in Florida and along the American Gulf Coast.) Shortly thereafter, in 2013–2014, a powerful ebola outbreak occurred in Africa and scores of infected air travelers spread the infection throughout the world. But this was not the last of it; in 2015 a massive Zika virus outbreak occurred in South America. The outbreak was larger than any previously known and a little-known side effect of the infection emerged with it: brain damage in the unborn children of pregnant women. This is the shape of things to come. It is the new normal.

Although we've just finished looking at dengue (DENV), I also want to mention it here because dengue, Zika, and chikunguna viruses have very similar infection and symptom patterns. In fact, Zika and CHIKV infections are often misdiagnosed as DENV. DENV and Zika are both flaviviruses and are closely related to some of the other viruses discussed in this book such as tick-borne encephalitis virus, Japanese encephalitis virus, Murray valley encephalitis virus, St. Louis encephalitis virus, and the West Nile virus. CHIKV is an alphavirus and is more closely related to a different group of viruses, also discussed in this book, specifically the eastern, western, and Venezuelan equine encephalitis viruses. DENV, Zika, and CHIKV are all spread by mosquitoes, all are primate-mosquito-primate transmitted viruses (multiple species in the *Aedes* genus are the primary transmitters), and all cause similar symptoms. As Roth et al. (2014) comment:

> Manifest dengue, chikungunya, and Zika virus infections have a similar initial clinical presentation and may be reported as any of the first three of the following four monitored syndromes: (i) acute fever and rash, (ii) prolonged fever, (iii) influenza-like illness and, (iv) diarrhoea.

The rash (maculopapular) looks a bit like chicken pox and may cover the body. Most commonly, all three are accompanied by severe joint, bone, or muscle pain. There may be severe headache and eye pain as well. Under certain circumstances neurological damage, of varying severity, can occur as well.

These viruses are responding to planetary ecological disruption and expanding their geographical range at a rapid rate. (The same factors are stimulating their mosquito vectors to expand similarly.) In December 2013, for example, CHIKV was isolated for the first time in the Western hemisphere. As of February 2016, it had spread to 42 countries or territories in the Caribbean, Central, South, and North America. All four strains of the dengue virus are now widespread in the Americas and outbreaks are commonly occurring in southern Europe (as well as France and Croatia). Zika was first detected in May of 2015 in Brazil; it then spread rapidly throughout 26 other countries and territories in the Americas. By early 2016 over a million cases of infection had been reported motivating the World Health Organization to declare it a "Global Emergency." Outbreaks of each of these viral infections are commonly preceded by drought conditions. There is some evidence that the viruses are synergistic with each other in that the appearance of one seems to prepare the way for the emergence of another. As Fauci and Morens (2016) comment, "Decades ago, African researchers noted that aedes-transmitted Zika epizootics inexplicably tended to follow aedes-transmitted chikungunya epizootics and epidemics." Dengue aggressively entered the Western hemisphere in the 1990s (there are some 50 million infections worldwide every year), West Nile virus in 1999, CHIKV in 2013, and Zika in 2015. As Fauci and Morens go on to say, "As was realized more than 50 years ago, when enzootic Zika virus was linked to human activity, arboviruses continually evolve and adapt with ecological niches that are increasingly being perturbed by humans." All these viruses seem to be undergoing genetic alterations that are speculated to be root to the increased virulence of the organisms.

Dengue, chikungunya, and Zika are all single-stranded, positive-sense RNA viruses. The most research has occurred with dengue, next with chikungunya, and only a smattering (as of spring 2016) with Zika. Rather than somewhat rare periodic outbreaks, all three viruses are becoming established as endemic emerging viral pathogens. Humans are considered to be primary reservoirs for all three viruses. Concurrent infection with all three viruses (and other similar viruses such as West Nile and tick-borne encephalitis virus) does occur. There are no technological medicines that are effective in their treatment. There is significant crossover in treating these infections with herbal medicines.

Enterovirus 71

Enterovirus 71 (EV-71, sometimes called hand, foot, and mouth disease) is a single-stranded, nonenveloped RNA virus in the Picornaviridae family. Rhinoviruses (common cold), hepatitis A, and the poliovirus are some of the better-known members of the family.

Enterovirus 71 was first identified in 1965; the first known outbreak was in 1969 in the United States. There are severe outbreaks every 3 to 4 years throughout the world with smaller annual episodes here and there. A huge outbreak in Taiwan in 1998 infected over 130,000 people, mostly children. The virus is usually spread by respiratory droplets or contact with an infected person's body fluids. It is excreted in feces for weeks or months after infection.

The virus initially colonizes the GI tract and from there, if the disease does increase in severity, it invades the spinal cord and ascends to the brain. In the CNS and brain it produces high levels of cytokines: IL-1ß, IL-6, IL-8, and TNF-α. Enterovirus 71 is considered to be an emerging pathogen, with the number of cases increasing each year.

The most common symptoms of infection are fever, headache, fatigue, malaise, ear pain, sore throat, body rash, blisters on the palms of the hands or soles of the feet (they may also occur in the nostrils and buttocks), oral ulcers, loss of appetite, diarrhea, vomiting. Fever and sore throat are usually the first signs.

Unfortunately, there are some more serious complications that can occur during infection: high fever, meningitis, encephalitis, paralysis, cardiopulmonary edema, and sometimes coma and death.

There is no pharmaceutical treatment or vaccine.

A Brief Look at Enterovirus D68

Enterovirus D68 (EV-D68) is an emerging virus that is relatively new to Western medicine; little technological treatment is available or effective for it. In 2014 a major outbreak occurred in the United States. EV-D68 is considered to be a sort of combination of a rhinovirus and an enterovirus. Unlike EV-71 it is primarily a respiratory infection. Its impacts can range from no symptoms at all to a mild flu-like infection to bronchitis to pneumonia and more seriously to meningitis, encephalitis,

paralysis (similar to polio), and death. The virus is highly infective for young children, much less so for adults. Treatment with herbs can be highly effective. Note: Dosages must be adjusted for child age and weight; the dosages in this book are for an adult weighing approximately 150 pounds. To find the proper child dosage, divide the child's weight by 150. So, if the child weighs 50 pounds, the proper dose will be one-third that of the dosages listed on page 184.

Epstein-Barr

Epstein-Barr is a member of the Herpesviridae or herpesvirus family; it's sometimes referred to as human herpesvirus 4 (HHV-4). It is the cause of mononucleosis and a very common cause of chronic fatigue syndrome. It is considered an oncovirus, meaning it can, under some circumstances, over long periods of time, cause certain forms of cancer. Most children become infected with it at a young age, in which case the symptoms are usually mild—it looks like a mild case of the flu. The older the person at infection, the more severe the symptoms. In teenagers it causes infectious mononucleosis. This is usually accompanied by a severe sore throat, fever, and extreme fatigue and malaise. The infected may also experience swollen throat, loss of appetite, enlarged lymph nodes, swollen spleen and liver, jaundice, petechiae. Some of the very serious complications are splenic rupture or hemorrhage, meningitis, peripheral neuritis, and pneumonitis. An autoimmune hemolytic anemia can occur as well, though it is much rarer.

There are no reliable pharmaceutical treatments for the disease. Acyclovir can help reduce viral shedding. Valacyclovir has been found to, sometimes, significantly reduce the viral load and thus the symptoms of the disease.

Herpes Simplex 1 and 2

Herpes is probably the most famous (or infamous) member of the Herpesviridae family of viruses. There are two types, herpes simplex virus 1 (HSV-1) and herpes simplex virus 2 (HSV-2). The most common sites of infection are around the lips (cold sores, fever blisters) and on

the genitals (herpes). Cold sores are usually caused by herpes simplex 1, genital herpes by herpes simplex 2. A less common form is infection of the fingers, called herpetic whitlow. It is caused by either type. There are some more serious forms of infection: ocular herpes (which infects the eyes, causing a form of keratitis), herpes gladiatorum (which broadly infects the skin), herpes encephalitis (which infects the brain), Mollaret's meningitis (which infects the protective membranes covering the brain and spinal cord), and neonatal herpes (where the baby is infected as it is born or in utero).

Herpes, either oral or genital, is usually cyclic with long (or short) periods of remission between episodes. With genital herpes, once the virus enters a new host it travels along the nerve paths, where it takes up residence. For many people, after an initial outbreak, the virus may become dormant indefinitely. For others, every so often, the virus becomes active. It then travels back along the nerve paths to the surface of the skin, where it creates blisters that shed viruses, spreading the disease.

Just prior to a new episode, there may be a feeling of pain in the nerves in the genitals. Soon small blisters may appear; they eventually break open and produce very painful sores that scab over and heal after several weeks. Sometimes people with an active infection feel as if they have the flu, and the lymph nodes may be swollen.

Oral herpes and herpetic whitlow symptoms are similar but occur around the lips and fingers respectively.

There are generally three types of ocular, or eye, herpes. Herpes keratitis is the most common form; it's a viral infection of the cornea or surface of the eye. This usually heals without damage to the eye. Stromal keratitis is a deeper form, where the virus penetrates deeper into the layers of the cornea. It can cause scarring of the cornea and, sometimes, blindness. The final type is iridocyclitis. Here the iris and surrounding tissues of the eye are infected and become inflamed. There is often severe light sensitivity and blurred vision, and the infected eye is red. Sometimes this kind of infection can occur in the retina or inside lining of the back of the eye; it's then called herpes retinitis.

Signs and symptoms of herpetic eye infections are swelling, tearing, irritation, foreign body sensation, redness, eye sores, discharge, light sensitivity.

The normal treatment for eye herpes is ganciclovir as an ophthalmic gel, 5 drops daily. Sometimes steroid eyedrops are used to decrease the inflammation. The infected cornea is sometimes scraped away. There is no effective cure; these interventions reduce the impact of the episode and, hopefully, prevent permanent damage.

Herpes gladiatorum is tremendously infective and is transmitted by skin-to-skin contact. It is essentially a cutaneous form of herpes, usually herpes simplex 1 (the same form of the virus that causes cold sores). It is somewhat common in sports clubs (which is where it got its name). In this condition, the sores, instead of being confined to the genitals or lips, are found in clusters over the body: neck, chest, face, stomach, legs. The lymph nodes are often enlarged, with fever, sore throat, headache. The blisters are very painful and take weeks to heal.

Herpes encephalitis and meningitis are uncommon but severe complications of herpes infection. Herpes meningitis is usually caused by the HSV-2 serotype and is an inflammation of the meninges, the tissue surrounding the brain and spinal cord. The normal symptoms are stiff neck, headache, fever, light sensitivity, fatigue, nausea, appetite loss, vomiting (sometimes). It is usually a self-limiting disease and clears on its own. For some people it recurs at periodic intervals just as genital and oral herpes do. In rare instances it may cause minor mental dysfunctions, slight confusion, and difficulty in problem solving that fail to resolve.

Herpes simplex encephalitis is usually caused by HSV-1. During herpes simplex encephalitis the temporal and frontal lobes are usually infected when the virus travels along the nerve lines into the brain. The usual encephalitic processes occur: inflammation of the brain, confusion, psychological alterations, fever, and in anywhere from 30 to 70 percent of those infected seizures, coma, and death. The infection is usually treated with IV acyclovir but there is still a 30 percent mortality rate. There is often long-lasting brain damage.

The usual treatment for herpes infections is valacyclovir, acyclovir (a.k.a. aciclovir), or famciclovir. All are forms of guanosine analogue antiviral drugs that, when taken, are metabolized by the body into an active form. These drugs are widely used to help control outbreaks of herpes simplex viruses, chicken pox, shingles, Epstein-Barr, and cytomegalovirus, all of which are closely related herpesviruses. They are strongest against the simplex types and have no effect on the viruses in

their latent form in the nerve sheaths; they are effective only when the viruses become active. The drugs can shorten the course of the disease to some extent and help prevent spread. They do not cure it.

Herpesviruses are becoming resistant fairly rapidly to acyclovir, especially in those with compromised immune systems.

Varicella Zoster Virus (Chicken Pox/Shingles)

Varicella zoster virus is also a member of the Herpesviridae family; it's sometimes referred to as human herpesvirus 3 (HHV-3). HHV-3 usually occurs first as chicken pox (i.e., varicella), later as shingles (herpes zoster).

During the initial infection, normally during childhood, spread via respiratory droplets in the air, the virus enters the bloodstream and travels everywhere, making spots all over the surface of the body. It takes about 2 weeks for the spots to appear and the infection period lasts for up to 3 weeks after that, until the spots crust over. At that point, still spotted, the child is no longer infected. ("Yes, you still have to go to school.")

Though the disease was usually self-limiting and fairly benign vaccines are now commonly used in the West for chicken pox. However . . .

Regrettably, the virus, once the initial infection has passed, settles in the dorsal ganglion, part of the nerve structure of the central nervous system. There it lives quite happily for decades, until in later age, usually as a result of lowering immune function, it sometimes reappears as shingles.

The virus leaves its happy home, travels along the nerves, and emerges at the surface of the skin, often on the face, and makes life intolerable. There is often hemorrhage, edema, and lymphocytic infiltration of the affected nerves. Usually, the person can feel the event beginning—as the virus travels along the nerves it creates odd feelings, tingling, burning, numbness, and a very definite feeling that something is wrong. Then, quite quickly, the virus emerges out of the body onto the surface of the skin, creating tiny blisters filled with virus particles. The pain is often excruciating and little in the pharmaceutical armamentarium can help. Occasionally there can be fever, enlarged lymph nodes, chills,

malaise, loss of appetite, and stomach upset. The lesions can last up to a week or so once they emerge. There can also, rarely, be more difficult complications such as encephalitis, peripheral nerve palsies, hemiparesis, and myelitis.

The most problematic aspect of shingles is postherpetic neuropathy, i.e., pain, at the site of the eruption. It can be an unrelenting sharp, burning, or stabbing pain that can become highly debilitating. About half the people who get shingles also develop postherpetic neuropathy. The neuropathy is usually caused by the infection itself as it blooms at its emergence location. The local nerves are damaged by the inflammation and it takes them time, sometimes a very long time (7 to 10 months), to recover. The older the person, the longer they take to heal. As long as the damage lasts, postherpetic neuropathy will remain.

The usual medical treatment is the use of antivirals such as acyclovir, valacyclovir, or famciclovir. Corticosteroids may also be used to lessen the inflammation, especially if the complications are serious. Over-the-counter analgesics are often used, with varying success. The pain is sometimes so severe that opiates are necessary. Pharmaceuticals don't heal the disease but they can help reduce the length of an episode and help with pain.

One of the better natural approaches for shingles is the use of herbs to increase immune function to reduce outbreak frequency and herbs that protect and restore the nerves. Specific herbs for nerve pain will also help tremendously, especially those that affect the P2X receptors in the brain.

Gastrointestinal Viruses (Rotaviruses and Noroviruses)

There are a number of common (and a few uncommon) viruses that cause gastrointestinal infections in people. The two primary ones are rotavirus and the Norwalk (norovirus) virus. Some countries now use vaccines against rotaviruses; there is none for the Norwalk virus, which is thought to cause 90 percent of all nonbacterial epidemic gastroenteritis worldwide and as much as half of all U.S. gastroenteritis infections. There is also an emerging group of viruses, discovered in the

1970s, the astroviruses, that are GI tract infectives. And the orthoreoviruses that are thought to only rarely cause human disease.

These groups of viruses are specialists in infecting human food plants and bind quite strongly to lettuce leaves, for example. They are the main cause of diarrheal outbreaks from agricultural food products. The symptoms are much the same for all: nausea, vomiting, diarrhea, abdominal pain, lethargy, weakness, muscle aches, headache, low-grade fever, dehydration. The diseases are usually self-limiting but they can be quite debilitating and a few people in the United States always die from them every year. Rotaviruses are still a major cause of child mortality throughout the world.

In essence this is what is usually called the stomach flu in the United States. It is a food-borne illness from virus-contaminated food, usually vegetables, and is commonly spread via salad bars or prepackaged spinach, bean sprouts, and so on.

The Treatment Protocols

Again, the protocols suggested in this section are *only* suggestions. There are many ways to approach treating these viruses; this is just a starting point. Comment: If encephalitis occurs from any of these viruses, the encephalitis protocol, with slight alterations for each virus, which would mean adding specific herbs to the protocol, should be used.

Cytomegalovirus (CMV)

Herbs (and supplements) that have been found active for this virus in vitro, in vivo, and in human clinical use are (in alphabetical order) *Artemisia annua* (or artemisinin), *Astragalus membranaceus* (astragalus), the berberine plants, *Bidens pilosa* (bidens), *Bupleurum kaoi*, *Forsythia suspensa*, *Geum japonicum*, *Glycyrrhiza glabra* (licorice), houttuynia, *Hypericum perforatum*, isatis, *Lomatium dissectum*, *Nigella sativa*, quercetin, *Scaevola spinescens*, *Syzygium aromaticum* (cloves), *Terminalia chebula*, *Urtica dioica* (nettles), *Zingiber officinale* (ginger).

Geum japonicum, licorice, isatis, and the berberines have been found as effective as ganciclovir in the treatment of CMV in vivo.

TREATMENT

To begin: Bidens is a potent systemic antimicrobial herb (it must be prepared from fresh leaves; for more information, see my book *Herbal Antibiotics*, second edition) and I would include it in any protocol for treating CMV. A systemic formulation that would be good is *Bidens pilosa*, houttuynia, isatis, licorice, and lomatium, equal parts of each of the tinctures, combined. Dosage: ¼–½ teaspoon of the tincture combination from 3–6x daily depending on severity of the infection.

Then, based on specific symptoms, add to this systemic formulation any of the following:

For GI tract symptoms: Berberine-containing plants are specific in this instance: goldenseal, phyllanthus, barberry, Oregon grape root, coptis, and so on will all work fine. My preference is for phyllanthus, barberry, or goldenseal. Dosage: ¼–½ teaspoon of the tincture 3–6x daily depending on severity of symptoms. The systemic tincture should also be used, with the same dosage range described above. The licorice will help any ulceration that has occurred.

For neurological symptoms: The encephalitis protocol (see page 154) plus bidens.

For pneumonia: Moderate to severe influenza protocol (see page 40) plus bidens.

Dengue Fever Virus

Some herbs and supplements effective for dengue are (in alphabetical order) *Alternanthera philoxeroides*, *Andrographis paniculata*, *Artemisia douglasiana*, *Azadirachta indica* (neem), the berberine (and palmatine) plants, *Cissampelos pareira*, *Cladogynos orientalis*, *Cryptocarya chartacea*, *Daucus maritimus*, *Distictella elongata*, *Ellipeiopsis cherrevensis*, *Eupatorium patens*, *Eupatorium perfoliatum* (boneset), *Flagellaria indica*, *Garcinia multiflora*, *Gastrodia elata*, *Glycyrrhiza glabra* (licorice), grape seed proanthocyanidins, *Hippophae rhamnoides* (sea buckthorn leaf), houttuynia, *Kaempferia parviflora*, *Lantana grisebachii*, *Momordica charantia*, *Ocimum sanctum* (mildly active), *Punica granatum* (pomegranate juice), *Quercus lusitanica* (gall oak seed), *Rhizophora apiculata*, *Salvia miltiorrhiza* (Chinese or red sage), *Stemona tuberosa*, *Tephrosia* spp., *Uncaria tomentosa*. Oligomeric procyanidins (OPCs) are also specifically antiviral for dengue. They are common in

cranberry juice, pomegranate juice, grape seeds, and unripe apple peels (for example). Any source can help, but that is the reason that pomegranate is included here. In addition pomegranate is a fairly potent synergist with a wide range of antimicrobial activity.

Many of the studies did not mention the serotype of dengue tested. Most of the herbs and supplements that did specify serotype have only been tested against type 2 (out of the four serotypes). The exceptions (that I can find) are andrographis, *Momordica charantia* (type 1), *Ocimum sanctum* (type 1), and *Cissampelos pareira* (all four serotypes). It is possible that many of these herbs are active against other serotypes but no testing has yet been done.

TREATMENT

I would suggest beginning treatment with the following: *Cissampelos pareira*, sea buckthorn leaf, boneset, houttuynia, licorice, pomegranate juice, and cat's claw. *Salvia miltiorrhiza* is for hemorrhagic dengue as an adjunct.

Cissampelos pareira (in English, velvet leaf) is a widely used herb in Chinese medicine, Ayurveda, and South and Central America (where it is called *arbuta*). It is moderately findable, if you look for it. It is somewhat invasive in Florida in the United States and should be harvested for use in that region.

The herb is antipyretic, anti-inflammatory, immunomodulatory, antiplasmodial, antinociceptive, antiarthritic, antioxidant protective, antileukemic, cardioprotective, constipative, abortifacient. It should not be used if you are pregnant or wanting to become pregnant. You can use it either as a strong decoction or as a 50 percent alcohol/water tincture. It is the only herb that is considered to have strong action against all four serotypes of dengue.

Sea buckthorn leaf and boneset are both antivirals specific for dengue. Boneset, additionally, will help lower fever, reduce pains in the body, and stimulate the immune response. Houttuynia and licorice are good supportive antivirals, and both are active against dengue. (Unfortunately, all have only been tested against serotype 2.) Licorice will also act as a synergist and anti-inflammatory. Pomegranate juice constituents are antiviral for dengue and it is a good synergist and anti-inflammatory so it is perfect for keeping up fluids. Cat's claw will protect

the macrophages and monocytes from dengue infection (it is very specific for this). Chinese sage should be used in cases of hemorrhagic dengue; it will lower circulating levels of von Willebrand factor in the blood. Here is a suggested protocol:

- **Strong decoction of velvet leaf:** Take 2 ounces 4x daily.
- **Tea from sea buckthorn and boneset leaves:** Add 1 ounce each of sea buckthorn leaf and boneset leaf to a quart jar (that can take heat), add boiling water, cover, and let steep 1 hour. Drink throughout the day, every day, for 2 weeks.
- **Sea buckthorn oil:** ½–1 teaspoon 3x daily.
- **Cat's claw:** Tincture, 1 teaspoon 3–6x daily, or capsules, 1,200mg 3–6x daily, for 2 weeks.
- **Tincture combination** of houttuynia, licorice and Chinese skullcap, equal parts of each, 1 teaspoon 3–6x daily for 2 weeks.
- **Pomegranate juice:** Drink throughout the day.
- **Vitamin C effervescent salts:** As needed if the velvet leaf causes constipation.

For dengue encephalitis use the dengue protocol outlined in Chapter 4.

Chikungunya and Zika

There are some good studies, both in vitro and in vivo on plants active against the chikungunya virus; those herbs are included in the protocol. There is much less on Zika, however the Chinese have been using a form of *Andrographis paniculata* with good success. That one herb seems to be highly effective for these kinds of infections. Here are the protocols. (Note: All the herbs should be taken; tinctures can be combined in liquid of choice. In severe cases, dosages can be increased.) I have disseminated these protocols in various places on the internet and heard from a few people using them that they have helped, reversing the infections when pharmaceuticals had failed to help.

CHIKUNGUNYA TREATMENT

- ***Andrographis paniculata* (andrographis):** Capsules or tablets, 600–1,200 mg 3–6x daily.
- ***Polygonum cuspidatum* (Japanese knotweed):** 1 tablespoon root powder 3x daily in liquid or 1 tablespoon tincture 3x daily, or resveratrol tablets made from knotweed root, 1,000 mg 3–6x daily.
- ***Glycyrrhiza* spp. (licorice):** Tincture, ½ teaspoon 3–6x daily for 30 days.
- ***Salvia miltiorrhiza*:** Tincture, 1 teaspoon–1 tablespoon 3–6x daily.
- ***Eupatorium perfoliatum* (boneset):** Strong infusion 3–6x daily.
- ***Azadirachta indica* (neem) leaf:** Strong infusion 3–6x daily.

ZIKA TREATMENT

- ***Andrographis paniculata* (andrographis):** Capsules or tablets, 600–1,200 mg 3–6x daily.
- ***Glycyrrhiza* spp. (licorice):** Tincture, ½ teaspoon 3–6x daily for 30 days.
- ***Isatis* spp.:** Tincture, ½–1 teaspoon 3–6x daily.
- ***Houttuynia cordata* (houttuynia):** Tincture, ½–1 teaspoon 3–6x daily.
- ***Scutellaria baicalensis*:** Tincture, ½–1 teaspoon 3–6x daily.
- ***Astragalus membranaceus* (astragalus):** Tincture, ½–1 teaspoon 3–6x daily.

For Zika with neural complications, add:

- ***Polygonum cuspidatum* (Japanese knotweed):** 1 tablespoon root powder 3x daily in liquid or 1 tablespoon tincture 3x daily, or resveratrol tablets made from knotweed root, 1,000 mg 3–6x daily.
- ***Pueraria lobata* (kudzu) root:** Tincture, ½–1 teaspoon 3–6x daily.
- ***Polygala tenuifolia* (Chinese senega) root:** Tincture, 30 drops 3x daily for 30 days.
- ***Hericium erinaceus* (lion's mane):** Tincture, 1 teaspoon 2x daily.

Enterovirus 71

Some herbs and supplements effective for enterovirus 71 are, in alphabetical order, *Amomum villosum*, *Ampelopsis brevipedunculata* (porcelain berry, invasive), *Azadirachta indica* (neem), *Elaeagnus oldhamii*, *Euchresta formosana* (shan dou gen), *Ficus pumila*, *Forsythia suspensa* (lian qiao), *Glycine tomentella*, *Glycyrrhiza glabra* (licorice), houttuynia, *Kalanchoe gracilis*, *Laminaria japonica* (kombu), *Ledebouriella divaricata* (fang feng), *Lemmaphyllum microphyllum*, *Lonicera japonica* (Japanese honeysuckle, invasive), *Melastoma candidum* (invasive), *Melissa officinalis* (lemon balm), *Ocimum basilicum* (basil), *Origanum vulgare* (oregano), *Phragmites communis* (a.k.a. *P. australis*; invasive), *Polygonum chinense* (Chinese knotweed), *Polygonum multiflorum* (fo-ti), *Psidium guajava* (guava), *Pueraria lobata* (kudzu, invasive), *Rheum officinale* (rhubarb), *Rosmarinus officinalis* (rosemary), *Schisandra chinensis* (schisandra), *Spatholobi caulis*, *Thymus vulgaris* (thyme), *Toona sinensis*, *Zingiber officinale* (ginger).

TREATMENT

Enterovirus 71 is an emerging pathogen that has no reliable pharmaceutical treatment. Numerous invasives are specific for this disease. I would begin with these herbs as systemic antivirals (then add others specific to the symptom picture): porcelain berry, neem leaf, licorice, houttuynia, fo-ti, ginger.

Japanese knotweed (*Polygonum cuspidatum*), a.k.a. hu zhang (huchang), has not been tested for activity against enterovirus 71 to my knowledge but its constituents and actions are very similar to the two polygonums that have been tested. This is of interest to me because Japanese knotweed is an invasive and thus presents a large, easily available source of medicine.

Interestingly, a Chinese combination used for measles for several millennia has been found effective in the treatment of enterovirus 71. It is composed of black cohosh rhizome, kudzu root, red peony root, and licorice. It's a nice combination, though I don't know where you can easily find it already prepared in the West. The kudzu and the licorice are antiviral. The licorice is synergistic and immune potentiating, the kudzu and peony neuroprotective, and to some extent so is the licorice. The black cohosh is pain relieving. You can, however, buy the herbs,

most of them, from 1st Chinese Herbs (https://1stchineseherbs.com). Just because of my own preferences, I would approach my own formulation slightly differently.

In addition to the systemic antibacterials, lemon balm, especially a prepared oil infusion, is specific for the blistering that enterovirus 71 can cause on the palms and soles of the feet. If neurological complications occur, you need to add specific anti-inflammatories for the brain and neural protectors. Kudzu is a good one, and a decent choice since it is active against the virus. My general preference for anti-inflammatories in the brain these days are Japanese knotweed, Chinese skullcap, greater celandine, and lion's mane.

For simple, uncomplicated enterovirus 71, here is a suggested protocol:

- **Tincture combination** of licorice, houttuynia, and porcelain berry, in equal parts. Dosage: From 1–5 ml, 3–6x daily, depending on the severity of the symptoms and age of child.
- **Tea** of ginger juice, neem leaf, honey, squeeze of lime, pinch of cayenne. Dosage: 1 cup 3–6x daily.

For specific symptoms or complications, incorporate these additional formulations into the protocol:

For blisters: Lemon balm infused oil or cordial, applied topically to blisters, as many times daily as seems appropriate.

For diarrhea: Blackberry root infusion, consumed throughout the day as tea, with honey added as desired. Pomegranate juice is highly suggested as it is antiviral, will reduce the cytokine cascade in the body, replaces fluids, helps reduce diarrhea, and is a good synergist. A combination of pomegranate and cranberry juice is just about perfect for this.

For fever: Boneset tea, 4–6x daily. Pasque flower tincture, 5–10 drops every hour or so, can also help. It will also help reduce anxiety levels.

For encephalitis and meningitis: I would essentially use the protocol outlined in Chapter 4. However, add a tincture combination of fo-ti, cordyceps, and Chinese skullcap, in equal parts, ¼–½ teaspoon (or more, depending on the severity of symptoms) up to 6x daily. This will reduce the cytokine cascade in the brain and CNS, protect neural structures, and reduce the symptom picture. Lion's mane

tincture, at the same dosage, will help restore neural function. If neurological symptoms are very severe, add greater celandine tincture, 30–60 drops, 3–6x daily for a max of 30 days.

Note: When treating a child, all dosages need to be adjusted for the child's weight and age; see page 384 for specifics.

Enterovirus D68

TREATMENT PROTOCOL, GENERAL

- **Astragalus and *Salvia miltiorrhiza* tincture combination,** equal parts, 1 teaspoon 3x daily.
- **Houttuynia tincture,** ½ teaspoon 3x daily.
- **Kudzu root, licorice root, red peony root tincture combination,** equal parts, 1 teaspoon 3x daily.

Additionally, if needed for severe lung impairment/difficult breathing:

- **Datura leaf, inmortal (or pleurisy root), lobelia tincture combination,** equal parts, ¼–½ teaspoon 3x daily or smaller doses as needed during the day.

This protocol will reduce viral replication, inhibit inflammation, and modulate immune responses, including reducing the cytokine cascade that is caused during infection. The protocols for influenza in Chapter 2 can also be of benefit for lung damage and/or involvement.

TREATMENT PROTOCOL, CNS INVOLVEMENT

The central nervous system can be involved during the infection resulting in paralysis and a post-polio-like condition. The herbs specific for this are astragalus, *Salvia miltiorrhiza,* and kudzu root. Much of the CNS damage comes from perforin generation and its damage to CNS cellular tissue. Astragalus has been found in vivo to stop this and reduce the cytokine cascade that causes it. The salvia is specific for reducing the cytokine cascade and reducing viral presence. The

kudzu root is specific to reducing viral penetration and replication and is highly protective of brain and CNS structures.

- **Astragalus tincture,** 1 tablespoon 6x daily.
- ***Salvia miltiorrhiza*** tincture, 1 tablespoon, 6x daily to each hour if necessary.
- **Kudzu tincture,** 1 tablespoon tincture 6x daily.
- **Houttuynia tincture,** 1 teaspoon 6x daily.
- **Chinese skullcap tincture,** 1 teaspoon 6x daily.
- **Licorice root tincture,** 1 teaspoon 6x daily.

Take all until the condition resolves, usually short term, 30 days or less.

Epstein-Barr Virus

Some of the herbs and supplements effective for Epstein-Barr are *Ailanthus altissima, Alpinia galanga, Andrographis paniculata, Artemisia annua* (or artemisinin), *Azadirachta indica* (neem), *Calendula officinalis, Chrysanthemum indicum, Cochlospermum tinctorium, Coix lacryma-jobi, Curcuma longa* (turmeric), *Eucalyptus* spp., *Ganoderma lucidum* (reishi), *Glycosmis arborea, Glycyrrhiza glabra* (licorice), isatis, *Morinda citrifolia* (noni), *Opuntia streptacantha* (prickly pear cactus), *Passiflora incarnata* (passionflower), *Polygonum cuspidatum* (Japanese knotweed), *Prunus persica* (peach tree leaves), *Scutellaria baicalensis* (Chinese skullcap), *Thelypteris torresiana, Thuja* spp., *Usnea* spp., *Wolfiporia extensa* (a.k.a. *Poria cocos*, a.k.a. fu ling), *Zingiber officinale* (ginger).

Epstein-Barr acute episodes can often lead to severe chronic fatigue. Part of the fatigue comes from its impacts on mitochondrial function. It scavenges and damages mitochondria, so protecting mitochondria is important. The best herbs and supplements for this are cordyceps, *Leonurus cardiaca* (motherwort), *Passiflora* spp. (passionflower), rhodiola, schisandra, *Pueraria lobata* (kudzu), *Scutellaria baicalensis*, and N-acetylcysteine. Some of these are also very good immune herbs that will help raise immune function. Motherwort and passionflower will also help reduce anxiety and sleeplessness.

Epstein-Barr, during acute attacks, usually presents with a very severe, very painful sore throat, usually confined mainly to one pinpoint

location just at the back of the throat from the mouth. I haven't found *anything* that will truly relieve it. *Echinacea angustifolia* (not *E. purpurea*) tincture will help a bit, if you use the tincture full strength. Keep it on the tongue a bit, let the saliva be stimulated, then let the whole mix flow *slowly* over the affected area. (But it won't do anything for the disease itself.) The root of *Artemisia absinthium* can also help if you chew a fresh root, just a bit when needed. It will really cool (almost creating a freeze sensation) the back of the throat.

TREATMENT

- **Tincture combination of Chinese skullcap, isatis, and licorice**, in equal parts, ½–1 teaspoon 3–6x daily depending on the severity of the infection.
- **Fresh ginger juice tea** (see page 39), 3–6x daily.
- ***Andrographis paniculata* capsules or tablets,** 1,200 mg 3x daily for 30 days. (Caution: About 1 percent of people who use andrographis get a bad case of hives; if you do, discontinue the herb, and it will clear up in a week or so.)
- **Tincture combination of motherwort and passionflower,** in equal parts, ¼–½ teaspoon 6x daily.
- **Tincture combination of cordyceps and rhodiola,** in equal parts, ¼–½ teaspoon 3x daily.

If spleen enlargement occurs, use red root (*Ceanothus* spp.). With enlarged liver, milk thistle seed (*Silybum marianum*). With meningitis, use the protocols in Chapter 4 for encephalitic swelling.

Herpes Simplex Virus 1 and 2

There are many herbs (and supplements) useful against herpes simplex viruses; here are some of them: *Actinidia chinensis* (kiwi tree root), *Agrimonia pilosa* (hairy agrimony), *Andrographis paniculata* (andrographis), *Aristolochia debilis*, *Artemisia annua* (artemisinin), *Artemisia anomala*, *Astragalus membranaceus* (astragalus), *Azadirachta indica* (neem), the berberine plants, *Bidens pilosa* (bidens), *Boussingaultia gracilis*, *Byrsonima verbascifolia*, *Caesalpinia pulcherrima* (a strong decoction of the flower is strongest, followed by stem/leaf prepara-

tions), *Carissa edulis* (root bark), *Centella asiatica* (gotu kola), *Cordyceps sinensis* (cordyceps), *Crossostephium chinense*, *Cryptolepis sanguinolenta* (cryptolepis), *Cynanchum paniculatum*, *Distictella elongata*, *Ganoderma lucidum* (reishi), *Geum japonicum*, *Glycyrrhiza glabra* (licorice), honey, houttuynia, isatis, *Juniperus* spp., *Limonium brasiliense*, *Lindera strychnifolia*, *Melia azedarach* (chinaberry leaves, invasive in the United States), *Melissa officinalis* (lemon balm), *Ocimum americanum* (hairy basil), *Ocimum basilicum* (basil), *Ocimum sanctum* (holy basil, strongest of the three), *Patrinia villosa*, *Phyllanthus niruri*, *Pinus massoniana*, *Pithecellobium clypearia* (a.k.a. *Archidendron clypearia*), *Pongamia pinnata* (seeds), *Prunella vulgaris* (self-heal), *Psidium guajava*, *Punica granatum* (pomegranate juice), *Pyrrosia lingua*, *Rheum officinale* (rhubarb root), *Rhus aromatica* (fragrant sumac), *Rhus chinensis* (Chinese sumac), *Rhus javanica*, *Rosmarinus officinalis* (rosemary), *Salvia officinalis* (sage), *Sargassum fusiforme*, *Scutellaria baicalensis* (Chinese skullcap), *Serissa japonica* (a.k.a. *S. foetida*), *Sida acuta* (sida), *Stephania cepharantha* (stephania), *Syzygium aromaticum*, *Taraxacum mongolicum*, *Terminalia chebula*, *Thymus vulgaris* (thyme), *Usnea* spp., zinc, zinc sulfate cream.

Byrsonima verbascifolia is a South American herb; it is particularly antiviral for these two viruses but is hard to get in the United States. Bidens, reishi, licorice, and houttuynia are also especially strong. A combination Chinese formula, yin chen hao tang, composed of *Artemisia capillaris*, *Rheum officinale*, and *Gardenia jasminoides*, has shown good effect against HSV-1 and HSV-2.

Terminalia chebula, *Syzygium aromaticum*, *Rhus javanica*, and *Geum japonicum* have all been found potent against HSV serotypes. They have particularly strong actions in the brain and are synergistic with acyclovir, enhancing its actions and impacts. The use of these herbs, singly, or in combination, has been found to prevent recurrence of HSV blooms in vivo (mice). All are traditional herbs, long used in community medicine. They all have activity against a variety of viruses.

TREATMENT

- **Systemic antiviral formulation:** A combination tincture with equal parts of licorice, isatis, houttuynia, and sida, ½ teaspoon 3x daily. Houttuynia is particularly effective in reducing the cytokine cascade the virus starts, thus strongly inhibiting it. (In severe systemic infections, double the dose to 6x daily.)
- **Immune formulation:** A combination tincture with equal parts of astragalus, cordyceps, and reishi. (Reishi is particularly strong against these viruses.) Tonic dose: 20–60 drops 3x daily. In acute episodes: ½ teaspoon up to 6x daily.
- **Zinc,** internally, 25 mg daily. Double that for a few days if you feel an attack coming on.

To prevent outbreaks, try L-lysine, in a tonic dose of 1,000 mg 3x daily. (Note: If you do have an outbreak anyway, increase the dosage, up to 3,000 mg 3x daily. However, if taken regularly, the tonic dose can help prevent outbreaks from occurring.) Another helpful supplement is vitamin B_{12}, 500 mcg daily (a good B-complex is very helpful as well). Avoid L-arginine supplements and foods containing L-arginine such as nuts and chocolate. These can stimulate outbreaks.

To treat active sores, you can use zinc sulfate cream, applied 6–10x daily. This can reduce and eliminate sores within 3–5 days. The concentrated sore-relief herbal cream on page 196 can do the same even more quickly, within 3 days.

To reduce nerve pain, some of the things that can help are:

- **Geranium oil,** topical, applied as needed. If the pure essential oil is too strong, you can dilute it with olive oil if necessary.
- **Greater celandine (*Chelidonium majus*) tincture.** Typical American dosage is 10–30 drops 3x daily for 30 days. English dosage is higher, generally 40–80 drops 3x daily, again for 30 days. In rare instances use for longer than 30 days can cause severe inflammation of the bile ducts, so only use it for 30 days in a row.
- **Kudzu root (*Pueraria lobata*) tincture,** ½ teaspoon 3 or 4x daily. This alters certain neural receptors in the brain and peripheral nervous system.

- **Theramine** can also help, sometimes a lot. It is a prescription nutriceutical that is very good for certain kinds of recalcitrant pain.
- **Pasque flower tincture,** 10 drops every hour or so.

For some specific expressions of HSV, incorporate these additional formulations into the protocol:

For vaginal herpes: Prepare a douche by combining 1 ounce of a berberine plant (e.g., goldenseal, barberry) tincture and 1 ounce of lemon balm tincture in a pint of water and use it to douche 3x daily.

For herpes eye infection: Prepare eyedrops by combining 1 ounce each of dried licorice, houttuynia, and isatis in a heat-proof quart jar. Bring water to a boil, then pour it over the herbs, stir well, cover, and let sit overnight. Strain well the next day. Pour some of the infusion into a 1-ounce brown bottle with dropper. Store the rest in the refrigerator. Use the eyedrops throughout the day, a minimum of 6x daily. Just 1–3 drops in each eye every hour or two until the condition clears.

For herpetic encephalitis/meningitis: Use the protocols for viral encephalitis delineated in depth in Chapter 4. An ethanol extract of *Cynanchum paniculatum* has been found to be very effective in protecting the brain and neural structures in some studies and may prove useful here.

Caution: The use of L-arginine during an *active* herpes outbreak can sometimes exacerbate the condition excruciatingly.

Varicella Zoster Virus (Chicken Pox/Shingles)

Although chicken pox (varicella zoster virus) tends to be a self-limiting disease a vaccine has been developed in the West; a variety of it has also been crafted for herpes zoster (shingles). Shingles tends to be the most problematic element of this virus; it is often extremely painful and somewhat difficult to treat well.

I won't explore the use of herbs for chicken pox itself since, with the vaccine, episodes in the West are becoming rare. However, modifications of the protocols outlined for shingles will help with chicken pox, especially the systemic and topical formulations. Treatment protocols in this section are specific only for shingles.

Some of the herbs and supplements effective for herpes zoster (shingles) are *Ampelopsis brevipedunculata*, *Astragalus membranaceus* (astragalus), *Clinacanthus nutans*, *Ficus binjamina* (leaves), *Garcinia*

multiflora, *Glycyrrhiza glabra* (licorice), isatis, *Lonicera japonica* (Japanese honeysuckle), *Melissa officinalis* (lemon balm), *Polygonum cuspidatum* (Japanese knotweed), *Quillaja saponaria* (Chilean soapbark tree, inner bark infusion/decoction, low dosing), *Rhus succedanea*, *Ribes nigrum* (black currant).

Concentrated Sore-Relief Herbal Cream

INGREDIENTS

- 3 ounces licorice root
- 2 ounces birch bark
- 2 ounces lemon balm leaf
- 2 ounces rosemary leaf

To make:

Combine the herbs in a slow cooker with 32 ounces of water. Bring to a boil. Reduce the heat to barely under a simmer. Cook for 3 days. Turn off the heat, let cool, strain out the herbs. Return the liquid to the cleaned slow cooker, bring to a boil again, then reduce heat to just under a simmer. Let cook until the liquid is reduced to 2 ounces. (Caution: It can easily burn once it gets close, so watch it.) When reduced sufficiently, turn off the heat, let cool, and place the cream in a jar.

To use:

Apply 6–10x daily. This combination will eliminate sores, reduce pain, and promote healing very quickly, usually within 3 days or so.

TREATMENT

The basic treatment for shingles comprises 1) systemic antivirals, 2) an immune formulation, 3) topical creams for skin outbreaks, and 4) treatments to reduce nerve pain and regenerate nerve cells.

1. **Systemic antivirals.** I would suggest both of the following:

   - **Tincture combination of licorice, isatis, and Chinese skullcap,** in equal parts, ¼–½ teaspoon 3–6x daily, depending on the severity of the outbreak. This will actually help heal the nerves of the infection and prevent recurrences, if you use it over time.
   - **Lemon balm tincture** (I suggest the Herb Pharm brand), ¼ teaspoon 3–6x daily. This is *very* helpful over time. It is also very calming to the nerves and helps prevent recurrences.

2. **Immune formulation.** Tincture combination of astragalus, rhodiola, and cordyceps, in equal parts, ½ teaspoon 3x daily. This will increase immune function and help reduce the incidence of future outbreaks. (Lowered immune function due to age is the main cause of shingles outbreaks after age 50.)
3. **Topical healing.** A couple of formulations can help here:

   - **Lemon balm infused oil or cordial,** applied topically daily. This really does help.
   - **Pine pollen cream,** applied topically daily. This really does help if you can find the cream.

4. **Nerve pain.** The herbs and supplements recommended for reducing the nerve pain of herpes simplex virus outbreaks (see page 194) will also help reduce the nerve pain that accompanies a shingles outbreak.

Herbs can also help prevent or cure the postherpetic nerve pain that comes from nerve damage after outbreaks by regenerating the damaged nerves. Two of the best regenerators (both very high in nerve growth factor) are Chinese senega root and lion's mane; vitamin B_{12} is also very helpful:

- **Chinese senega root:** 30 drops of the tincture 3x daily for 30 days, and . . .
- **Lion's mane (*Hericium erinaceus*):** 1 teaspoon of the tincture 2x daily. Note: The fresh mushroom tincture, not the dried, is preferable if you can find it. Long-term use of this herb is fine. And . . .
- **Vitamin B_{12}:** 500–2,000 mcg daily.

Supplements for both deep healing and nerve pain:

- **L-lysine:** as a tonic dose, 1,000 mg 3x daily; during a shingles outbreak, up to 3,000 mg 3x daily.
- **L-carnitine:** 500–700 mg 3x daily.
- **Alphalipoic acid:** 200 mg 3x daily.
- **Inositol:** 500–1,000 mg 3x daily.

Regular fruit intake can also be important. Low fruit intake (less than one serving per week) can lead to more frequent outbreaks.

Note: L-arginine intake can sometimes *cause* a shingles outbreak. If you have a history of shingles, I would recommend avoiding L-arginine supplementation. (Nuts and dark chocolate are fairly high in L-arginine; caution is warranted.)

Rotavirus and Norovirus

Rotaviruses are the best studied of the viral gastroenteritis diseases, in terms of herbal treatments, but for all of them, tannin-containing plants are indicated for one big reason: they inactivate the viruses. In the case of the Norwalk virus, for example, tannic acid inhibits the binding of the viral proteins to HBGA (histo-blood group antigen) receptors, thus preventing infection. There have been a number of double-blind studies using tannic-acid-containing plants in the treatment of viral gastroenteritis, and all have shown good success. Researchers have tested plants traditionally used for this kind of condition and found many of them active. The most common and strongest: *Musa* spp. (the green, unripe banana fruit, usually cooked), *Potentilla erecta* (leaves), *Potentilla tormentilla* (root), and *Psidium guajava* (guava).

The plants found effective for rotaviruses are *Aegle marmelos* (unripe fruit), *Artocarpus integrifolia* (bark), *Byrsonima verbascifolia, Eugenia dysenterica* (a.k.a. *Stenocalyx dysentericus*), *Glycyrrhiza glabra* (licorice), *Haemanthus albiflos* (bulb), *Hymenaea courbaril, Lomatium dissectum, Myracrodruon urundeuva, Myristica fragrans* (seeds), *Panax ginseng, Potentilla erecta* (leaves), *Potentilla tormentilla* (root), *Wolfiporia* (a.k.a. *Poria*) and *Polyporus* in combination (as a decoction), *Psidium guajava* (leaves), *Punica granatum* (pomegranate leaves and juice), *Quillaja saponaria, Sophora flavescens, Spondias lutea* (a.k.a. *S. mombin*; leaves and bark), *Stevia rebaudiana, Vaccinium macrocarpon* (cranberry juice).

Plants have not been tested to any extent against astroviruses though *Detarium senegalense* and *Dichrostachys glomerata* are both active against the viruses. For orthoreoviruses, the barks of both *Castanea* (chestnut) and *Schinopsis* (quebracho) are active antivirals as well as tannin sources in general.

In essence, any plant that is strongly drying on the tongue is going to work: oak leaves or bark, *Krameria* (rhatany) root, pine needles (mature), acacia, agrimony, pinedrops (*Pterospora*), rose, raspberry, blackberry, and so on. Roots, leaves, and bark tend to be the most astringent parts of such plants. In my experience rhatany root, blackberry root, and pinedrops are some of the strongest, followed by quebracho, oak, and pine needles, but really, any astringent plants will do. It is just that the stronger they are, the faster they work.

Other useful plants are:

- **Berberine-containing plants.** These plants do have some antiviral properties but they also have strong actions on the GI tract membrane and help it resist any microbial infection.
- ***Bidens* spp.** These plants are strong, systemic antimicrobials and prostaglandin inhibitors and are very specific for diarrheal diseases and healing damaged mucous membrane systems.
- ***Alchornea cordifolia.*** This plant is a potent systemic antibacterial and antimicrobial that is used in traditional African medicine for a variety of conditions, including diarrhea. It has been found to reduce water and electrolyte loss during diarrheal diseases.
- ***Baccharis teindalensis, Carica papaya, Croton lechleri, Euphorbia hirta, Jatropha curcas, Jussiaea suffruticosa, Mangifera indica, Terminalia avicennoides, and Zingiber officinale.*** All have been found in a number of studies to help reduce diarrhea, limit water and electrolyte loss, and help with cramping.

TREATMENT

The treatment of these viral gastroenteritis diseases is straightforward, and while you can get fancy with this, it primarily entails the use of any plants strong in tannins, in large quantities. My preference is for their use as decoctions but even that is not necessary; unripe banana is especially useful in the regions in which it grows, for example, and is usually used as a mashed, cooked ingestible. Plants high in tannins bind the viruses while, at the same time, reducing fluid loss through firming up the stool.

Some suggestions:

- **Combination tincture of *Alchornea cordifolia* (or any berberine-containing plant), licorice, and lomatium,** in equal parts, 30 drops–1 teaspoon 3–6x daily depending on the severity of the condition and age of the person.
- **Strong infusion of blackberry root:** Add 4 ounces powdered or roughly ground blackberry root to a large heat-proof jar. Add 1 quart of very hot water, cover, and let steep overnight. Drink the whole thing over the next day. Repeat daily. Increase the dosage if diarrhea is not substantially helped within 24 hours. Or . . .
- **Strong decoction of blackberry root:** 4 ounces blackberry root to 1 quart of water; boil until the water is reduced by half. Let cool and consume in equal parts throughout the day. Repeat daily.

6

HERBAL ANTIVIRALS: THE MATERIA MEDICA

A large number of structurally unique antiviral compounds from medicinal plants (herbs) have been identified. The advantages of natural compounds are fewer side effects in comparison to orthodox medical drugs, and the production of synergistic effects for a more positive treatment outcome.

—Kaio Kitazato et al., "Viral Infectious Disease and Natural Products with Antiviral Activity"

There has been very little work in the popular press (especially in the West) on herbal antivirals—and most of what has occurred is embarrassingly poor. There are a number of reasons for this.

The field of antivirals itself, whether medical or herbal, is in relative infancy, which partly explains the problems in the literature. The general overemphasis on bacteria as disease-causing agents in the public (and medical) mind, irrespective of culture, also contributes to the problem. Then, there is the nature of viruses themselves and the difficulty of actually creating effective pharmaceutical antivirals. There are, in fact, very few pharmaceutical antivirals compared to antibiotics—generally people only hear of two: ribavirin and Tamiflu (oseltamivir). This is a much reduced pharmaceutical armamentarium compared to the scores of antibiotics that are common in most people's vocabularies; *penicillin* is, I suspect, a word known to most of the world's population.

Most of the scientific research (at least in the past and especially in the West) on viral treatment has been focused not so much on finding effective antivirals but on vaccines. And researchers have been pretty

successful at this over the past 50 years—the eradication of smallpox is one of the great triumphs of technological medicine, as is the polio vaccine. So, we have vaccines now for an increasing number of viral diseases: smallpox, polio, measles, hepatitis B, influenza strains, and so on.

Thus, the focus of most medical viral research, in contrast to bacterial research, has been on vaccines. In consequence, few people ever think of *antivirals* as a specific entity that might be useful in medicine; *antibiotic* is a word that everyone has heard of, has used, and knows. The word *antiviral* is not. Nevertheless, antivirals do exist in great quantity in the world. In plants.

Viruses are an intimate part of life on this planet and every life form, including plants, has experienced viral infections during the billions of years there has been life here. Plants, the finest chemists on Earth, have created, just as they have in their dealings with infective bacteria, a wide range of compounds in response to viral infection. Similarly to plant antibiotics and bacteria, while *all* plants have created a variety of compounds to protect them from viruses, some, when used as medicines, tend to be a great deal more effective than others. The trick is to find which are the most effective, the most reliable, the most potent.

The strongest herbal antivirals are more easily revealed *if* the medicinal plant world is examined through a number of lenses and then, afterward, those findings are cross-correlated. The lenses I used for this book are:

- The history of the plants' uses in community medicine in whatever cultures have access to them—what some people call indigenous or traditional practice;
- The history of the plants' uses in developed medical systems such as traditional Chinese medicine, Ayurveda, or Western botanic practice;
- Contemporary uses of the plants among community herbalists;
- Outcome experiences among peoples who are using the plants for healing;
- Scientific study of the plants' medicinal actions as viewed through in vitro, in vivo, and human clinical study[1]; and finally
- A factor that I have found a primary indicator of strong medicinal action—the invasive status of the plant. For, interestingly enough, many of the strongest antibacterial and antiviral plants are invasives.[2]

The Top Seven Antiviral Herbs

Chinese skullcap
Elder
Ginger
Houttuynia
Isatis
Licorice
Lomatium

And while I am not yet using this as a primary identifier, it is becoming evident that many of the most potent antivirals are also synergists. This is a relatively new category of herbal medicines—in the West at any rate. Synergists are plants that, when used with other medicinal substances (herbs, supplements, pharmaceuticals), through a variety of mechanisms, *increase* the potency of those substances against microbial pathogens. A number of the herbs in this book are very potent synergists.

The plants that show strong activity when viewed through a majority of these lenses end up on the list. It is then the final factor comes into play: access. There are some truly magnificent antiviral and antibacterial plants in Africa, South America, and China that simply are not to be had in the Western world, no matter how actively they are sought—unless you travel to those places. The plant medicines in this book tend to be somewhat easy to find and that is important. It's no good to know of a great antiviral if you can't find any of it to use as medicine.

Many of the antiviral herbs in this book are broad-spectrum antivirals, that is, they are active against a wide range of viruses. I tend to think of these as the most potent antivirals in a general sense (e.g., Chinese skullcap). There are others with a more narrow range but that are very antiviral for specific viruses (e.g., ginger, elder). *All* of them have shown potent activity in historical use across long timelines. *All* of them have been found effective in contemporary usage. Nevertheless, these are not the only antiviral herbs there are. These are just the ones that I have used with success, the ones that have shown up the most strongly in this examination of them, in this particular year, for these particular viruses.

Again, there are *a lot* of great herbal antivirals out there; over time, more will be understood and discussed *and* used as medicine. So, don't think these are the only ones to use; they are just the ones that I have used the most, that have the deepest historical use, that have the greatest presence in the literature, and that have the best research on them.

In this section I include the top seven antiviral herbs, five honorable mentions, two very useful antiviral supplements, and one truly important supportive herb for nearly all viral infections. Here they are.

Chinese Skullcap

Family: Lamiaceae or is it the Labiatae, or are those synonyms? (I feel another taxonomic rant coming on.)

Species used: *Scutellaria baicalensis* is the primary species used in China and the one meant when Chinese skullcap is talked about (and the one this monograph will focus on). It most definitely does not mean the American skullcap, *Scutellaria lateriflora*—or any of the other American species. For reasons I will discuss in this monograph I strongly suggest you *not* use *S. lateriflora* as a substitute for treating viral infections. Note that *Scutellaria macrantha* is a synonym for *S. baicalensis*.

Common names: Legion for the many different skullcaps, however for *S. baicalensis*: English—Chinese skullcap, baikal skullcap, scute (really hate that one—"you are so *scute*"), golden root (really like that one). Chinese—huang qin. (Ban zhi lian is the Chinese for *S. barbata*).

Part Used

The root and the root only—generally only from plants older than 3 years. There is reason to believe that the increase in pharmacological action of Chinese skullcap over the usual American species used as medicinals is due to the difference between using the root in Chinese practice and the leaf in American practice. As far as I know there has been virtually no examination of the root as a medicinal in any of the Western species.

There are, as well, no *substantive* studies on the difference between roots and leaves of any species of skullcap as regards their chemical compounds. The few I have seen that touch on it do show substantial differences between the leaf and the root and this really does need to be explored, especially if American herbalists are insisting that the American skullcaps are interchangeable with the Chinese. (And some of them are.) However, as some researchers have commented, "The results showed that the components and relative contents of the essential oils among flowers, stem, leaves, roots, and seeds have significant differences."[3] This is, of course, true of nearly all plants on Earth and one of the most basic understandings of herbal medicine.

Preparation and Dosage

Medicinals prepared from this plant are a bit hard to find in the United States, though not impossible. (I know only a few sources for Chinese skullcap root tinctures, but hundreds for the American leaf tinctures.)

TINCTURE

If making it yourself, again, use the root. After harvesting the root, cut it into easy-to-use pieces, let it dry in a cool, shaded location, then powder the root pieces and tincture them. The ratio should be 1:5 (one part herb, five parts liquid), with the liquid being 50 percent alcohol, 50 percent water. Take ¼–½ teaspoon 3x daily. In acute conditions, double that. Remember: If using for CNS damage or encephalitis, you want to flood the brain and CNS with the compounds over a long enough time period to sharply reduce the inflammation and protect and restore the neural structures of the brain. I think the tincture best for this purpose.

For sleep: The plant and root are high in melatonin, so they can help with sleep. If you are using it for that, take just before bedtime, ½ teaspoon of the tincture.

Fresh leaf tincture: If you want to use the aerial parts of the plant, get or make a 1:2 tincture of the fresh leaves and stems. Tonic doses run from 10–30 drops up to 6x daily but I have taken up to ½ ounce of the tincture at a time (of the American skullcaps anyway) without side effects; there apparently are none even at high doses.

POWDER

The Chinese dosages are large, as usual, generally 3–9 grams at a time. Most of the clinical studies and trials used similar dosing. If you are using capsules this is the dosage range you should be exploring, divided into three equal doses every 4 hours or so. Capsules are pretty much impossible to find but you can get the powdered herb fairly easily. I would use 1 teaspoon of the powdered root 3–6x daily.

Note: The herb reaches peak levels in the plasma and body organs in about 1 hour and only lasts in the body for about 4 hours, so you really do need to dose about every 3–4 hours.

AS A WASH

The fresh juice of the plant can be used as an eye wash for eye infections, as can the cooled infusion or decoction of the root.

Exploring *Scutellaria* Species

The Chinese do use another skullcap species, *S. barbata*, which is considered much weaker but still specific for certain conditions. There is, however, an important distinction about the medicines made from these various skullcap species. The most studied species, and the one considered the strongest, is *S. baicalensis* and with that species the Chinese use the root *only*. (See "Part Used" and "Western Botanic Practice" for more on all this.) With *S. barbata* and the various American species the aerial parts are used. This difference, aerial part versus root, makes *all* the difference in the medicinal impacts of the plants. The leaves just aren't as strong, especially for viral infections. After several millennia of use, the Chinese consider all other skullcaps to be inferior to *S. baicalensis* in their medicinal effects. And I assume (makes an ass of u and me) that they must have tried the roots of the other species and found them not as strong; nevertheless, what if they didn't . . . ? Perhaps we in the West should do a little experimenting with some roots of our own.

Now, that being said, there are 200, or 300, or 350 species in the genus *Scutellaria* . . . taxonomists are absolutely positive about those figures. Many of the various skullcaps are used similarly as medicines in the regions in which they grow; most of them contain the same constituents. However, there has been very little study on species other than *S. baicalensis*. As illustration, there are over 600 journal articles on PubMed on *S. baicalensis* (and its major constituents), but only 88 on *S. barbata*, just 24 on *S. lateriflora* (the main American species), a mere eight on *S. viscidula*, six on *S. indica*, four on *S. racemosa*, three on *S. galericulata*, and one each *on S. regeliana*, *S. incana*, *S. taiwanensis*, and *S. austrotaiwanensis.* There's even less on the others.

Some studies have shown that *S. baicalensis*, *S. barbata*, *S. lateriflora*, and *S. racemosa* have similar constituents in their roots—but then the roots of those other species are almost never used in medicine making so there

Side Effects and Contraindications

Side effects from skullcap are rare, mostly gastric discomfort and diarrhea. It should not be used during pregnancy. Caution should be exercised if you are taking pharmaceuticals as it can increase the bioavailability of the drugs, thus increasing their impacts. It may interact

is no way to know if in practice they are as potent as the Chinese skullcap root. (If you try roots other than those of *S. baicalensis*, let me know how they work. Let's start something here.)

S. lateriflora, a.k.a blue skullcap, mad dog skullcap, Virginia skullcap, hoodwort (along with *S. galericulata*, i.e., marsh or common skullcap), is the species most commonly used in the United States. *S. racemosa* is more common in South America and *S. barbata* is the other main species used in traditional Chinese medicine. Constituent studies have found that those three varieties all contain baicalin, baicalein, scutellarin, wogonin, melatonin, and serotonin—the constituents considered to be the most active in Chinese skullcap. Other studies, on the roots of *S. viscidula* and *S. amoena*, found very similar constituents in *them*. It does seem that most of the species contain baicalein, baicalin, and wogonin at the very least—though the amounts in differing species have not been *comparatively* studied to any degree.

However, one study that did examine the constituent levels in the roots of *S. planipes* and compared them with *S. baicalensis* found that the root constituents were very similar in nature, degree, and number, and were equally active as antibacterials and antiallergics. Another study (Chinese) showed that the root of *S. rivularis* stimulated the production of monoclonal antibodies to encephalitis virus E protein, just as the root of *S. baicalensis* does.

So . . . it may turn out that many of the skullcaps can be used interchangeably with *S. baicalensis* as medicines—but *only* if the roots are used, which they generally aren't. Again, I think the roots of various skullcap species should be explored to find out if they can be used interchangeably with the Chinese variety. So, if you are using the American skullcaps, try harvesting some of the root of whatever species you are growing, and make it into medicine and find out how it works. (Then email me.)

additively with blood-pressure-lowering drugs. Type 1 diabetics should exercise strong caution with the herb as it can affect insulin and blood sugar levels.

Herb/Drug and Herb/Herb Interactions

Lots. Chinese skullcap is a synergist, perhaps as efficacious as licorice, ginger, and piperine, and should probably be added to that category of herbs. Among other things it inhibits the NorA efflux pump, which inactivates some forms of antibiotic resistance. Like the other synergists I know of, it is also a strong antiviral, which is beginning to stimulate speculation. Nevertheless, the herb strongly affects pharmaceuticals and herbs taken along with it.

Baicalein, one of the major compounds in *S. baicalensis*, is synergistic with ribavirin, albendazole, ciprofloxacin, amphotericin B.

S. baicalensis is strongly inhibitive of CYP3A4, a member of the cytochrome oxidase system. And this inhibition is dose dependent; the more you take, the more it is inhibited. CYP3A4 is a type of enzyme, strongly present in the liver, and is responsible for catalyzing reactions involved in drug metabolism. Many of the pharmaceuticals that are ingested are metabolized by the CYP3A4 system, meaning that some portion of the drug is inactivated, usually by being altered to another molecular form. With pharmaceuticals, the normal dosage range you are given is adjusted to take this metabolization into account. If you are using Chinese skullcap, then less of the pharmaceutical is going to be metabolized. In some cases this will make the impacts of the drug stronger, with the bioavailable dose higher. With other drugs it is the metabolites created by CYP3A4 that are active in the body. In this circumstance, since the herb inhibits CYP3A4, the metabolites of the pharmaceutical you are taking will be reduced in degree and have *less* effect in the body. Acetaminophen, codeine, cyclosporin, diazepam, erythromycin, and so on are all affected in one way or another. The herb does affect the amount of antibiotics that enter the system.

To make things more complicated, one of the herb's constituents, oroxylin A, is a strong P-glycoprotein inhibitor. P-glycoprotein is strongly present in the blood-brain barrier, the lining of the GI tract, renal tubular cells, capillary endothelial cells, and the blood-testes barrier. It reduces the amount of substances that cross over those barriers in order to protect what is on the other side. (This is why berberine is mostly confined to the GI tract.) P-glycoprotein inhibitors allow more of a substance to cross barriers that are high in P-glycoprotein. That means that if you are taking skullcap, any substance taken with it will

end up in higher levels in the bloodstream, thus increasing its impacts in the system.

This means that Chinese skullcap will act through two different mechanisms to increase drug and herb uptake in the body.

Also, because cancer cells use P-glycoprotein as a form of efflux pump in order to eject drugs designed to kill them from the cancer cells, Chinese skullcap will increase the effectiveness of anticancer drugs by inhibiting P-glycoprotein-mediated cellular efflux. Paclitaxel uptake, for example, was increased over twofold when administered with oroxylin A.

All this applies equally to herbs and supplements that you take along with skullcap. If you are using this herb along with licorice, which is also a potent synergist, through its own mechanisms (as is ginger), everything else you take will be much more potent and pronounced in the body. (This is one reason why licorice and skullcap are considered such important medicines in Chinese practice and why they are commonly added to many herbal combinations.) *Keep this in mind.*

Habitat and Appearance

This plant likes to grow in wettish, sandy, and rocky-type seashore or creekish locations in the wild, from sea level to 6,000 feet in altitude. It is native to east Asia: China, Mongolia, Japan, Korea, Siberia, Russia. (Many of the skullcaps like it wet and tend to grow along streambeds and creeks.)

It's a perennial growing up to a foot in height (hardy to zone 5). It flowers in August, seeds are ready in September. The plants are hermaphrodites and are pollinated by insects.

And I have to admit, Chinese skullcap is beautiful, the lanceolate leaves a vibrant green, the flowers the most delicious purple. The flowers are supposedly shaped like little skullcaps—people used to wear similar, though larger, ones in the Middle Ages . . . so they say. But *they* also said that the Earth was flat and that pharmaceuticals were safer than herbs and that there was no one on the grassy knoll.

I think Chinese skullcap the most beautiful of the skullcaps. Richo Cech, at Strictly Medicinal Seeds, describes it like this: "The purple flowers are like schools of dolphins breaking through green waves in a summer sea." Beautifully put and very apt.

It is commonly grown in the United States as a garden plant due to its resistance to drought and cold. It can survive nearly anything. (And so can you if you use it as medicine.)

Cultivation and Collection

Sow seeds outdoors in late spring (or in a pot or cold frame in early spring). The seeds generally sprout within 10 to 20 days. Separate the sprouts when large enough to handle. Grows easily in sunny locations in ordinary garden soil that remains a bit moist. Soil needs to be well drained in sun or partial shade. It is hardy to zone 5 and survives dips as low as -10°F (-23°C). It will survive drought pretty well once established.

You can harvest the aerial plant at flowering and make a fresh plant tincture (if that is how you are going to use it). But, again, the roots are more potent. They should be harvested after 3 years of growth (or more), cut to usable lengths, and dried carefully in the shade (though the Chinese do it in light sun). Once well dried, they should be stored in plastic bags in a plastic tub in a coolish location in the dark. Spring roots are more potent (as usual). Good-quality organic freshly dried roots should be yellowish, even a bright yellow, in color. Most of the imported Chinese roots tend to be a bit oxidized, the yellow color fading a bit. Poorly prepared or severely oxidized roots will be greenish or even black in color. Don't bother with them.

You can, if you wish, and if you are a fan of the aerial tinctures of the skullcaps, harvest the root *and* aerial parts of the plants and make a tincture from both, separately.

Properties of Chinese Skullcap

Actions

Chinese skullcap is a broad-spectrum antiviral. It inhibits hemagglutinin and neuraminidase, inhibits viral replication, suppresses viral gene expression, reduces viral RNA in infected cells, inhibits viral fusion with cells, protects cell membranes from virus-initiated cytokines, reduces the expression of the viral matrix protein gene, interferes with viral entry by interacting with viral envelope proteins and cellular CD4 and chemokine receptors, regulates the innate antiviral immunity of the host by modulating cytokine production at the time of viral insult, lowers host cell membrane fluidity thus inhibiting the formation of virus-induced membrane pores in host cells (which in itself stops viral entry into host cells), inhibits viral release from infected cells, inhibits viral cytokine cascades, increases apoptosis in infected cells, stimulates innate resistance to viral infection, promotes the development of monoclonal antibodies to encephalitis virus E protein, and is directly virucidal.

S. *baicalensis* is also:

Anodyne
Antianaphylactic
Antiangiogenic
Antibacterial
Anticholesterolemic
Anticonvulsant
Antidiarrheal
Antidysenteric
Antifungal
Antihypertensive
Anti-inflammatory
Antimetastic
Antioxidant
Antispasmodic
Antitumor
Astringent
Cholagogue
Diuretic
Expectorant
Febrifuge
Hemostatic
Hepatoprotective
Sedative (mild)
Nervine
Neuroprotective

Active Against

This plant is a major broad-spectrum antiviral with a wide range of activity against viruses. It is active against:

Adenovirus (3 and 7)
Avian infectious bronchitis virus
Coliphage MS2
Coxsackie B virus (3, 4, and 5)
Epstein-Barr virus
Hepatitis A
Hepatitis B (resistant and nonresistant)
Hepatitis C
Herpes simplex viruses
HIV-1
Human T cell leukemia virus
Influenza A (H1N1, H3N2—resistant and nonresistant)
Influenza B
Measles virus
Mosaic virus
Parainfluenza viruses (in general)

Continued on next page

Continued from previous page

- Poliovirus
- Porcine reproductive and respiratory syndrome virus
- Respiratory syncytial virus
- SARS coronavirus
- Sendai virus (parainfluenza type 1)
- Vesicular stomatitis virus

Chinese skullcap is synergistic with other antivirals (as is licorice, they should be used together if possible). It does have a fairly wide range of action against bacteria and some other microbes but the effects are variable; by this I mean it is not primarily a major systemic antibacterial as cryptolepis is, for example. Many of its antibacterial and antiviral effects tend to be not direct, that is, through antimicrobial actions, but sideways through the stimulation of the body's own immune responses, reduction of cytokine cascades, protection of host cells. Antibacterially, the herb does have some really potent effects against some bacteria, particularly staph organisms, resistant and nonresistant. It is also nicely active against mycoplasma and some others such as klebsiella organisms. In general, the herb can be used as a primary adjunct to any treatment of resistant bacterial disease as it is synergistic with both herbs and pharmaceuticals, will markedly reduce cytokine cascades along a rather broad range, and has its own antibacterial actions to add to the mix.

The entire list of organisms it is active against includes:

- *Actinomyces viscosus*
- *Angiostrongylus cantonensis*
- *Bacillus subtilis*
- *Bacteroides melaninogenicus*
- *Bordetella pertussis*
- *Candida albicans*
- *Chlamydia trachomatis*
- *Corynebacterium xerosis*
- *Diplococcus pneumoniae* (a.k.a. *Streptococcus pneumoniae*)
- *Enterococcus faecalis*
- *Escherichia coli*
- *Helicobacter pylori*
- *Klebsiella pneumoniae*
- *Lactobacillus plantarum*
- *Micrococcus sedentarius* (a.k.a. *Kytococcus sedentarius*)
- *Microsporum audouinii*
- *Microsporum canis*
- *Mycobacterium smegmatis*
- *Mycobacterium tuberculosis*
- *Mycoplasma hominis*
- *Neisseria meningitidis*
- *Proteus vulgaris*
- *Pseudomonas fluorescens*
- *Salmonella* spp.
- *Shigella dysenteriae*
- *Shigella flexneri*
- *Staphylococcus aureus* (resistant and nonresistant strains)
- *Staphylococcus epidermidis*
- *Staphylococcus hominis*
- *Streptococcus hemolyticus*
- *Streptococcus mutans*
- *Streptococcus sanguis*
- *Toxoplasma gondii*
- *Trichophyton violaceum*
- *Ureaplasma urealyticum*
- *Vibrio cholerae*

Use to Treat

Viral infections, especially pandemic influenzas and encephalitis, respiratory infections, pneumonia, infections that affect the central nervous system (essentially *any* infection that has accompanying meningitis or encephalitis such as viral encephalitis or meningitis, mycoplasma, Lyme, viral and bacterial CNS infections, and so forth), impaired brain function, fevers, intermittent fevers, GI tract disorders with accompanying inflammation, diarrhea and dysentery, hepatitis, nephritis, urinary tract infections, nervous irritability, epileptic seizures, convulsions, sleep disruptions. You can also use Chinese skullcap as supportive therapy in cancer.

Note: The root tincture of this plant is extremely specific for reducing inflammation in the brain, reducing the cytokine cascades initiated by viral and other microbial agents in the CNS, and alleviating CNS impacts of those microbes. It should be used in any treatment of viral or bacterial CNS infection.

Other Uses

Some cultures use the leaves as a steamed vegetable; the dried leaves are common as a tea.

Finding It

The best root tinctures come from Elk Mountain Herbs (https://elkmountainherbs.com) and Woodland Essence (https://woodlandessence.com). The powdered root can be purchased from 1st Chinese Herbs (https://1stchineseherbs.com). You can get really good seed from Strictly Medicinal Seeds (https://strictlymedicinalseeds.com).

Plant Chemistry

More than 295 different compounds have been found in *S. baicalensis* so far. The six most important are presumed to be baicalein, wogonin, oroxylin A, baicalin, wogonoside, and oroxylin-A 7-O-glucuronide. All are strongly anti-inflammatory, antiviral, and antitumor in action. Some of the other important compounds are considered to be scutellarin, naringenin, apigenin, luteolin, melatonin, and serotonin. All are strongly biologically active. All are synergistic with each other.

Until relatively recently it was supposed that melatonin was not present in plants, only in animals. (Wrong, as such definitive pronouncements often are.) A number of plants are now known to produce melatonin (and serotonin). Chinese skullcap has some of the highest levels (7 mcg/g) so far discovered (some rice family plants have more). Studies have found that the melatonin in plants is strongly present in the plasma of animals that ingest them and that it does bind to melatonin-binding sites in the brain, creating specific effects. (One being helping with sleep cycle normalization.) Melatonin is highly active in the brain; it detoxifies hydroxyl radical, hydrogen peroxide, nitric oxide, peroxynitrite anion, peroxynitrous acid, and hypochlorous acid.

Melatonin is an upstream antioxidant; many of its metabolites, created when our bodies process it or when the plant compound detoxifies oxidants, are also potent antioxidants. It is also synergistic with a number of antioxidant enzymes and other antioxidants such as vitamin C, vitamin E, and glutathione. Melatonin is active at both the micro and macro levels, exerting antioxidative effects at the level of cells, tissues, organs, and organisms. It is a very unique substance, much different than other antioxidants—and not well understood even now. Not only does it work to repair other biomolecules but under in vivo conditions it is four times more potent than vitamin C and E in protecting tissues.

Melatonin is intimately involved in the regulation of people's circadian rhythms, including their healthy sleep cycle. Part of the reason that the sleep cycle is interrupted as people age (and during inflammatory infections in the CNS) is that the oxidative events in the brain are higher and the levels of melatonin (and its regulatory effects) are much lower. Using plants high in melatonin (which is more effective than using melatonin supplements) can normalize the circadian rhythms, including the sleep cycle, and reduce inflammation in the brain and CNS.

The constituents of Chinese skullcap do enter the plasma in substantial amounts. Baicalein is strongly concentrated in the lungs, brain, and hippocampus, wogonin in the liver, kidneys, and lungs. Baicalin concentrates in the brain, specifically in the striatum, thalamus, and hippocampus. Many of their metabolites are strongly present in those locations as well. These all provide potent CNS protection and amelioration of existing infection dynamics.

Traditional Uses

Outside traditional Chinese medicine and American Eclectic practice the uses of the skullcaps around the world are fairly uniform.

The Tibetans, for instance, use five species, including *S. barbata*, but their usages are interesting. Usually the juice of the plant or root is used for wounds, fevers, indigestion, and gastric troubles. Essentially this is showing both antiviral, antibacterial, and fever-lowering actions (and yeah, GI tract actions).

The indigenous tribes of the United States used eight different skullcaps including *S. lateriflora*. Again, the usage range is interesting. It is a bit broader than the Tibetan and includes, for the aerial parts, decoctions and infusions for sore eyes, chills and fever, colds, coughs, heart troubles, and as laxatives; they used root decoction and infusion as emmenagogues and abortifacients, to expel afterbirth, as antidiarrheals, to treat the nerves and breast pain, as kidney medicine, to prevent smallpox, to prevent colds and flu, to keep the throat clean. Again, the range shows antiviral and antibacterial uses, fever-lowering actions, GI tract dynamics, female reproductive tract action, and a tiny bit of nervine action.

What is interesting about this is that the nervine use of the plants among traditional cultures is almost entirely absent. Given the historical American use of the plant, for centuries, is the leaves as a primary nervine agent and that there is pretty much no indigenous use (out of millennia of their contact with the plants) of skullcaps along that line, well, it's intriguing.

The indigenous uses, when examined in depth, do however show a difference in action between plant and root, and the root actions tend to mirror those of the Chinese skullcap.

AYURVEDA

Several skullcaps are listed in my older Ayurvedic herbals but there is nothing on use.

TRADITIONAL CHINESE MEDICINE

Skullcap is one of the 50 fundamental herbs of Chinese medicine and has been in use for over 2,000 years. It is one of the most widely used herbs in Chinese medicine.

It is considered bitter and cold and to dispel heat (fever reducer, anti-inflammatory), to expel damp heat (e.g., lung infections), to be a detoxicant, to stop bleeding, and to prevent abnormal fetal movements (which I think, in part, is an indication of its effectiveness for fetal mycoplasma infections). It is specific for fever, cough, pneumonia, hemoptysis, jaundice, hepatitis, dysentery, diarrhea, bloody stool, vexation, insomnia, headache, enteritis, acute conjunctivitis, uterine bleeding, abnormal fetal movements, hypertension, carbuncle, and furuncle. And, again, there is nothing in traditional use that emphasizes its nervine actions.

Most of the scientific studies on the plant have been conducted in China.

WESTERN BOTANIC PRACTICE

The Europeans tended to use *S. galericulata,* the American Eclectics *S. lateriflora,* and it is probably from the Europeans that the Americans got their usage range for the plant. Both were used similarly primarily as tonic, nervine, and antispasmodic herbs. The Eclectics considered their species specific for chorea (involuntary movements), convulsions, tremors, intermittent fever, neuralgia, to help sleep, and for nervous afflictions such as delirium tremens and hysteria with involuntary muscle movements. It was used in all cases of nervous excitability, restlessness, and wakefulness, especially after acute or chronic illness. It was considered to be a cerebrospinal specific. The usual dose was half an ounce of the recently dried herb in half a pint of boiling water. The herb was considered to lose its effectiveness if kept too long in the dried state.

The English use was similar with the addition of uses for nervous headaches, headaches from coughing, St. Vitus' dance (has *nothing* to do with rock and roll), hiccups, and tertian ague.

This is in fact the usage range still employed by American herbalists and very few of them, unless using the Chinese skullcap, use the root. In current American practice skullcap is considered to be a mild, soothing, and reliable nervine, less stimulating than pasque flower and without the druggy feeling that accompanies valerian if used for sleep.

Scientific Research

There are a lot of studies on Chinese skullcap: in vitro, in vivo, and human and clinical trials and studies. The compounds in the root are, not surprisingly, synergistic with each other. All that have been studied are potently antiviral, anti-inflammatory, antioxidative, and free radical scavenging. Wogonin is the most potent nitric oxide (NO) inhibitor, oroxylin is the most potent in inhibiting lipid peroxidation, baicalein appears to be the most potent antiviral compound. Together they produce effects beyond the individual constituents. (In one study the bacteriostatic effect of the root decoction was compared with that of both baicalin and debaicalin. The root decoction was the strongest.)

The root has strong cytokine impacts, reducing NO, iNOS, IL-3, IL-6, IL-17, COX-2, PGE2, NF-κB, IkappaBalpha (IκBα), IL-1α, IL-2, IL-12, TNF-α, VEGF, TGF, IFN-γ, and tends to upregulate IL-10. It inhibits the production of IgE thus suppressing the expression of histamine. It has especially strong impacts in the spleen. It attenuates the activity of c-Raf-1 kinase, MEK1 and MEK2, ERK-1 and ERK-2, p38 MAPK, and JNK.

IN VITRO STUDIES

Flavones from the root are strongly neuroprotective. Baicalein strongly inhibits the aggregation of neuronal amyloidogenic proteins and induces the dissolution of amyloid deposits. Wogonin stimulates brain tissue regeneration, including the differentiation of neuronal precursor cells. Baicalin promotes neuronal differentiation of neural stem/progenitor cells by modulating p-STAT3 and bHLH (basic helix-loop-helix) protein expression.

Wagonin is neuroprotective against cerebral ischemic insult, and at tiny micromolar concentrations completely suppresses the activity of NF-κB, and inhibits the migration of microglial cells to ischemic lesions, thus reducing inflammation at the site of injury. It inhibits the movement of the cells in response to the chemokine MCP-1.

Baicalein attenuates the induced-cell death of brain microglia in mouse microglial cells and rat primary microglia cultures by strongly inhibiting NO through the suppression of iNOS. The compound inhibits NF-κB activity in the cells as well.

Four compounds in the root inhibit prostate cancer cell proliferation.

Baicalin suppresses IL-1ß-induced RANKL (receptor activator of NF-κB ligand) and COX-2 production at a concentration of 0.01 mcg/ml. The longer the constituent is applied, the stronger the effect. Used on human periodontal ligament cells it shows highly protective effects.

Baicalein inhibits IL-1ß- and TNF-α-induced inflammatory cytokine production from human mast cells via regulation of the NF-κB

pathway. It inhibits NF-κB and IκBα phosphorylation.

Baicalin promotes repair of DNA single-strand breakage caused by hydrogen peroxide in cultured fibroblasts.

The plant inhibits aromatase, thus reducing the conversion of androgens into estrogens.

There are scores of other in vitro studies such as these, all showing potent anti-inflammatory and cytokine-modulating actions of the herb and its constituents.

IN VIVO STUDIES

In rats, oroxylin A markedly enhances cognitive and mnestic function in animal models of aging brains and neurodegeneration. Baicalein is anticonvulsive, anxiolytic, and sedative in rats.

Flavonoids from the stems and leaves of *Scutellaria baicalensis* improve memory dysfunction and reduce neuronal damage and levels of abnormal free radicals induced by permanent cerebral ischemia in rats. Other studies have found that the compounds can enhance and improve learning and memory abilities and reduce neuronal pathological alterations induced by a variety of chemicals in mice.

S. baicalensis reduces symptoms associated with chronic cerebral hypoperfusion (and chronic lipopolysaccharide infusion), including spatial memory impairments, hippocampal MAPK signaling, and microglial activation.

Baicalein protects mice hippocampal neuronal cells against damage caused by thapsigargin (TG) and brefeldin A (BFA). The constituent reduces TG- and BFA-induced apoptosis of hippocampal cells, reduces the induced expression of endoplasmic reticulum stress-associated proteins, and strongly reduces the levels of MAP kinases such as p38, JNK, and ERK. It reduces ROS accumulation and levels of MMPs. It strongly protects the mitochondria from oxidative damage.

A number of in vivo studies have found baicalein to reduce both edema and intracranial hypertension during brain infection due to pertussis bacteria. It also inhibits the neurotoxic action of kainic acid in the rat brain.

Scutellaria baicalensis (in combination with bupleurum) is strongly neuroprotective against iron-reduced neurodegeneration in the nigrostriatal dopaminergic system in rat brains, showing it to be useful for treating CNS neurodegeneration.

When mice, subjected to transient global brain ischemia for 20 minutes, are treated with baicalein (200 mg/kg once daily), neuronal damage is minimal compared to controls, and MMP-9 activity in the hippocampus is inhibited. Pretreatment with baicalein prevents the damage.

Wogonin is also strongly protective in the brain. In rats damaged by either four-vessel occlusion or excitotoxic injury (systemic kainate injection), wogonin confers protection by attenuating the death of hippocampal neurons. It inhibits the inflammatory activation of the microglia by inhibiting iNOS, TNF-α, NO, IL-1ß, and NF-κB. In vitro studies have found that lipopolysaccharide-activated macrophages are protected similarly.

An ethanol extract of *S. baicalensis* has been shown to prevent oxidative damage and neuroinflammation and memory impairments in artificial senescence mice (mice that get old very fast artificially—to study aging). The hippocampus and the mitochondria are strongly protected and neuroinflammation sharply reduced. Expressions of COX-2, iNOS, NO, PGE2, Bax, cleaved

caspase-3 protein are all reduced. Bcl-2 was increased. The effects are dose dependent and are most effective at 100 mg/kg (that would be 7 grams for a 150-pound person, just in the dosage range usually used in China).

Baicalin reduces the severity of relapsing-remitting experimental autoimmune encephalomyelitis induced by proteolipid protein in a mouse model of multiple sclerosis. All the histopathological findings decrease in the mice given the extract.

Baicalin has a protective effect against induced encephaledema in neonatal rats; glutamate and glutamic acid levels decrease, reducing excitotoxicity, and GABA (gamma-aminobutyric acid) increases.

Baicalin, administered to mice infected with influenza virus, increases survival time, eliminates the virus from the lungs, reduces hemagglutination titer and infectivity in the lungs, and reverses pneumonic pathological changes.

Baicalin protects rat brains from pertussis bacilli-induced brain edema. It is 20 times more potent than deferoxamine in reducing lipid peroxidation, and is markedly better at reducing edema, chelating iron, and activating SOD.

Baicalin reduces intracranial hypertension from pertussis bacilli in rabbit brains better than tetramethylpyrazine. The pathologic alterations in the brain are significantly reduced by the use of baicalin.

Baicalin, given to pregnant rats, increases the lung surfactant phospholipids in the fetus and accelerates fetal lung maturation.

Baicalein has been found to be antidepressant in animal models of depression. It reverses the reduction of extracellular ERK phosphorylation and the level of BDNF (brain-derived neurotrophic factor) expression in the hippocampus of CMS (chronic mild stress) model rats.

Oral administration of baicalein in mice infected with Sendai virus results in a significant reduction of viral titers in the lungs and a reduction in the death rate.

Oral administration of baicalein in mice infected with influenza A virus shows significant effects in preventing death, increasing life span, inhibiting lung consolidation, and reducing lung virus titer in a dose-dependent manner. Amounts as low as 1.2 mcg/ml of baicalin (the metabolite of baicalein) result in significant inhibition of the virus. (Note: Plasma levels of baicalin from the ingestion of skullcap root are significantly higher than this after dosing with 3–9 grams per day.)

Baicalein is highly synergistic with ribavarin against H1N1 influenza. The combination produces much better outcomes in mice infected with influenza A infected than ribavirin alone.

Baicalein and wogonin inhibit irradiation-induced skin damage by suppressing increases in MMP-9 and VEGF through the suppression of COX-2 and NF-κB.

In mice infected with hepatitis C virus and treated with *S. baicalensis*, the serum virus content of the mice decreases after treatment with the herb.

S. baicalensis treatment inhibits passive cutaneous anaphylaxis and reduces histamine release in rats receiving intradermal injections of anti-DNP (dinitrophenol) IgE. It is also effective in reducing IL-6 and TNF-α in mouse models of pelvic inflammatory disease. The herb is both anti-inflammatory and antinociceptive.

S. baicalensis extract stimulates the formation of red blood cells and their precursors under conditions of cyclostatic myelosuppression and sleep deprivation.

There are many more studies than these; this just gives a very good overview of the range of actions of the plant and its constituents.

HUMAN STUDIES

The root decoction has been used in a number of clinical situations in China to effectively treat scarlet fever, chronic bronchitis, and epidemic cerebrospinal meningitis. (Details are unfortunately sketchy.) The herb is almost always used in combination, so individual studies are few. But there are some here and there:

Sixty-three people with bacterial meningitis were split into two groups; 32 were treated with both an antibiotic and baicalin, 31 were treated with an antibiotic alone. The inflammatory cytokines in the CNS were markedly lower in the baicalin group, mortality was significantly reduced, and the symptom picture was markedly improved.

Sixty patients with pulmonary infection were treated with either piperacillin sodium or injection of *Scutellaria baicalensis*. Before treatment there was no difference in clinical data. Treatment outcomes were similar in both groups.

In 63 children with upper respiratory infections (51 upper acute, 11 acute bronchitis, 1 tonsillitis) 51 benefited from using the decoction of the root; temperature normalized in 3 days.

A decoction of *S. barbata* was used with 14 women with metastatic breast cancer in a trial at the Memorial Cancer Institute (Hollywood, Florida), as supportive therapy to normal chemo and radiation. The study authors commented that the herb was safe, well tolerated, and showed promising clinical evidence of anticancer activity.

A 12-week randomized trial of *Scutellaria baicalensis* and *Acacia catechu* in Alabama in the dietary management of knee osteoarthritis found that the placebo group had a much higher incidence of respiratory infections than the herbal group. (No mention is made in the study abstract of the herbal compound's effects on osteoarthritis.) However, another study in Arizona, in a randomized, short-term, double-blind event, found that the same mixture (code-named flavocoxid) was as effective as naproxen in controlling signs and symptoms of osteoarthritis of the knee. There was, again, a higher incidence of other effects in the nonherbal group, including more edema and musculoskeletal discomfort.

Russian studies with the root found that it increased the relative number of T lymphocytes in lung cancer patients receiving antineoplastic chemotherapy. Another Russian study with 88 lung cancer patients found that ingestion of a powdered extract of *S. baicalensis* root was accompanied by increased hemopoesis and an increase in immune markers.

There have been a number of combination therapies using *S. baicalensis* in China in the treatment of minimal brain dysfunction, bacillary dysentery, eye infections, and leptospirosis. All showed good outcomes.

Baicalin has been used effectively in treating meningitis, infectious hepatitis, hepatitis B, and acute biliary tract infections. In one study, baicalin was used in the treatment of bacterial meningitis. Sixty-two people with the condition were separated into two groups; one received an antibiotic, the other baicalin. The levels of TNF-α, NO, IL-1 in both plasma and cerebrospinal fluid were monitored. The cytokines were significantly lower in the baicalin group, and mortality was significantly reduced as well.

S. baicalensis has also been found to ameliorate irinotecan-induced gastrointestinal toxicity in cancer patients.

The tincture of *S. baicalensis* was used effectively in treating 51 cases of hypertension. Blood pressure levels dropped with accompanying symptom improvement.

Elder

Family: Caprifoliaceae or maybe Adoxaceae, the literature isn't clear.

Species used: There are five or maybe 30 species of elders. Taxonomists are not sure . . . again. They used to be in the Caprifoliaceae or honeysuckle family (the taxonomists, not the elders) but DNA scientists got involved again and decided that, no, *this* is not a honeysuckle, as anyone looking at a taxonomist can plainly see. It is an Adoxaceae. The suffix *-aceae*, by the way, indicates the members of a plant family, and the prefix *adox-* is the descriptive of that family. *Adox* comes from the ancient Greek and means "not according to right reason, absurd, opposed to common sense." The taxonomists are, however, pretty sure that the elders are in the genus *Sambucus*.

Roughly, there are two forms of elder, the red and the blue. (The red are smaller, conservative, and somewhat toxic, the blue are larger, more progressive, and people friendly.) Some people (i.e., taxonomists) say, inevitably, that there are really *only* two species of elders: *Sambucus nigra* (the blue/black berry group) and *Sambucus racemosa* (the red berry group). So, *Sambucus canadensis* is not really *Sambucus canadensis* anymore but *Sambucus nigra* ssp. *canadensis*. Because of the (reputed) higher toxicity of the *S. racemosa* group, the blue species are the ones usually used for medicine. (Though, to be clear, indigenous peoples use every species irrespective of berry color for both medicine and food. And the medicinal uses are very similar, irrespective of color. Please see the "Preparation and Dosage" section on page 223 for more on this.)

And just to make it more complicated, there are two species that have white berries (red, white, *and* blue—they are a patriotic genus): *S. australasica* (okay, okay, the berries on this one *are* kind of yellow, hence the name *yellow* elderberry) and *S. gaudichaudiana*, the Australian white elder.

The most commonly used medicinal species is *Sambucus nigra*, which grows throughout North America, Europe (into the Scandinavian countries), western Asia, northern Africa, New Zealand, Australia, many Pacific islands, and so on. It's an invasive, making it dear to my heart.

Even though the other blue-fruited species are not quite as widely established, being, I guess, less invasive, most, if not all, of them can be used as medicine: *S. australis* (southern elder, found in South America and Australasia), *S. canadensis* (American elder, found in eastern North and Central America to Panama), *S. cerulea* (blue elderberry, found in western North America), *S. ebulis* (European dwarf elder, found in central and southern Europe, northwest Africa, southwest Asia), *S. javanica* (Chinese elder, found in southeast Asia, Malaysia, the Philippines), *S. lanceolata* (Madeira elder, found on Madeira island), *S. melanocarpa* (found from the western United States into Canada), *S. mexicana* (Mexican elder, found in the Sonoran desert), *S. neomexicana* (New Mexico elder, found from the western United States up into Canada), *S. palmensis* (Canary Island elder), *S. peruviana* (Peruvian elder, found in South America), *S. simpsonii* (Florida elder, found in the southeastern United States), *S. velutina* (velvet elder, found in southwestern North America). The only blue/black berry species I know personally are *S. nigra, S. cerulea, S. mexicana,* and *S. canadensis*. I've used them all for medicine at one time or another. They seem pretty interchangeable to me.

No matter where you live, you will probably be able to find an elder someplace near that you can use as medicine. However . . . I will talk mostly about *Sambucus nigra* with a bit on the others here and there.

Synonyms: The genus and its various species are in flux due to taxonomitis and synonym accusations are flying everywhere. For sure, *S. cerulea* and *S. caerula* are probably the same plant, but then again, maybe they are really *S. mexicana,* or maybe *S. nigra,* or something.

Common names: Legion. Everyplace the plant grows, it has a local name. The plants have been used in food, medicine, and crafts since people have been. In the West, elder—reputedly from the fact that if you use the plant as medicine you will become one. Some say that the plant itself is an elder to the plant communities around it, a gateway to the depths of the plant world.

Parts Used

Most commonly the berries and flowers but the leaves, bark, and root all have a long tradition of medicinal use. You will often see warnings not to use the inner bark, leaves, or root of this plant but that is a rather recent

phenomenon (since 1910 or so). Historically, they have all been used with good effect. (The secret is in the preparation.)

This particular genus, like some others such as comfrey, is suffering from a certain, unfortunately common, form of phytohysteria known as "this-plant-will-poison-you-because-someone-got-sick-from-it-once." So, *don't use herbs!* Most of us who are members of the neo-herbal renaissance have suffered its effects in one form or another. Nevertheless, I have used most parts of this plant for medicine and found them all beneficial. It just depends on what you are needing, why, how you prepare it, and the dose.

More on this next. Kind of a rant actually.

Preparation and Dosage

Most people these days are working with the berries only (a very few American herbalists use the flowers medicinally, but not normally as a primary treatment approach). Usually what is used, especially in Germany—and probably because of German approaches everyplace else—is a standardized liquid extract (or standardized lozenge) or some other variation of the berry juice: expressed juice, syrups, a tea, or a juice decoction. Dosage usually being a cup of the tea or a glass of the juice or a couple tablespoons of the syrup for influenzal infections for reducing fever. I don't agree with this limitation on the medicinal use of the plant but then I tend to be grumpy.

Rant: In reading articles about elder it is common to continually be exposed to the phytohysterical pronouncement that the plant is poisonous. Well, it is not. The various parts of the plant are emetic (and purgative if you take enough) *if used fresh*. That simply means that you will feel nauseous and possibly vomit if you take too much. The flowers are the least likely to cause any nausea or vomiting and thus are considered safe by most phytohysterians everywhere. The berries may cause some degree of vomiting and nausea if you take too much at once (see the "Side Effects and Contraindications" section on page 229 for an amusing anecdote) or if you are especially susceptible to the compounds in the plant. But they are fairly safe in that respect, so the hysteria alert level is only Orange with the berries. The rest of the plant, however, is in the Red alert level range and from reading about it, I am pretty sure the plant could kill off most of the Western hemisphere with just a few drops of the leaf tincture.

Here is a sampling. This first one is from the American Botanical Council and runs mild on the Panic Index:

> Improperly prepared elder preparations can induce toxic effects in humans through poisonous alkaloid and cyanogenic glycosides that are found in the roots, stems, leaves, bark, and unripe berries. Effects of cyanide, also known as hydrocyanic acid (NCN [*sic*, this should be HCN]), on humans include nausea, vomiting and diarrhea, as well as central nervous system and respiratory depression, and general lethargy.[4]

Here's one, however, that tips the needle into the Red, and it is not uncommon: "One word of caution: don't eat raw elderberries. They are poisonous! In the raw form, they produce cyanide."[5] I can just hear the exclamation points on that last word, can't you? There's about eight of them! Then there is this one: "Elderberries must never be eaten raw. All parts of the plant contain the toxin hydrocyanic acid which is destroyed by cooking."[6] (That last bit is the important part and I will get back to it in a minute.)

The hysteria about the red berry varieties is even more pronounced because the HCN in them is in higher quantities. Nevertheless, they can be used as well . . . if you treat them just as the black berry varieties, discussed below, are. Essentially you *heat* them.

The cyanogenic compounds in elder, which are also strongly present in cherries and apples, for example, *can* poison you . . . if you take them as isolated compounds. But the "poisoning" they are talking about here merely consists of nausea, weakness, dizziness, and vomiting—the usual things that happen when you eat something that disagrees with you. The plant is *not* a poisonous plant the way hemlock is (see Socrates for more on this); it's an emetic (vomit) and in large doses a purgative (poop) and the word "poisonous" really should not be used to describe it.

The plant uses these compounds to protect itself from predators, especially vegetarians. When plant-eating animals forage too much from elder, they get dizzy and weak and if that does not stop them and they keep at it, they eventually wander away to vomit. At that point, they pretty much forget about eating altogether, which is the point. If the plant were poisonous, it would just kill the animals, but it doesn't. In fact, it likes to be foraged a bit; it helps the growth and health of the plant (and the health of the animal). However, the compounds in the plant that cause vomiting occur in just the right amounts to stop

foraging when the limit of the plant's tolerance is reached. The cyanide compounds in plants such as elder are normally held separately in different parts of the plant. When the animal chews the leaves, the crushing of the leaves, bark, and so on frees the compounds and combines them, making, in the case of cherry, cyanide gas. Both this and HCN slow (or even paralyze) respiration by inhibiting an enzyme in the mitochondria of cells, cytochrome c oxidase. This is what makes the eater dizzy and a bit breathless. It is also why cherry bark is such a good herb for coughs, or why it has antitussive actions (this is basically what *antitussive* means). In essence, it paralyzes the lungs, which is how it stops hacking coughs. If understood properly, elder can also be used as a potent antitussive herb for unremitting coughs.

Raw kidney beans (and a few other beans) are also considered poisonous unless they are cooked sufficiently by briskly boiling for at least 10 minutes—after having been soaked for 5 hours. (Slow cookers that never reach high temperatures can increase the toxicity fivefold.) But you never see the same kind of phytohysteria about kidney beans as you do with elder. People are simply told to cook them sufficiently to avoid the problem. So, let's stop it all right here and begin talking about what is true for this plant.

Boiling the plant (that is, the leaves, berries, bark, or root), beginning with cold water and raising the heat, for 30 minutes will reduce the cyanide (or HCN) content to nearly nothing. (With cassava, for instance, the fresh leaves contain 68.6 mg/kg of HCN. Boiling them, beginning with cold water, for 30 minutes reduces this to 1.2 mg/kg, making them safe for use. If you start with hot water, the reduction is only to 37 mg/kg.) The longer the boil, the lower the cyanide compound content.

This is why, in the Asian traditions, they use the stems, leaves, and roots (of the *red* species no less) with impunity. To treat broken bones ½ to 1 ounce of the leaves is boiled in 3 cups of water until reduced to 1 cup, which is then consumed. And this is continued for 2 weeks. The root is used similarly for arthritic conditions. The boil time on this is long, much longer than 30 minutes. They are producing what is called a concentrated decoction.

The many chemical compounds contained in the plant are much stronger in the leaves, stems, and roots and by this I am talking about not just the HCN content but the antiviral compounds, the antibacterial

compounds, the anti-inflammatory compounds, and so on. The best medicines are going to come from a much more sophisticated preparation process of the plant than any I have so far read of. To elucidate . . .

The leaves, like peach leaves, are a very reliable nervine. That is, they relax the nervous system. That is why the herb was used for epileptic fits and various dementias and uncontrollable movements by both European and American herbalists for centuries. Dosage of the fresh leaf tincture runs from 5 to 10 drops, taken no more than each hour (though some people can take much higher doses—in fact up to 1 teaspoonful every hour).

Because the fresh stem, leaf, and root can cause nausea, they also cause sweating. This helps lower fevers and is very useful during viral infections. A tincture of the stem can be used to initiate sweating if you take just enough to cause that and not enough to start vomiting. This varies for each person but in general, the dosage range is similar to that of the fresh leaf tincture. (I haven't yet worked with the root and so can't comment on it. However, the Asians use it, as a concentrated decoction, for arthritic inflammation and, given its constituents, it would be very good for that.)

The flowers are best if they are prepared as a hot infusion, covered. That is, put 1 ounce of the flowers, dried or fresh, in a quart of hot water, cover, and let sit until cool. This retains the essential oil compounds of the flowers in the liquid and they have unique antiviral qualities themselves. You can drink as much as you wish of it.

The berries are fine, but the seeds possess HCN and this is what makes some people vomit. So nearly all sources recommend cooking them first. See the facing page for a very good recipe.

Eating Fresh Berries

Because the fruits contain potently active antioxidants, if you juice the berries in the presence of oxygen (essentially in your kitchen or herb room) many of the constituents will combine with oxygen as the cell walls are broken, reducing the potency of the compounds. Eating the berries fresh eliminates this problem.

Elderberry Syrup for Colds and Flu

INGREDIENTS

1 cup dried elderberries (or 2 cups fresh)
2 quarts water
20 cups sugar (yes, yes, I know)

To make:

If you are using dried elderberries, soak them overnight in the refrigerator in the water. In the morning, put the pot on the stove and bring the contents to a boil. Once boiling, reduce the heat to a simmer, and cook until the liquid is reduced by half. (Hours.) Do *not* skim the surface of the liquid; this keeps the resins in the syrup, which you want to do. When the liquid is reduced by half, remove from the heat, let cool, then strain through a wire strainer, mashing the remaining liquid out of the berries with a strong spoon. Throw the berries away, return the liquid to the pot, set it over medium heat, and add the sugar, stirring until it is completely dissolved. Let cool.

The amount of sugar in the syrup will stop it from spoiling. Bacteria cannot live in high-sugar-content solutions such as honey. If you don't want this much sugar, you can use half as much but you should add 20 percent alcohol to keep the mix from going bad.

To use:

Dosage for adults is 2–4 tablespoons every 2–4 hours during the early stages of a cold or flu infection. This is what is called a concentrated decoction and it is pretty good, if used at the first signs of infection.

If the flu gets established, use this syrup along with the influenza protocol in Chapter 2 or, at the very least, the fresh ginger juice tea on page 39. If you'd prefer an elderberry-only treatment, you can add the syrup to 8 ounces of an infusion of the leaves and drink every 3 hours or so. I would also use 8 ounces of the flower infusion and 5–10 drops of the leaf tincture on the same time schedule.

If you are using the fresh juice, which many do, it will be less strong than the syrup, so you will need more. Just test it to make sure you are not easily nauseated.

It is possible, with practice, to create a mixture based on this type of formulation that would be a powerful treatment for respiratory viral infections, one much more sophisticated than just the syrup. It would be strongly antiviral, strongly anti-inflammatory, and potently analgesic. See, for example, the formulation on page 228.

Antiviral Elder Recipe

INGREDIENTS:

- 1 cup dried elder leaves
- ½ cup dried elder stems
- 2½ quarts water
- 1 ounce elderberry syrup (see recipe on page 227)
- 1 ounce fresh elder leaf tincture
- 1 ounce elder stem bark tincture

To make:

Powder the dried leaves and stems as finely as possible in a blender or grinder. Place in a pot with the water and bring to a boil. Reduce the heat to a simmer and cook until the liquid is reduced by two-thirds. Remove from the heat and let cool. Press the decoction through a cloth to remove the plant matter. Add the elderberry syrup and tinctures to the liquid and stir well.

To preserve this, you may need to add sugar to the stem/leaf decoction after you press it. If so, reheat, add enough sugar to bring the sugar content up to 65 percent or so, and let cool before adding the rest of the ingredients. However, you can also refrigerate it, or add enough alcohol to bring the alcohol content up to 20 percent, in order to preserve it.

To use:

Take 2–4 tablespoons every 2–4 hours, less if you feel nauseous. Start slow and work up.

Caution

I have seen people eat handfuls of ripe elderberries or drink copious amounts of the fresh-juiced berries without nausea *and* I have seen one person eat a small handful of ripe elderberries or drink a small amount of the juice and vomit explosively. *There seems to be a wide range of sensitivity to the HCN in the plant.* And there is HCN in the fresh tinctures that has not been removed by heating. If you are going to use the antiviral elder recipe above—or any of the fresh leaf and stem tinctures—I recommend that you test your sensitivity by beginning with small doses and working up until you find your nausea level. The majority of people appear able to take the herb with impunity.

Also note that the fresh juice of the leaves, the stems, and the roots are potently emetic. If you need to vomit, for whatever reason, they can be used in small doses for this purpose.

Plants harvested near roadways and industrial sites have been found to have much higher levels of heavy metals than those in other locations. Careful where you harvest.

Side Effects and Contraindications

Sometimes . . . diarrhea, nausea, vomiting, depending on the dose, what part of the plant you are using, how it is prepared, and your individual biological response to the medicinal. There are few reports of side effects from these plants except for that.

From individual reports, *S. mexicana* berry appears to be a bit more nausea-inducing than the other varieties of blue/black berry species. There is one report of a group of people drinking juice pressed from "the berries, leaves, and stems" (Juice from the leaves and stems? Are these crack babies?) and 11 of them, within 15 minutes, experienced weakness, abdominal cramps, nausea, and vomiting. Eight of them were taken (by helicopter for god's sake!) to the hospital, where the physicians remarked on the dangerousness of self-medicating and the natural world in general (especially the intersection of the two). "All recovered quickly." Well, I guess so. Look, it will just make you vomit and only then if: 1) you take too much, or 2) you have an individual reaction to the plant, or 3) you take too much of the fresh or raw leaves, stem, root, or, sometimes, the uncooked berries. Once the stuff is out of your system, that's it. No more trouble.

Because the individual response to the herb varies so widely, you should start with low doses and work up. Some people can take large amounts—that is, handfuls of raw (or dry) berries all day long, or large doses of the leaf tincture, while with others, 10 ripe, raw or dried, berries will cause nearly immediate vomiting.

Herb/Drug and Herb/Herb Interactions

None have been noted but speculation abounds that elder may exert additive actions when combined with laxatives or diuretics or decongestants or various jams and jellies (producing sugar overload, just an FYI on that one). A couple of reports say that, in rats, the herb interferes with the impacts of phenobarbital and morphine, reducing their effects.

Alternatives: Poke root, *Phytolacca americana*, has a number of similarities to elder including its medicinal actions and the hysteria

about being poisonous. The plant (all parts: leaves, roots, berries) contains a tremendously potent antiviral compound, pokeweed antiviral protein (PAP), that is broad-spectrum against a wide range of viruses (and other antiviral compounds as well). Used in its purified form it has inactivated the HIV virus in mice, making them HIV free. The poke plant itself could very well be a potent broad-spectrum antiviral and it should be examined in some depth for this use. As well, the root is a very strong lymph system herb, one of the few that I know of besides red root, so it also helps clear the lymph system of viral and bacterial debris and potentiates the actions of the nodes, spleen, and so on. I suspect that poke, as a medicinal plant, can be prepared identically to elder in order to use the plant as a reliable antiviral. Because the plant also has major impacts on the spleen and lymph system, that would make it a primary plant to use for viral encephalitis.

Habitat and Appearance

Most people describing elder say it is a largish shrub, occasionally a small tree. But all the elders I have known look like trees to me and pretty much to everyone I know who has seen a live one. (Well, okay, *S. canadensis* is more bush-like; it does stay pretty small. And, well, *S. neomexicana* is pretty bush-like, too, I guess.) I have seen *S. cerulea* up to 40 feet tall and sources say others in this genus can reach 50 feet. I just can't get the descriptive "small" from that. But I guess when they were young . . .

The branches of the tree/bushes have leaves that are pinnate, meaning for every leaf on one side of the stem, there is another opposite, and in spite of the fact that everyone I know calls these leaves they are really leaflets, I guess; the whole stalk that has leaves on it is a pinnate leaf, or something. Anyway, once seen never forgotten. The leaflets usually are a bit serrated along the edge. However . . . one species, black lace elder, a developed species, has leaves a bit more "lacelike." It's an ornamental, if you like that sort of thing, but not really a member of the medicinal group, historically speaking. I don't know if it is usable medicinally.

The flowers form an umbel (flattish umbrella-like cluster), often the size of an adult hand or larger. They produce black or blue or purplish or black/blue/purplish berries when ripe (except for the red/white/yellow berry species, which I am not going to go into here). In some species, the

flowers are usually white (occasionally pink) and possess a marvelous smell. All the ones I use usually smell heavenly. But in some species they reputedly smell fetid. The flowers are upright on the stems in very prolific clusters. The berries, when ripe, are heavy and hang down from the weight.

The bark is very rough and corrugated when older. I've never harvested the root, so don't know what it looks like (yet). It is reported to be a potent emetic, so, if you need to vomit . . . and we all need to sometime or another . . . that's the ticket.

S. nigra grows in pretty much any kind of terrain: from floodplains to forest gaps to suburbs to industrial wasteland. The only limit seems to be highly shaded areas; it needs some sun to be happy. I have seen various *Sambucus* species growing from sea level to over 9,000 feet in altitude, in wet locations, in dry locations, in hot, rarely cold climates, in extremely cold climates (in Canada, for example), in wilderness, and in cities. It's a great invasive medicinal.

Cultivation and Collection

Richo Cech, of Strictly Medicinal Seeds, my go-to guy for reliable information on growing medicinals, recommends that you take the dried berries, soak them overnight, "smash them, and remove the seeds. Sow in outdoor conditions, in pots or flats, and expect germination in the spring. . . . The best conditions for germination are cool, moist shade. . . . Elderberries will not grow properly in sterile soil. Sow seeds in very rich and composty soil medium. . . . Once germinated, the seedling grows very rapidly into a handsome bush or small tree. Grow out in a shaded place in pots for a year before transplanting to final location." The flowers appear in about 3 years and are normally pollinated by beetles and flies of various sorts with a few bees thrown in for good luck.

The flowers are collected in full bloom (June/July), the berries when ripe (August/September), and either dried out of the sun and stored in plastic or glass containers in coolish, dark locations or used fresh in tinctures, cordials, cough syrups, and so on. The leaves can be picked at any time and tinctured fresh or dried for use later, with the same storage conditions. The inner bark is treated similarly.

Properties of Elder

Actions

In my opinion this herb is a narrow-spectrum antiviral but a fairly good one in its range. (I am starting to suspect its range is much greater than any of us realize, however.) Currently, I think it the least strong of the herbs listed in this section but I suspect that is because of the failure to commonly use the leaves and bark as antivirals. The flowers and berries are most commonly used and they appear to be the weakest parts of the plant. Nevertheless, the berries do possess some good activity against, primarily, influenza viruses (and some other enveloped viruses, especially respiratory) *and* the plant is a moderate invasive and broadly available throughout the world. If you have nothing else handy for a severe respiratory infection, use it. For some people it works very well, especially if you use the herb *right at the beginning of an influenza infection*, just when that tingle in the bones begins.

As an antiviral, elder inhibits viral replication, inhibits neuraminidase, reduces hemagglutination, binds influenza viruses thus inhibiting them from infecting host cells, contains nontoxic type 2 ribosome-inactivating proteins, is directly virucidal, inhibits maturation of viruses, is a depurinating agent—with depurinating activity against both viral nucleic acids and infected host cell ribosomes—and protects against viral infection if taken prophylactically.

It is also antibacterial (directly and through anti-quorum-sensing activity), antifungal, analgesic, anti-inflammatory, antinociceptive, anticancer, anti-angiogenic, antiteratogenic, diaphoretic, diuretic, prostaglandin synthesis inhibitor, antipyretic, antioxidant (the berries have more antioxidant strength than vitamins C and E), moderate immune stimulant.

Active Against

As an antiviral, elder is primarily active against enveloped viruses (see the "Plant Chemistry" section, page 235, for more). The flowers, berries, leaves, and bark all have a range of activity against microbial pathogens; however, the berries have been the most exhaustively tested. Still, in spite of the hysteria about using the bark and leaves, they are also potently antimicrobial though more needs to be done in looking at just *what* range of microbes they are active against and *how* they are to be prepared for use. I suspect the antimicrobial actions are consistent among the different parts of the plant but no one has really explored that to any extent. However, a look at the plant chemistry reveals that all parts of the plant contain compounds

specifically active against enveloped viruses; these are not just confined to the berries and flowers. And *the compounds are much stronger in the leaves, bark, and roots.* Given the historical importance of the plant and its common use as a medicinal throughout the world, there is just too little research on it. This section contains *only* those microorganisms that some part of the elder plant has been tested against:

Elderberries: influenza A (H1N1—various strains; H5N1—KAN-1; H3N2—various strains), influenza B (three different strains), animal influenza (three different turkey and swine strains), HIV (four serotypes), feline immunodeficiency virus (FIV), herpes simplex viruses, tobacco mosaic virus, various mycoviruses, *Haemophilus influenzae*, *Staphylococcus aureus* (resistant and nonresistant), *Streptococcus pyogenes*, group C and G streptococci, *Branhamella catarrhalis*, *Helicobacter pylori*, *Bacillus cereus*, *Pseudomonas aeruginosa*, *E. coli*, *Salmonella poona*, *Shigella* spp., *Mycobacterium phlei*

Elder flowers: influenza A and B, *Staphylococcus aureus* (resistant and nonresistant), *Bacillus cereus*, *Salmonella poona*, *Pseudomonas aeruginosa*, *Mycobacterum phlei*

Elder leaves: tobacco mosaic virus, lymphocytic choriomeningitis virus, Columbia SK virus, *Bacillus cereus*, *Serratia marcescens*, *E. coli*, *Epidermophyton floccosum*, *Microsporum canis*, *Microsporum gypseum*, *Trichophyton mentagrophytes*, *Trichophyton rubrum*

Elder stem bark: respiratory syncytial virus, lymphocytic choriomeningitis virus, Columbia SK virus, *Candida albicans*, *Trichosporon beigelii*, *Malassezia furfur*, tobacco mosaic virus

A combination formula made from *Sambucus nigra*, *Gentiana lutea*, *Primula veris*, *Verbena officinalis*, and *Rumex* spp. (called Sinupret in Europe) was found to be broadly antiviral, showing activity against influenza A, parainfluenza, human rhinovirus B, Coxsackie virus, adenovirus C, and respiratory syncytial virus.

Use to Treat

Influenza and other respiratory infections. Most commonly this usage includes only the berries as a concentrated decoction or syrup (or, more weakly, an infusion of the flowers). However, a concentrated decoction of berries, stem bark, and leaves gives the most potent antiviral combination of all; see page 228.

The leaves, root, and stem bark, decocted, for internal inflammations, broken bones, and arthritic complaints. The leaves, decocted, for liver disease and inflammation. But again, see the "Preparation and Dosage" section on page 223 for more on this.

Continued on next page

Continued from previous page

The leaves and bark for topical fungal infections. All parts topically for herpes sores and skin inflammations.

The leaf tincture in small doses as a nervine relaxant, an internal anti-inflammatory, and an analgesic for pain.

The root tincture if you really, really need to vomit explosively. (Really, it's much better than ipecac.)

A number of cultures use elders (e.g., *Sambucus ebulis*) as a primary, first-use anti-inflammatory, feeling that it is especially useful when compared to pharmaceutical anti-inflammatories. A brief look at the Iranian usage range gives a good overview of the plant's capacities.

In Iran the root is particularly valued as an anti-inflammatory (the leaves as well) and is used topically to reduce swellings from bites, stings, infected wounds. There is also judicious use of the root and leaf internally as non-steroidal anti-inflammatories for rheumatoid complaints along with the topical use. (Normally they are decocted prior to internal use; see the "Preparation and Dosage" section on page 223.) The leaves are widely used in Iranian herbal practice to reduce swellings in the liver and kidneys, as a diuretic, and as a liver protectant. The undecocted root (tincture or infusion) is commonly used, in tiny doses, as a tea for dropsy (essentially reducing leg edema through diuresis). Larger amounts are used as a purgative, to induce vomiting in case of poisoning or to realign the stomach mucous membranes (similarly to the American physiobotanical uses of lobelia in the nineteenth century).

Other Uses

The flowers as fritters or as adjuncts in wine and beer, the bark for dye, the berries for wine, beer, jellies, jams, and pies (black *and* red varieties), and the odd whistle here and there from the stems.

Finding It

Not hard. Decent tinctures that have moved outside the elderberry box are rare though.

Plant Chemistry

The elders contain several hundred identified compounds including fairly high levels of phosphorus, vitamins A, B_6, and C, and most if not all the amino acids. They are also heavy in polyphenols and anthocyanins. The main anthocyanins are cyanidin 3-glucoside (Cy3G), cyanidin 3-sambubioside, cyanidin 3,5-diglucoside (Cy3,5dG), and cyanidin 3-sambubioside-5-glucoside. These have a wide range of effects; cyanidin-3-glucoside, for example, is a rather potent anticancer agent. They are all highly antioxidant. Fruits of the plants contain from 360 to 1,300 mg/100 g and from 270 to 660 mg/100 g of CY3G and Cy3,5dG, respectively.

As with all plants, there are variations in chemical profiles even among different plants of the same species. Nevertheless, all elders (red and black berry types) contain very similar chemical profiles. All elders are particularly high in compounds with a wide range of antiviral activity. To elucidate further . . .

They contain a unique group of compounds called ribosome-inactivating proteins (RIPs). There are two known types of these proteins, types 1 and 2. Until recently the main ones known were type 2 RIPs such as ricin and abrin. All are extremely toxic. In contrast, those in elder are *not* toxic, putting them in a unique chemical class of their own.

S. nigra bark contains a complex grouping of type 2 RIPs such as nigrin b, basic nigrin b, SNA, SNA 1′, and SNLRP. The berries contain nigrin f and nigrin s. Leaves of the dwarf elder, *S. ebulis*, contain type 2 RIPs ebulin 1 and ebulin r1 and r2 as well as type 1 RIPs called ebulitins. The fruits contain a type 2 RIP called ebulin f. These ebulins are strongly antiviral with low toxicity and represent a novel antiviral mechanism in plants—they essentially use these compounds to protect themselves from plant viruses. As researchers have noted, "Biochemical and molecular studies have shown that the elderberry tree expresses a complex mixture of type-2 RIPs and/or lectins in virtually all tissues . . . [and] the antiviral activity of RIPs against plant viruses is well-documented."[7] But these compounds also have activity against a wider range of viruses such as HIV and influenza.

The plants are also very high in flavonoids that have been found to bind to H1N1 virions, inactivating them. Once the viruses are bound, they can't infect host cells. Elder flavonoids have a very strong affinity

for influenza viruses, somewhat like a magnet and iron filings. The degree of inhibition of these flavonoids is similar to that of Tamiflu.

Specific compounds such as apigenin, beta-sitosterol, betulin, caffeic acid, chlorogenic acid, cyanin, ferulic acid, glycyrrhetic acid (present in some species), isoquercetrin, kaempferol, linoleic acid, linolenic acid, lupeol, malic acid, oleanolic acid, oleic acid, palmitic acid, quercetin, rutin, sambucine, shikimic acid, stigmasterol, tannic acid, tyrosine, undecylenic acid, and ursolic acid are also antiviral, some strongly so.

These compounds are active across a range: herpes simplex viruses, Coxsackie B1 and 3, enterovirus 71, HIV (and SIV), influenza, chikungunya virus, Epstein-Barr, hepatitis B, hepatitis C, hepatitis E, Japanese encephalitis, West Nile encephalitis, vesicular stomatitis virus, poliovirus, adenovirus-3, porcine circovirus, porcine epidemic diarrhea virus, rhinoviruses, Junin virus, cytomegalovirus, respiratory syncytial virus, tobacco mosaic virus, SARS and other coronaviruses, dengue, fowlpox, rotavirus, canine distemper virus, murine norovirus, feline calicivirus, and Ebola.

The compounds in elder are particularly active against enveloped viruses. These include the influenza viruses, herpesviruses, pox viruses (shingles/chicken pox), hepatitis B and D, the flaviviruses (West Nile, dengue, tick-borne encephalitis, yellow fever, Japanese encephalitis, and so on), coronaviruses (upper respiratory and GI tract infections and SARS), paramyxoviruses (mumps, measles, respiratory syncytial virus, parainfluenza), rhabdoviruses (vesicular stomatitis virus), bunyaviruses (hantavirus), filoviruses (Ebola, Marburg), and retroviruses (HIV). Various parts of the plant *have* been tested against some of the viruses in these groups and have been found active against them. Further, historical use in a number of cultures includes some of these disease categories. It seems as if the plant may in fact be a broad-spectrum antiviral for all enveloped viruses.

Many of these compounds act synergistically. The result is a fairly potent grouping of compounds that deeply affect influenzal and other enveloped organisms. As only one example . . .

Palmitic acid is a rather novel CD4 fusion inhibitor that blocks HIV entry and multiplication while a number of other compounds are directly virucidal against the virus and still other compounds such as astragalin stimulate immune function and response. Palmitic acid also

stimulates viral clearing responses against influenza viruses. But it, at the same time, induces a long-term memory (up to several years) in the body's CD8 T cells of that response against influenza viruses *and* inhibits the production of mature viral particles while, again, numerous other compounds in elder are directly virucidal for the organisms, and others inhibit viral binding to host cells, and others stimulate specific immune responses.

The elders also contain cyanogenic glycosides such as sambunigrin and ebuloside—and these compounds are higher in leaves, stems, and roots, respectively, which is why people rather frantically say you should not use those parts of the plant for medicine. These are the compounds that raise the phytohysteria levels among those who understand medicinal plants . . . not.

The particular compounds that cause these effects have very specific medicinal actions as well and very useful ones at that. As usual the dose is the thing (eat 5 pounds of sugar and tell me how you do) *and* the preparation. Again, please see some details on this in the "Preparation and Dosage" section (page 223). There is more to this than meets the stomach lining.

Traditional Uses

This plant has been used by people as medicine since people have been on every continent on which it grows.

The genus name, *Sambucus*, comes from ancient Latin, Greek, and Aramaic roots and is the name of an ancient musical instrument, a.k.a. panpipes, made from the hollow stems of the plant. The plants used to be (or maybe still are) in the Caprifoliaceae family. *Caprifolia* itself is from the ancient Greek as well and means "goat leaf." It was the ancient name for a honeysuckle. But the meaning goes deeper than that as the ancient god of wild woods, fields, and music, Pan, possesses, in part, a goat shape. The elders were felt to be a primary plant of the god Pan. The plant was felt to cure nearly all the ills of humankind (thus allowing one to *become* an elder). That is, it is a *pan*acea.

The word *pan* is also from the ancient Greek, meaning "all, wholly, entire, altogether, by all, of all"—and yes, I have read philological treatises on the topic, so tear thy hair not oh ye reasonless reductionists.

AYURVEDA

Sambucus nigra and *S. ebulis* both exist in Ayurvedic practice but not in any depth. *S. ebulis* has a minor role, the roots used for dropsy.

S. nigra use is more extensive; the flowers, berries, roots, leaves, and stem bark are used, the flowers and leaves used fresh. The inner bark is a hydrogogue, cathartic, and antiepileptic. The flowers are diaphoretic, sudorific, and laxative. The berries increase renal function, the root is an aperient.

TRADITIONAL CHINESE MEDICINE

I don't find anything on this genus in my research library, which is extensive in some respects, though it seems as if there should be something since the plant grows in that region. But, after way too much searching of journals on the internet, I did begin to find some information here and there. Still, it took a bit of serendipity and some cleverness to find it. Despite this, the genus doesn't seem to be considered a major medicinal in any sense of the word. The usage range is odd as well. It doesn't overlap well with the genus use in the Americas or in Europe, in spite of the fact that constituent studies find very similar compounds in the Chinese species.

Three members of the genus, all red berry species, have been used in China for millennia (they just don't seem very excited about it).

Sambucus formosana (one site lists the Chinese name as mao gu xiao) appears to be the least used member of the genus and I can find little on it. It, like *S. chinensis*, has been used to treat liver disease. One site lists this species name as a synonym of *S. chinensis*, another site as a synonym for *S. javanica.* (There are apparently taxonomists *everywhere*.)

I do find a bit more on *Sambucus chinensis* (in traditional Chinese medicine known as lu-ying, in English as Chinese elder) but it did take some searching. (At least one site lists this plant name as a synonym for *S. javanica*.) Syrups of the berries *and* the leaves are used (decoctions again). The herb is considered to be warm and bitter and is used for dispelling blood stasis and dispelling wind rolling. Lu-ying's primary uses seem to be for the treatment of hepatitis and liver injury, for inflammations, and as an analgesic. It is also used to induce sweating, as a diuretic, for bruises, rheumatism, dislocations, nephritis, edema, beriberi, and urticaria.

Assuming this species name *is* a synonym, then the use data expands a bit (but . . . the plant under the name *Sambucus javanica* is listed by a number of sources as *not* growing in China even though it is called Chinese elder—are taxonomists even human? Have they no mercy?). The whole plant would be then anodyne, antiphlogistic, depurative, diuretic, emetic, and purgative. The leaves and root are used for pain and numbness, bone diseases, and rheumatic problems. The fruit is used as a depurative and purgative. A decoction of the berries is used to treat injuries, for skin diseases, and for swellings. A decoction of the leaves is used as a diuretic and an anodyne (pain reliever).

In Indonesia *S. javanica*'s leaves are primarily used for pain relief. One-half to 1 ounce of leaves is boiled in 3 cups of water and reduced to 1 cup, and the decoction consumed for 14 days. (Note: No emetic warnings are shouted.) For beriberi 1 to 2 ounces of fresh root, stems, and leaves is treated similarly, the cooled decoction consumed for 14 days. (Again, no emetic warnings occur.) For jaundice, the roots only are used, as a concentrated decoction. (Another site lists multiple synonyms for this plant, including *Ebulis chinensis*, though, upon examination, that also seems to be the name of an Argentine mussel. So, no, taxonomists have no mercy.)

Sambucus williamsii is also listed as a species long in use in traditional Chinese medicine. On that I do find a bit more usage, over centuries, for inflammation, broken bones, and joint diseases. There are actually some good studies on the actions of the plant leaves in healing broken bones and osteoporosis (see the "Scientific Research" section, page 240). This use for broken bones and bone loss is interesting and potentially opens up a whole new range of action for this genus. *S. javanica* is also used for this; it makes sense to explore it further. The action appears specific to the leaves (concentrated decoction).

Sambucus williamsii is considered anodyne, carminative, diaphoretic, diuretic, and emetic. The leaf is used to treat (and alleviate, i.e., break) ague fits (essentially intermittent fevers with sharp swings into both extremely hot and cold states, i.e., fits). The flowers are diaphoretic and diuretic. The juice of the stem is used as an emetic. A decoction of the root for arthritis inflammations.

Still, there is so little on the genus compared to so many others; it doesn't appear to have a major place in traditional Chinese medicine.

WESTERN BOTANIC PRACTICE

Elders have been used in European medical practice for over 2,500 years for inflammatory conditions, for sore throats, as a purgative, as an emetic, and for wounds. The leaves were a major ingredient in salves for the treatment of wounds, bruises, and sprains. Used internally, they are expectorant, diaphoretic, and diuretic. The berries were used for rheumatism, erysipelas, colic, diarrhea, epilepsy, and dropsy.

The use of the various *Sambucus* species by the indigenous peoples of the Americas was extensive, for both red and blue berry species. *S. canadensis* bark was used as an emetic, laxative, blood purifier, for wounds to prevent infection, for skin inflammations, for jaundice, as a wash for pain, as a poultice for headaches, as a laxative for children, to treat measles, diphtheria, and mumps. The leaves were used in ointments for wounds and burns, and a leaf infusion to wash skin sores to prevent infection, as a diuretic, for dropsy, and for jaundice. The berries for rheumatism, as a wine as a tonic, and for fevers. The flowers to sweat out fevers, for colds and pulmonary troubles, and to treat colic in infants. The root as an emetic, for liver troubles, as a poultice for swollen breasts, and as a poultice for a baby's unhealed navel.

And so on and on and on.

The various species (*S. cerulea, S. mexicana, S. neomexicana, S. nigra, S. velutina,* and so on) were all used pretty similarly. In essence: for fevers and colds, as an emetic, as a purgative, for skin inflammations, for rheumatism (arthritic complaints) and sore joints, to treat wounds, as a diuretic, and for liver troubles.

The American Eclectic botanical physicians had a similar range of use.

There has been very little new exploration of this plant as a primary medicinal by contemporary American herbalists, probably due to phytohysteria contamination.

Scientific Research

There hasn't been nearly enough study on this plant. However, the pharmacokinetics of this plant, at least of the anthocyanins, are good.

Anthocyanins are a group of water-soluble pigment compounds that exist in plants, in all their tissues, but we notice them primarily in the fruits. They create the various colors of the fruits: red, purple, or blue. They are a kind of flavonoid. Closely related compounds called anthoxanthins are what are contained

in the white and yellow berry species of elder. These kinds of compounds have been extensively studied as medicinals and they do have a wide range of actions: anticancer, antiaging, neuroprotective, anti-inflammatory, antioxidant, antibiotic, analgesic, and blood sugar regulating. The anthocyanins in elder are particularly potent and are the compounds in elder that have been most extensively studied.

They reach peak presence in the body within 30 to 60 minutes after ingestion. They are high in the urine and GI tract mucosa, and in the liver and bloodstream to a lesser extent. The half-life is about 2 hours. They are absorbed in the small intestine and can be found in the body (and urine) as intact glycosides, methylated forms, and glucuronidated derivatives.

There have also been some studies of the pharmacokinetics of ursolic acid, which is present in fairly high quantities in the plant. Ursolic acid has anticancer properties, is potently anti-inflammatory through its downregulation of MMP-9 and inhibition of COX-2, stimulates anabolism (increasing muscle mass and decreasing fat accumulation), thus reducing muscle atrophy, and is cardioprotective, analgesic, antibacterial, antiviral, antidiabetic, and antioxidant. Once ingested, it is widely present in blood plasma, reaches peak concentration in 1 hour, and reaches its half-life in 4 hours.

Elder is a potent COX-2 inhibitor and inducer of quinone reductase. (*S. racemosa* inhibits ornithine decarboxylase.) It also tends to act in the body as a cytokine modulator; it increases the body's production of IL-10 if it is taken during an infection, downregulating the levels of other cytokines as necessary, but if taken early in the disease process, it inhibits viral upregulation of IL-10 and increases antiviral cytokine production and activity. One study found that ethanol leaf extracts of *S. ebulis* reduce TNF-α and its associated induction of VCAM-1 (vascular cell adhesion molecule-1). Intracellular adhesion molecule-1 (ICAM-1) levels were also reduced in that study. Elder modulates the production of interferon gamma (stimulating it if necessary, lowering it if it is too high) and hematopoietic growth factor GM-CSF by monocytes and lymphocytes. In vivo studies with mice have found that elderberry extracts enhance the immune system through increased levels of T cells, B cells, interferon, and IL-2. (There are some reports that show that Sambucol, a proprietary elderberry extract, stimulates TNF-α production. This perplexes scientists but in fact the herb tends to modulate cytokine production, raising it if necessary, lowering it if it is too high.)

Normally, when herbs or their compounds are tested by scientists in their labs, they do it in vitro (lab/test tube), in vivo (animal), or with human clinical study or trial. Unknown to most people there is a fourth category, in planta (i.e., in plants). This will, I suspect, be a growing category for research as time goes by. Most of the studies on elder and its constituents have been in vitro, and many of those findings have already been scattered throughout this monograph. Here are a few from the other categories.

IN PLANTA

Elder has been found to be directly virucidal against tobacco leaf virus, to inactivate the virus through depurination, and to be host protective if later challenged by viruses.

IN VIVO

Sambucus ebulis protects mice from the teratogenic effects of albendazole. TheraMax, a proprietary blend of green tea and elderberry (*S. nigra*), is effective against seven of eight strains of influenza A and B (in vitro); when used on mice infected with mortal doses of influenza viruses, it significantly slowed the arrival of death, curtailed weight loss, and improved lung hemorrhage scores. *S. ebulis* leaves were found to possess potent wound healing activity when used as an ointment on mice. *S. williamsii* extracts (part of plant not stated but it was apparently the leaves or stems) exerted protective effects on ovariectomy-induced bone loss in rats. It improved trabecular bone mass and cortical bone strength, decreased urinary calcium excretion, increased serum calcium levels, increased tibial bone mineral density, and exerted beneficial effects on the microarchitecture of the trabecular bone. A methanolic extract of the stems of *S. sieboldiana* (*S. racemosa* subtype) was found to possess antiosteoporotic activity. It inhibits bone resorption in ovariectomized rats.

In a particularly interesting study, mice were given Sambucol, an extract of elderberry. It caused a shift in immune response in the face of microbial challenge. In mice then infected with leishmania parasites, Sambucol delayed the onset of the disease by upregulating Th1 cytokines. However, when given to mice challenged with malarial parasites, the incidence of cerebral malaria increased substantially. In essence it exacerbated the Th1 dominant cytokine cascade during malarial infection, leading to a worsening of symptoms. The berries appear to *possibly* act as a Th1 activator, while the leaves act as a Th1/Th2 modulator. (So, don't take the berry syrup if you have malaria.)

HUMAN STUDY

Sixty patients, aged 18 to 54, suffering from influenza symptoms for 48 hours or less, were enrolled in a randomized, double-blind, placebo-controlled study of the effectiveness of elderberry syrup. Participants received 15 ml (½ ounce) of an elderberry syrup or placebo for 5 days. Symptoms were relieved on average 4 days earlier in the elderberry group.

Sambucol, in a placebo-controlled, double-blind study, was used to treat a group of individuals in Panama during an influenza epidemic. Those using the elderberry extract experienced a significant improvement in symptoms, including fever, in 2 days. A complete cure was recorded for most within 2 to 3 days. Serum examination showed high hemagglutination inhibition titers to influenza B.

Ginger

Ginger is a decent antiviral *only* if you are using the fresh rhizome, not the dried root. Specifically: the juice of the fresh root (though an alcohol tincture of the fresh root *will* work, it's just not as good). In Chinese medicine the dried root and the fresh root are considered different medicines with *very* different actions—because they are.

The plant's constituents alter considerably with drying as many of the volatile oils are lost; other constituents morph as they dry.

Family: Zingiberaceae. There are about 1,400 members of the family, ordered in four subfamilies, five tribes, and 52 genuses, genii, or genera (whatever). The *Zingiber* genus is usually referred to as the *true* gingers and is the one most people have heard of though nearly everyone knows *only* the main culinary variety, *Z. officinale*.

Members of the *Alpinia* genus (whose members are known as the galangals—and I actually know a person with that last name) are probably the other most commonly used medicinals (and culinary additives) in the family. Some of them do have a similar range of actions. (Cardamom and turmeric are both gingers but they belong to other genera—or perhaps genuses, maybe genii.)

Species used: There are 85 or maybe 100 species of plants in the genus *Zingiber*. (Why have taxonomy anyway?) *Z. officinale*, the common food ginger, is the most famous and the one generally used for medicine. Many of the species in this family contain similar constituents and can be used medicinally. Some are similar in their antiviral actions, some are very different. This short monograph explores only the culinary ginger, *Z. officinale*.

Common names: Ginger in English and about a billion other names depending on which culture and language you are using.

Part Used

The root (yes, I know it's really a rhizome, but no one cares).

Preparation and Dosage

If you are using ginger as an antiviral, the fresh juice cannot be surpassed in its effectiveness. It takes about 30 minutes after drinking the fresh juice as a hot tea for ginger's compounds to enter the bloodstream; they reach peak concentration in about 60 minutes and then begin to decline. The fresh juice tea should be consumed every 2 to 3 hours in acute conditions or at the onset of colds or flu to keep the constituents at high levels in the blood.

FRESH GINGER JUICE TEA

Juice one or more pieces of ginger, in total about the size of a medium to large carrot, or four pieces the size of your thumb. *Save the plant matter that is left over* after juicing (for making an infusion; see below) or else squeeze it as dry as you can to extract all the juice still in it—there's a lot.

Combine 1 to 2 ounces of the fresh juice with 8 to 10 ounces hot water, 1 tablespoon wildflower honey, one-quarter of a lime (squozen), and ⅛ teaspoon cayenne. Drink 4–6 cups per day.

INFUSION

Method one: The leftover plant matter from juicing the root can be put into 1–2 cups hot water, depending on how much you have left, and allowed to steep for 4–8 hours, covered. Strain, and use the infused liquid as you would ginger juice in making fresh ginger juice tea (above). It will be almost as useful as the fresh juice but not quite.

Method two: This is the method to use if you don't have a juicer for juicing the ginger root. Grate or chop the ginger (a piece about the size of your thumb) as finely as you can. Steep in 8–12 ounces hot water for 2–3 hours, *covered* in order to preserve the essential oils in the tea. Drink 4–6 cups daily.

In acute conditions: 6 cups of the infusion per day minimum.

TOPICALLY

Ginger juice is exceptionally good (sometimes) in relieving the pain of burns and speeding up healing. Apply the fresh juice topically to the affected area with a cotton ball. It is also a good antibacterial and antifungal when applied to skin infections.

AS TINCTURE

Fresh root, 1:2 (1 part ginger to 2 parts liquid), in 95% alcohol. Dosage: 10–20 drops up to 4x daily. (I do not prefer this approach, as the fresh juice is much, much better—nevertheless it is a million . . . well, okay, a billion . . . times better than using the dried root.)

AS FOOD

In everything and anything, often.

Side Effects and Contraindications

Large doses should be avoided in pregnancy due to the plant's emmenagogue effect, though the dried root can be used to help morning sickness in moderate doses. May aggravate gallstones, so caution is advised. Rarely: bloating, gas, heartburn, nausea—usually when using the dried, powdered root.

Herb/Drug Interactions

The root is synergistic with a number of antibiotics, especially the aminoglycosides, increasing their potency, especially against resistant organisms.

Alternatives: *Alpinia galanga*, also known as galangal, is a close relative of culinary ginger and is also used in cooking, primarily in Asia (it is common in Thai food, for example). It has a similar range of antiviral action. Again, the fresh juice and the ethanol extract of the fresh root are the strongest antiviral forms of the medicine to use. Other edible gingers, such as *Zingiber zerumbet*, another Southeast Asian culinary ginger, are also very high in antiviral activity.

Habitat and Appearance

The exact geographical location of the original ginger plant is unknown—most likely someplace in Asia. It has been cultivated for 4,000 or more years in China and India and reached the West around 2,000 years ago. The genus name *Zingiber* is of ancient Hindu extraction; it means "horn-shaped." ("They" say it's from the shape of the root, but I don't believe it; I've seen ginger roots. But perhaps it came from a double-blind study . . . that's where they blind *both* the taxonomist's eyes and . . .) The roots form dense clumps as they grow and that is what everyone harvests.

The plant is a perennial and likes warm, humid climates from sea level up to about 5,000 feet in altitude. It is rarely found wild; it's a cultivated medicinal.

The plant grows 2 to 3 feet in height and looks like sort of a shortish bamboo with a thin central stalk. *Zingiber* plants look much alike and are often confused with the alpinias, another genus in the family.

Cultivation and Collection

Again, the root of ginger is really a rhizome but nobody cares about the distinction except for phytogrammarians, so I will just call it a root as nearly all people who use language do.

Ginger is almost always cultivated from pieces of the living root, like potatoes. Simply allowing some ginger root to begin budding, then cutting it into pieces, each with a bud, and planting them is usually how it is done. Most ginger plants on Earth are rootstock clones (kind of a Stepford Wives sort of thing). It is one of the most heavily cultivated plants on Earth. *Everybody* loves it (well, almost everybody). The plant is considered a perennial but it generally depletes the soil in which it is grown so it's usually rotated every other year. Unless it is in the exact right location it won't last once the soil is depleted.

Ginger is a tropical plant. It likes sheltered locations, filtered sunlight, warmth, humidity, rich soil. It hates direct sun and so on; basically it wants to be pampered and protected from the elements. It can't take freezing.

The root cuttings should be planted in late fall or early spring. No direct sun locations. Plant the cuttings 2 to 3 inches deep with the bud upward.

The plants need a lot of water, so don't let the soil dry out. Mulch them thickly. They hate dry air. The leaves die back in 8 to 10 months, and that is when the roots should be harvested. The roots will last a long time before they dry out; they should be used fresh if you are using them as antivirals.

Plant Chemistry

There are over 400 constituents in the root, including gingerols, zingiberol, zingiberene, zerumbone, shogaols, 3-dihydroshogaols, gingerdiols, mono- and diacetyl derivatives of gingerdiols, dyhydrogingerdiones, labdadiene, and so on. The volatile oils such as the gingerols are very potent but much reduced in the dried roots. They are present at levels 6 to 15 times higher in fresh roots. Many constituents convert to shogaols as the root dries. The volatile constituents are the most antiviral.

Properties of Ginger

Actions

As an antiviral, ginger inhibits the attachment of viruses to the cell, inhibits hemagglutinin, inhibits viral proteases, inhibits neuraminidase, stimulates antiviral macrophage activity, is virucidal. It is also:

Analgesic
Anthelmintic
Antiarthritic
Antibacterial
Antidiarrheal
Antiemetic
Antifungal
Anti-inflammatory
Antispasmodic
Antitussive
Carminative
Circulatory stimulant
Diaphoretic
Elastase inhibitor
Hypotensive
Immune stimulant
Synergist

Active Against

Ginger has been used across the world for treating a large range of viral infections including colds, influenza, hepatitis, herpes, yellow fever, measles, chicken pox, and enterovirus. Note: The list that follows contains *only* those viruses (and other microbes) that it has been found effective for in medical research studies; it has been used against a wider range in historical practice.

Given the test range, ginger should be thought of as a narrow-spectrum antiviral—primarily specific for respiratory viral infections, though its range of actions makes it a very good supportive herb for most viral infections. It is active against influenza A, rhinovirus (especially 1B), human cytomegalovirus, hepatitis C, HIV-1, Epstein-Barr, HSV-1 and HSV-2 (resistant or otherwise), Newcastle disease virus (Ranikhet strain), vaccinia virus, tobacco mosaic virus, and poliovirus (type 3—mildly so).

The herb has a decent range of antimicrobial actions as well, against:

Acinetobacter baumanii
Angiostrongylus cantonensis
Anisakis simplex
Aspergillus niger
Bacillus subtilis
Campylobacter jejuni
Candida albicans
Candida glabrata
Coliform bacilli
Dirofilaria immitis
Escherichia coli
Fusarium moniliforme
Haemonchus contortus
Haemophilus influenzae
Helicobacter pylori (cagA+ strains)
Klebsiella pneumoniae
Listeria spp.
Porphyromonas endodontalis
Porphyromonas gingivalis
Prevotella intermedia
Proteus vulgaris
Pseudomonas aeruginosa
Salmonella typhimurium
Shigella dysenteriae

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Shigella flexneri
Staphylococcus aureus
Staphylococcus epidermidis
Streptococcus viridans
Toxoplasma gondii
Trypanosoma evansi

Use to Treat

Ginger is best thought of in the following way: as a respiratory antiviral circulatory stimulant that will calm nausea, reduce diarrhea and stomach cramping, reduce fever (by stimulating sweating), reduce cold chills, reduce inflammation in bronchial passageways, thin mucus and help it move out of the system, reduce coughing (as much as codeine cough syrups), ameliorate anxiety, and provide analgesic relief equal to or better than ibuprofen. It is a synergist, increasing the actions of other herbs and boosting their effectiveness by relaxing blood vessels and increasing circulation, thus carrying the active constituents of the other herbs more efficiently throughout the body.

If used at the onset of a cold or flu, i.e., *the very day you sense it coming on*, it can cut down sick time to 3 days or less and the episode will often be mild. If used once the flu or cold is fully blown it will help ameliorate the symptoms considerably and shorten the illness. How much depends on your general immune health. If you've been burning the candle at both ends and putting off resting for too long . . . well, get some soup and settle in for some time off.

The herb can also be used in some bacterial diarrheal conditions, especially where there is cramping (cholera, dysentery, *E. coli*, etc.), for reduced circulation with coldness in the extremities, for migraine headache if accompanied by cold hands or feet, and for a sluggish constitution.

Finding It

Grocery stores everywhere.

Traditional Uses

Ginger has been used every place it is grown as a medicine. *Everyone* not trapped in a technological culture uses it for healing (colds and flu, nausea, poor circulation), for food preservation, and so on. In general, the methods of preparation are the same and they entail the use of the fresh root, *not* the dried.

In Burma, fresh ginger root is boiled in water (with palm sap to sweeten it) to get a hot infusion for treating colds and flu. In Congo, ginger is crushed and mixed with mango tree sap for colds and flu. In the Philippines fresh chopped ginger is boiled with water, and sugar added, for sore throats. It is used similarly in China and India. (There is a reason it is done this way.)

Ginger has a long historical tradition in warm climates as a food additive. Like many culinary spices it possesses strong antibacterial activity against a number of food-borne pathogens—especially against three of those now plaguing commercial foods: *Shigella, E. coli,* and *Salmonella*.

Two of the best ways to take ginger as food are the pickled ginger often served along with sushi in Japanese restaurants or candied ginger root slices. Both make great snacks, can be eaten in large quantities, and are a healthy stimulant for the system.

AYURVEDA

Ginger has a very long history in Ayurveda, which calls it srangavera and about 50 other names depending on where you go. It is used for dyspepsia, flatulence, colic, vomiting, spasms of the stomach and bowels attended by fever, cold, cough, asthma, indigestion, lack of appetite, diarrhea, fever. The fresh juice (ahh!), mixed with sugar and water, is a common form of preparation.

TRADITIONAL CHINESE MEDICINE

Fresh root: sheng jiang. (The dried root is termed gan jiang—a very different medicine.) Considered pungent and warm in traditional Chinese medicine, it is used as a diaphoretic, antiemetic, mucolytic, antitussive, detoxicant, anti-inflammatory. It is considered specific to warm the lungs, for pathogenic wind-cold conditions (i.e., severe intolerance to cold), and for slight fever, headache, general ache, nasal

congestion, runny nose, cough, vomiting. It is usually prepared by decoction in water or pounded and the juice added to warm water (ahh! once more). Ginger is generally combined with other herbs in traditional Chinese medicine as it is considered to be a "guide" drug that carries the other herbs where they need to go. Ginger is also considered to be specific for ameliorating the toxic effects of other drugs or herbs. Estimates are that up to half of all Chinese herbal formulas contain it.

WESTERN BOTANIC PRACTICE

Everyone in the West has used ginger in much the same ways though, historically, most of them tended to focus on its use for stomach and bowel complaints.

Scientific Research

The research on ginger has been problematic in that distinctions haven't been made (or looked for) between the actions of the fresh root and the dried root. (Common among scientists.) Nor has there been clarity about *how* the herb is prepared or what effect that might make on outcomes. (Common among scientists.) It is very rare that fresh preparations have been tested. (Ridiculous since that is the *primary* form of the medicine the world over.) Water extracts of the dried roots show very little antimicrobial activity—though they remain potently anti-inflammatory.

If you don't understand the problems inherent in the journal papers, the outcomes—which vary all over the place—are hard to understand. Sigh. Plants possess very different medicinal actions depending on when they are harvested, how they are harvested, if they are dried or fresh, how they are prepared as medicines, how often they are taken, how much is taken, and if they are taken in isolation or in combination. Scientists coming from a reductionist orientation have a hard time understanding all that; they don't understand that herbal medicine really *is* rocket surgery.

In the case of ginger, a further irritant is that there has been no clinical work on its use for viral diseases in spite of the fact that everyone on Earth uses the herb for colds and flu. (And if there were studies, they would probably have used the dried root and found it, unsurprisingly, to be useless.)

As an overview: There have been some 30 clinical trials with 2,300 people using ginger root. Following is just a sampling of a few of those and of a few in vivo and in vitro studies. There are about 1,400 journal listings at PubMed for studies on the plant.

Anti-inflammatory actions. Gingerol and its related compounds are potent inhibitors of lipopolysaccharide-induced PGE2 production in vitro. They are also very strong inhibitors of NF-κB expression and TNF-α. In vivo, through such inhibition, the herb reduces the incidence of liver neoplasms in mice and blocks the development of liver cancer. Ginger inhibits both COX-1 and COX-2 in vitro through inhibiting several genes involved in the inflammatory response (acting on cytokines, chemokines, 5-lipoxygenase, and COX-2). In a trial with 56 people (28 with rheumatoid arthritis, 18 with osteoarthritis, 10 with muscular discomfort) who took dried ginger, 75 percent reported relief from pain and swelling. In a double-blind, randomized, placebo-controlled clinical trial with 102 people with osteoarthritis, ginger was found to be as effective as ibuprofen in relieving pain and swelling. Numerous other in vivo studies have shown that ginger root has both anti-inflammatory and analgesic actions; some used the essential oil massaged into the affected area—it works really well.

Antiemetic/antinausea actions. Various clinical studies have found that ginger root is especially effective for treating severe morning sickness in pregnant women. The dried root was used, of course, and was found more effective in severe cases. (The fresh root is better for nausea.)

Antiadhesion actions. In vivo, ginger root interferes with the adhesion of enterobacterial disease organisms to the intestinal wall. This, in essence, reduces entero-infection of the GI tract, short-circuiting the disease process. Ginger is also an elastase inhibitor. Many bacteria use elastase to break down cellular tissue, helping their penetration of the body. (Ginger also reduces spasms in the intestinal tract, relaxing the intestinal wall, at the same time.)

Antidiarrheal actions. Ginger root interferes with the colonization of cells by enterogenic bacteria, thus reducing diarrhea and reducing bacterial load. The root alters bacterial and host cell metabolism through a unique-to-ginger mechanism.

Cerebroprotective actions. In vivo studies found that ginger root protects rats from brain damage and memory impairment.

Immunostimulant actions. In vivo studies with ginger root have found that it increases immune markers across the board, pre- and postinfection.

Detoxification actions. In vivo rat studies found that ginger reduced cadmium levels and toxicity in rats, acting as a heavy metal detoxifier. And ginger root in vivo reduces the effects of organophosphate insecticides.

Anthelmintic actions. Ginger was found to be effective in the treatment of endoparasites and stomach problems in ethnoveterinary practice in Pakistan, killing all red stomach worms (*Haemonchus contortus*) in test animals. It has been found active against a number of other endoparasites in other trials.

Synergist actions. Compounds from ginger have been found to be not only antibacterial but to modify bacterial resistance in *Acinetobacter baumanii* and to help potentiate the action of tetracycline. Ginger also potentiates the activity of aminoglycoside antibiotics (arbekacin, gentamicin, tobramycin, streptomycin) and other antibiotics such as bacitracin and polymyxin B against vancomycin-resistant enterococci.

Other studies have found antiulcer, antitumor, gastric antisecretory, antifungal, antispasmodic, anticonvulsant, and antiallergenic actions in the plant.

Houttuynia

Family: Saururaceae

Species used: *Houttuynia cordata* almost always. There are two species in this genus but the most recent one, *Houttuynia emeiensis*, was only discovered in 2001. Some taxonomists consider this second species identical with *Houttuynia cordata*, the species used for millennia in the East and first identified by the West in 1783. Some insist it is different. There has been some intense name-calling as a result. (Taxonomists are like our children.) The two species are used interchangeably but the newest one grows in a very limited range and is not widely available.

There is reportedly a wide range in the taste of *Houttuynia cordata*, which a number of sources attribute to chemical variations in the species depending on where it is grown. The Chinese/Vietnamese chemotype is reported to possess a taste/smell similar to coriander; the Japanese chemotype, according to one anonymous reporter, has "a strange lemon or orange odour that is often compared with ginger." To those who hate the taste every species apparently tastes like rotten fish. To those who like it, heaven itself has become food, and, they insist, the plant never has smelled or tasted like fish.

One of the plant's closer relatives is yerba mansa (*Anemopsis californica*), which grows in similar terrain, has a very similar flower, and has a somewhat similar range of medicinal actions. Yerba mansa was once named *Houttuynia californica* in the nineteenth century, which does lead, occasionally, to some confusion.

Synonyms: *Houttuynia foetida*, *Polypara cochinchinensis*, *Polypara cordata*, *Gymnotheca chinensis*, and, sometimes, if one group of taxonomists wants to make the other group really mad: *Houttuynia emeiensis*.

Common names: It's pronounced "hoo-TY-nee-ah" big fella, and, yes, you may yell that loudly at square dances, hootenannies, and cattle roundups. Other names: heart-leaved houttuynia, lizard tail, Chinese lizard tail, chameleon plant (as opposed to the specific variety called 'Chameleon'), heartleaf, fishwort, fishmint, bishop's weed, dokudami (Japan), and yu xing cao (China). The Chinese name literally means "fishy-smell herb" because, well, it smells like fish.

Parts Used

The aerial parts are used for medicine, the roots and leaves as pot herbs everywhere they grow (well, except in the United States).

Preparation and Dosage

The fresh plant is much more antibacterial/antiviral (as is the tincture) and is traditionally pounded to make juice for oral administration internally, on wounds, or as eyedrops. The remaining mashed plant can be used as a paste applied topically to wounds and bites; the decoction (allowed to cool) can be used for an external wash. The Japanese use a tea, taken regularly, as a tonic medicine. I prefer a tincture of the fresh plant leaves.

FRESH PLANT TINCTURE

The tincture should preferably be made 1:2, that is, one part herb to two parts liquid. The liquid should be pure grain alcohol if you can get it. So, if you have 16 ounces of plant leaves, you would add 32 ounces of alcohol, let it macerate for 2 weeks, decant, and press out the liquid.

Contrariwise, you can make it 1:5 from the dried leaves. That is, one part dried leaves, five parts liquid. In this instance the liquid should be half pure grain alcohol and half water, essentially a 50 percent alcohol liquid. So, if you have 1 ounce of leaves you would use 5 ounces of liquid and then continue as above.

As for dosage:

For viral infections: ¼–½ teaspoon up to 6x daily, depending on how acute the condition is.

For mycoplasma and bartonella: ½ teaspoon 3x daily.

The tincture can taste nasty, very fishy to some, so put it in something with a strong taste to cover it (fish soup?). Otherwise it can be hard to get it down—for some. I don't find it all that bad myself. It is not great but is only mildly fishy to my incredibly sensitive ("Hey! Are you looking at me?") taste buds.

There are a few companies selling the tincture for absurd prices, which, given that the plant is an invasive and very easy to grow, I find obscene.

DECOCTION

Traditionally the herb (sometimes the root) is used, either dried or fresh, to make a decoction. For dried, 15 to 30 grams of the dried plant is decocted (that is, briefly boiled), allowed to cool, then consumed. Fresh, 30 to 50 grams of the fresh herb is decocted similarly. Examination of the decocted herb has, however, revealed that it loses much of its antibacterial/antiviral actions upon being boiled (which is why the Chinese tend to boil it really, really briefly). If decocted intensively, the plant works well to stop diarrhea but is relatively inactive antimicrobially.

POWDERS AND CAPSULES

You can also find the powder, sometimes concentrated at 5:1, sometimes just the regular old powdered herb, from some Chinese herb companies. You can encapsulate the powder if you cannot take the taste of the tincture. I have been unable to locate any pre-encapsulated forms on the market. The herb really isn't that popular in the West at this point.

If you do encapsulate it yourself, use "00" capsules. I would begin with two capsules 3x daily and see how it goes, adjusting the dose depending on how it works for you. Contrariwise, you can work with the powder directly. I would begin with ½ teaspoon 3–6x daily and see how it works. (I normally only use the tincture.)

I have never been sure of how to dose the 5:1 concentrated powders that the Chinese often make; presumably you would take one-fifth the dose of the nonconcentrated form but that is just a guess.

Side Effects and Contraindications

Fishy-smelling breath (according to several former spouses). The taste can be terrible to the point of gagging (some say). Other than the nausea from the taste there are no reported side effects in the literature from oral ingestion of the plant.

It does have emmenagogue actions (though oddly enough the herb is not traditionally used for starting menstruation) so it should not be used in pregnancy. However, a few individual reports from China say it can, very rarely, cause congestion in the vagina (but I am not really sure what that means unless it is an overproduction of mucus, as in "congested" lungs or nasal passages; it certainly can't be congestion as in

"traffic congestion," as in "traffic on I-80 is backed up to the State Street Parkway so take an alternate route").

The Chinese sometimes use it as an injectable and there have been some severe anaphylactic reactions to that. So . . . don't inject it.

Herb/Drug and Herb/Herb Interactions

None have been noted in the literature or in any anecdotal reports that I can find.

Habitat and Appearance

Houttuynia is a creeping perennial. The stem is a sort-of trailing viney thing that creeps along the ground and from which substems sprout vertically to about 14 inches in height at most. When thickly growing, it can look a bit like a small bush. The leaves are heart-shaped, alternate, from 1 to 3 inches wide, and 1 to 3 inches long. The (original, noncultivar) plant leaves actually look quite a lot like those of the common violet. The noncultivar flowers are pretty, four-petaled, greenish-white with an upraised spike sort of like a tiny cattail (a.k.a. the terminal spike).

A number of cultivated varieties have been mucked about with to give color variations. They may be mottled green and red, green and yellow, green-leaved with a rim of red, magenta and green, and orange or even mixes of all of those. There is even a nearly black-leaved variety. And of course the flowers have been altered as well, with some of the varieties sporting red blooms. These varieties have the usual ridiculous names associated with them: Chameleon, Flame, Joker's Gold, Sunshine, Variegata, and so on. They are common in gardens throughout the world.

The roots are more correctly creeping rhizomes that run just under the soil in a tangled mat something like a writhing mass of spaghetti noodles. They are sort of golden in color.

The original native range of the plant was wide, from Nepal and India through China and Indochina into Japan, and south into Vietnam, Thailand, and Java. But it's a good hitchhiker and spread, in its original form, throughout the Pacific islands, Australia, and New Zealand as ships sailed here and there over the centuries. Once it was introduced as an ornamental it went everywhere and is now common throughout Europe and North America. It has an especially strong presence in eastern Europe, the United Kingdom, and Russia.

The plant will grow from sea level to 7,500 feet and it likes it wet, that is, if it is growing wild. Its standard terrain is ravines, streamsides, forests, wet meadows, slopes, thicket and field margins, trailsides, roadsides, and ditch banks.

It tends to prefer moist loamy soils, shallow water, and low light conditions (dappled sun/shade) either in forests or not. It is particularly fond of growing on the margins of ponds and waterways—really any type of wettish to boggy location that is coolish and shaded. It is highly tolerant of cold and is hardy to about 0°F (–18°C) but can tolerate dips to –30°F (–34°C). (Strictly Medicinal Seeds grew it in unheated greenhouses in February in the state of Oregon just fine.) It is hardy in zones 4 through 11, essentially most of the United States. In spite of its natural preferences, the plant can tolerate full sun (though it may scorch—it really does like a bit of shade) and is drought resistant. Few animals will eat it; rabbits (reportedly) detest it (they have the second kind of taste buds—*bunny* buds).

Cultivation and Collection

Houttuynia is an invasive, which makes it of particular interest to me. Like many invasives it is potently medicinal and a decent edible, if you like the flavor. I consider it another plant that should be planted by everyone who wants healing independence and wide availability of potent medicinal plants for themselves and their families. Once it's planted you will have both food and medicine forever. Just don't let your neighbors catch you—practice the confused look you developed in high school for the time someone brings up the fact the plant is killing off the *real* American plants and that you, yes *you*, have a duty to stop it.

The plant is banned from importation in New Zealand (too late) and a number of other places (still too late). The plant is reportedly invasive in Texas, Louisiana, Alabama, Florida, North Carolina, and Pennsylvania.

Houttuynia is a hermaphrodite—another reason many Americans are uncomfortable with the plant. Thus, it can self-seed—there doesn't have to be a mommy *and* a daddy. There are some 1,000 seeds per 0.04 gram of seed weight and every plant produces a lot of seed. Any and all portions of the root will grow as well. It is just reproductively irritating no matter how you look at it.

Once established it just doesn't let go. There are numerous humorous reports on the internet of people planting it as a ground cover and having it take over their gardens, yards, and lives. Digging it out is difficult as every piece of overlooked root, no matter how small, will, well, root and produce a new plant, something every eradofanatic discovers the next spring. From one killed plant, a thousand rise to take its place. Often the internet stories end with the writers adding rather glumly that their houses are for sale.

Plant sex, destroying American suburbs everywhere.

Reportedly the growing condition that will produce the strongest growth is a clay, loam soil, 5.9 pH, with 78 percent moisture content. Still, houttuynia can be grown in much drier conditions than it normally likes as long as you keep it watered. It will tolerate sandy, loamy, and clay soils. It can even grow in water up to 2 inches deep. It flowers in June (usually). It grows extremely quickly.

Houttuynia is reportedly very hard to grow from seed but those who have done it say that you should sow the seeds in a greenhouse in spring, keeping the soil moist, then separate them into individual pots when they are big enough. Transplantation can occur any time after that.

The more variegated the plant, the less hardy, the less medicinal, the less invasive. Most of the variegated species will tend to relapse into the more basic unvariegated state if you let them. Gardeners are warned to pick off and dispose of any green leaves that show up to keep this from happening. I would suggest the opposite approach.

In China, the plant is traditionally harvested after flowering and then dried in the sun. It is usually used as a decoction, 15 to 30 grams of the dried plant or 30 to 50 grams of the fresh. It is usually not decocted for long in order to prevent the heat loss of volatile oils. The more it is heated, the worse its antimicrobial actions become. The primary reason it is heated is to get rid of the fishy smell—about 1 minute does it, so they say. However, in spite of all that, the Chinese consider the fresh plant the strongest form of the herb.

If you are making tinctures use the fresh plant picked just at flowering. If you want to dry the plant, bundle and hang it, and when it is completely dry store it in plastic bags out of the sun, preferably in plastic tubs, well sealed.

Note: Houttuynia hyperaccumulates lead and arsenic, so don't harvest it from around mines.

Properties of Houttuynia

Actions

As an antiviral, houttuynia inhibits viral replication, interferes with the function of the viral envelope, is directly virucidal, stops virion release from infected cells, prevents viral infection if taken prophylactically. It is also:

Analgesic
Anthelmintic
Antibacterial
Anticancer
Antifungal
Anti-inflammatory
Antileukemic
Antimicrobial
Antioxidant
Antitussive
Astringent
Diuretic
Depurative
Emmenagogue
Febrifuge
Hemostatic
Hypoglycemic
Immunomodulatory
Larvacidal
Laxative
Ophthalmic

Active Against

Moderately broad-spectrum antiviral. It is active against influenza virus A (H1N1 strains), SARS-related coronavirus (FFM-1, FFM-2), dengue virus serotype 2, avian infectious bronchitis virus (a coronavirus), enterovirus 71, enteric cytopathic human orphan (ECHO) virus, herpes simplex virus 1, herpes simplex virus 2, HIV-1, cytomegalovirus, porcine epidemic diarrhea virus, and pseudorabies herpesvirus. Studies have found it ineffective against polio and Coxsackie viruses.

The herb also has a good range of action against bacteria and other microbes. It is active against:

Aedes aegypti larvae
Aspergillus spp.
Candida albicans
Chromomycosis fungus
Colletotrichum capsici
Corynebacterium diphtheriae
Cryptococcus neoformans
Diplococcus pneumoniae (a.k.a. *Streptococcus pneumoniae*)
Epidermophyton rubrum
Fusarium oxysporum
Haemophilus influenzae
Hymenolepis diminuta
Leptospira spp.
Malassezia pachydermatis
Microsporum ferrugineum
Microsporum gypseum
Mycobacterium tuberculosis
Mycoplasma hominis (30 strains)
Neisseria catarrhalis (a.k.a. *Moraxella catarrhalis*)
Proteus vulgaris
Salmonella choleraesuis (a.k.a. *S. enterica*)
Salmonella enteritidis
Sarcina ureae
Shigella flexneri
Shigella schmitzii (a.k.a. *S. dysenteriae*)
Shigella shigae (a.k.a. *S. dysenteriae*)
Shigella sonnei
Sporotrichum spp.
Staphylococcus albus
Staphylococcus aureus
Streptococcus hemolyticus
Tinea imbricata
Vibrio cholerae

There are a number of internet sites insisting that the herb is active against trichophyton and gonococci but intensive searching has failed to turn up any relevant documents supporting it. Every site I can find simply repeats the same thing—all apparently from the same initial site, wherever that is.

Use to Treat

Respiratory viral infections—especially SARS and influenza, ECHO infection, neurological enterovirus infections, neurological encephalitis infections, and dengue fever.

The herb is also excellent for mycoplasma infections, any serious infections in the lungs especially with abscesses, infections in the urinary passages and kidneys, genital infections, dysentery and any bacterial diarrheal conditions, various diseases of the eye (fresh juice or tea applied topically), skin infections with pus or boils. It is especially indicated if any of these conditions are accompanied by foul-smelling discharge.

Other Uses

As food. The young shoots and leaves are very tender (later in the year the leaves become somewhat bitter) and are a major food source in all the plant's native regions. There are varying reports on the taste and smell. Some say that the leaves have a slight orange smell and taste, others that it is reminiscent of coriander, others insist it is actually a slight fishy smell and taste, and still others report that it is more akin to rotten fish. From all accounts this is another of those plants like cilantro—you either love it or hate it. If you hate it, the taste is rank (in varying degrees of rankness depending on your level of hate). However, if you love it, it is wonderful with delicate shadings of flavor. (Taxonomists are now arguing that there are two species of taste buds, one that . . .)

The leaves, for those who like it, are used in salads or are steamed as a pot herb or even blended with rice as a main dish. The rhizomes are washed and cut in 3- to 4-inch lengths and added to stir-fry. Both are sometimes used as a main ingredient in meat and other dishes. The leaf tea (dokudami cha) is common in Japan as a medicinal drink.

Finding It

If you are going to grow it for medicine, try to get the original, unmucked-about-with plant if you can, not the highly variegated varieties ('Chameleon' is the worst to use for medicine). Strictly Medicinal Seeds sells the plants and they are pretty good ones—find them at https://strictlymedicinalseeds.com.

You can get the seeds from suppliers here and there but the plants are reportedly hard to grow from seed; the easiest way to grow the herb is from divided rootstock. If you want a very good fresh tincture at a decent price try Woodland Essence (https://woodlandessence.com). The dried herb (in both powder and cut form) is available from 1st Chinese Herbs (https://1stchineseherbs.com).

Plant Chemistry

Houttuynoside A, various houttuynoids (A through E), houttuynin, lauryl aldehyde, caprylic aldehyde, quercetin 3-rhamnoside, quercetin 7-rhamnoside, n-capric acid, cordarine, quercitrin, isoquercitrin, decanoyl acetaldehyde, alpha-pinene, beta-pinene, linalool, camphene, myricene, limonene, caryophyllene, afzerin, hyperin, chlorogenic acid, beta-sitosterol, stearic acid, oleic acid, linoleic acid, myrcene, 2-undecanone, hyperoside, p-cymene, eucalyptol, beta-ocimene, nonanal, fenchyl alcohol, menth-2-cn-l-ol, trans-pinocarveol, verbenol, camphor, beta-terpineol, pinocarvone, isoborneol, pelargol, terpinen-4-ol, myrtenal, alpha-terpineol, verbenone, trans-carveol, piperitone, isopulegol acetate, bornyl acetate, isobornyl acetate, benzyl isobutyrate, undecanal, alpha-terpinyl formate, dihydrocarvyl acetate, neryl acetate, undecyl alcohol, geranyl acetate, 4-acetamido-l-hexanol, beta-caryophyllene, beta-farnesene, lauryl alcohol, beta-chamigrene, valencene, methyl undecyl ketone, alpha-bulnesene, dodecanoic acid, nerolidol, spathulenol, caryophyllene oxide, viridiflorol, juniper camphor, methyl tridecyl ketone, phytone, heptadecanol, phytol, phytol acetate, a variety of aristolactams, piperolactam, aporphines, splendidine, lysicamine, 4,5-dioxoaporphines, norcepharadione B, noraritolodione, various amides, and so on. The herb is reportedly high in potassium, magnesium, and sodium.

A significant number of these compounds possess antiviral and/or antibacterial actions. The whole herb was found to be more effective

than any of the isolated constituents, showing a profound synergism in its chemical actions. One study found *Houttuynia emeiensis* to be more potent in its antibacterial effects than *H. cordata.* (Several taxonomists were found fighting in the parking lot just afterward.)

Traditional Uses

There is a lot of indigenous and local use of the plant throughout its native range, both for food and medicine. The tribes of the Kameng district of Arunachal Pradesh (which is in northeast India) use 1½ ounces of the fresh root, boiled as a decoction until reduced by half. The dose is half a glass of the decoction twice daily for a week in the treatment of dysentery, diarrhea, and cholera.

In Nepal the fresh juice of the root is used for indigestion, topically for skin diseases, as eyedrops for eye infections. The juice of the leaf is dripped into wounds to prevent infection, to kill maggots in the wounds, and to accelerate healing.

In Thailand the herb is used to treat venereal and skin diseases and as a diuretic and urogenital antiseptic.

It is used in Japan as a tonic tea, for chronic earache, as a lotion, and as a liquor. Its name in Japanese, dokudami, literally means poison-blocking. It is considered to be an herb for detoxification of the blood to increase overall health of the body. Usually 4 to 12 grams (1/7 to a bit less than ½ ounce) are used to make the tea. The lotion is made by soaking the dried plant in shochu liquor (about 25 percent alcohol) for 10 days, straining it, then adding glycerin. It is then used on the skin for healing. The liquor is made by putting the dried plant in a bottle, adding shochu to about three-quarters the level of the bottle, then adding honey to top it off. After 3 months, the herb is filtered out and the liquor stored in the refrigerator. It is drunk as a tonic. Essentially this is what we herbalists in the United States would call a cordial.

The greatest depth of use has been in traditional Chinese medicine.

AYURVEDA

In spite of tribal use, I can't find much on the plant as a part of formal Ayurvedic practice. It is apparently used in combination formulas for the treatment of AIDS (as an antiviral adjuvant) throughout the country but that is about all I can find.

TRADITIONAL CHINESE MEDICINE

In traditional Chinese medicine yu xing cao is considered to be slightly cold, pungent, and specific for the lung channel. It has traditionally been used for removing toxic heat, eliminating toxins, reducing swelling, discharging pus, and relieving stagnation. It is specific for promoting drainage of pus, lung abscess with purulent expectoration, heat in the lung with cough, lung abscesses, cough with thick sputum, dyspnea, edema, carbuncles and sores, dysuria, leukorrhea, acute dysentery, and acute urinary infections. It is considered latent-heat clearing, antipyretic, detoxicant, anti-inflammatory, and diuretic. The fresh juice is used for snakebite and skin infections. Common medicinal use in China is for chronic nephritis, inflamed pelvis or cervix (pelvic inflammatory disease), gonorrhea, rheumatism, anal prolapse, hemorrhoids, inflamed respiratory tract (including pneumonia and bronchitis with or without edema), prevention of postoperative infections, inflammation and pus in the middle ear, measles, tonsillitis, chronic sinusitis, nasal polyps, inhibiting anaphylactic reactions, and various cancers.

Again, the fresh leaf is considered to be more efficacious than the dried.

WESTERN BOTANIC PRACTICE

Very little use in the West until recently. Few American herbalists use it. Most understand it not at all. It has achieved some prominence from those treating Lyme and its coinfections, usually bartonella.

Scientific Research

Most of the scientific studies have occurred in China, often with injectable forms of the herb. Numerous others have been conducted in India and Thailand. Few have occurred in the United States. (The herb tastes funny.) In general, the studies found that the effects were dose dependent, in other words, the more they gave, the better the outcome. Since the herb has shown no toxicity from oral ingestion (up to 16 grams per kilogram in mice, a huge dose), that would indicate that largish doses can be used very effectively.

In vitro studies found that the herb (water extract) significantly increases IL-2 and IL-10 cytokines. IL-10 is also known as cytokine synthesis inhibitory factor; it is an anti-inflammatory cytokine. It essentially downregulates other cytokines and blocks NF-κB activity. It is specific for counteracting the effects of mast-cell-initiated allergic reactions, which is why the herb is good for stings and bites and anaphylactic reactions. The herb also stimulates the production of CD4+ lymphocytes. It is especially active in the spleen.

Herpes simplex virus (HSV) 1 and 2 depend on NF-κB activation for replication. In vitro studies found that houttuynia suppresses HSV infection by inhibiting NF-κB activation. The herb's inhibition of NF-κB has also been found to significantly reduce chemotaxis during infection, reducing cellular migration. The herb also inhibits hydrogen peroxide impacts on cells and lipid peroxidation by 80 percent.

In vitro research found that the herb is active against 21 staph aureus strains (but is not very active in stopping biofilm formation in that organism).

In vitro study found that while the herb is strongly active against enterovirus 71, its activity is much higher in cells pretreated with the herb than if it is administered postinfection.

In vitro the herb inhibits the production lipopolysaccharide-induced COX-2 and PGE2 in mouse macrophages. The herb reduces Th2 cytokines, specifically IL-4 and IL-5. It inhibits HMC-1 cell migration. It also inhibits DNA topoisomerase 1 activity.

Houttuynia liquid extract protected and restored white blood cell counts in mice X-rayed and administered cyclophosphamide. It normalized connective tissue growth factor and increased levels of adiponectin in streptozotocin-induced diabetes in rats.

Eight hundred mg/kg of the herb reduced the numbers of *Hymenolepis diminuta* flatworms in rats by nearly 75 percent and the egg count by nearly 60 percent. In comparison the drug praziquantel showed 87.5 and 80 percent effectiveness.

Water extracts of the herb significantly reduced NO levels in *Salmonella*-infected macrophages and extended life spans of *Salmonella*-infected mice given a lethal dose of the bacteria from 7 days (no herb) to 23 days. The effects were dose dependent.

The herb is strongly inhibitive of avian infectious bronchitis in vitro and was found to protect chicken embryos from infection by the virus and to protect 50 percent of mature chickens from infection.

The herb, as a water extract, was used to treat bleomycin-induced pulmonary fibrosis in rats. The herb significantly decreased SOD, malondialhehyde, hydroxyproline, interferon-gamma, and TNF-α. The morphological appearance of the lung was markedly improved.

A number of studies found that the herb strongly inhibited induced-oxidation events in rats. It specifically inhibited NF-κB, TNF-α, NO, COX-2, and PGE2. It also inhibited passive cutaneous anaphylaxis (PCA) in mice, inhibited IgE-mediated systemic PCA, reduced antigen-induced release IL-4 and TNF-α, inhibited degradation of IkappaBalpha. It specifically inhibited antigen-induced phosphorylation of Syk, Lyn, LAT, Gab2, and PLC gamma-2. Further downstream it also inhibited Akt and MAP kinases ERK-1, ERK-2, JNK-1, and JNK-2 but not p38.

Extracts of the herb protected rat kidneys when rats were injected with streptozotocin. TGF-ß1 and collagen type 1 levels in renal tissues decreased, and BMP-7 increased.

Water extracts of the herb showed antiobesity effects in mice by inhibiting glycerol absorption and corn-oil-induced increases in triglyceride levels. The herb inhibited oleic acid increases in blood plasma.

Cows with bovine mastitis were treated with a form of houttuynin, a compound from houttuynia. In acute mastitis 88 percent were cured and 53 percent showed microbiological clearance. In cows treated with combination penicillin/streptomycin the rates were 90 and 55 percent respectively. In subacute conditions the houttuynin results were 94 and 48 percent. In the pharmaceutical group they were 94 and 44 percent. (This finding is significant because of the degree of mycoplasmal

infection in dairy herds, a major cause of bovine mastitis.)

A modified form of houttuynin both protected mice from and corrected induced membranous glomerulonephritis in mice. It inhibited the expression of NF-κB and MCP-1.

A water extract of the herb protected primary cortical cells in rats from beta-amyloid-induced neurotoxicity, specifically through modulating calcium influx and protection of mitochondria.

Researchers in Korea are interested in a class of food herbs they are calling phytobiotics, among which is houttuynia. In one study, researchers found that adding it to chicken feed instead of antibiotics has similar effects on weight, disease reduction, and health as pharmaceuticals without the negative side effects, including antibiotic resistance. Lipid oxidation in the meat was significantly reduced. In another study, the mortality of chicks challenged by *Salmonella* was significantly reduced when the herb was included in their feed. PGE2 synthesis decreased, CD4+ increased, the CD4+:CD8+ ratio balanced, immune function in all chicks was enhanced.

Many of the human studies with the herb used an injectable form and found it highly effective for treating bronchopulmonary complaints including pneumonia. Oral dosing of a compound formula that included *Platycodon grandiflorum* was also effective. Both oral and injectable forms were found prophylactic for leptospira infections. Used alone or in combination with *Artemisia annua* the herb was an effective treatment for leptospirosis.

Cotton impregnated with the water extract (or oral tablets) was used in treating chronic cervicitis with lesions. Applied once every day for 5 days (243 people participating) the cure rate was 81 percent.

Of 100 cases of chronic suppurative otitis media treated with ear drops of the distillate of the herb, 95 were cured. Thirty-one of 33 cases of atrophic rhinitis benefited from nose drops of the solution. Irrigation with the extract was effective in treating chronic maxillary sinusitis. And in other studies houttuynia (water extract) was used to irrigate nasal passages after endoscopic sinus surgery for those with chronic sinusitis and/or nasal polyps. The herbal extract was found to be more effective than two other irrigants used.

A clinical trial in China in the treatment of chronic-relapsing ulcerative colitis found that herb cured 20 of 21 and gave improvement in the other. Stool normalization, cessation of diarrhea, reduction of blood in stool, and abdominal pain disappearance all were faster in the herbal group than in those being treated with pharmaceuticals. (Again this is an important study as this kind of condition is relatively common in chronic mycoplasma infection.)

The herb's constituents move fairly rapidly into the bloodstream and maintain a high presence. The absorption half-life after oral ingestion is 3.5 hours. The herb's constituents are present in the highest amounts in the lungs, heart, liver, kidneys, and serum in that order. Elimination of constituents, metabolized or not, in the urine and feces is very low; the main route of excretion is the lungs (breath). Radioactively labeled houttuynine was found in rat tissues for up to 48 hours. The highest levels were in the bronchi (especially at 1 and 4 hours postinjection) and in descending order in the gallbladder, liver, ovaries, intestine, spleen, kidneys, and lungs. Oral dosing found the highest levels in the bronchi after 24 hours.

Isatis

Family: Brassicaceae. (This family is the home of all cruciferous vegetables such as cabbages, broccoli, and brussels sprouts and the reason why the tincture of this plant tastes kind of like spoiled broccoli/cabbage. So, if you combine it with houttuynia . . . well, let's just say you are in for a treat.)

Species used: There are somewhere between 30 and 80 species in this genus. Well-degreed taxonomists speak authoritatively, and rather insistently, of just how many species really are in this genus—few of them cite the same figures, or the same plants. As yet, I cannot find a definitive work on the genus. So, there's 30, or 48, or 79—a bunch of plants anyway. (Taxonomist: a person who is taxing, that is, someone "not easily borne, wearing, burdensome, onerous.")

The most commonly used species is *Isatis tinctoria* (worldwide) but *I. indigotica* is fairly prominently used in China (as is *tinctoria*), *I. costa* is used in Pakistan, *I. cappadocica* in Iran, and still others here and there. All the species seem to contain similar chemistry; all (at least all the sources I have read say so) have been used to produce the indigo dye that the genus is known for.

Synonyms: Some taxonomic compulsivists list *Isatis indigotica* as a synonym for *tinctoria* but chemical examination of the two continually reveals significant differences (not that *that* will deter them).

Common names: Isatis, woad, dyer's woad.

Parts Used

Root and leaves.

Preparation and Dosage

Isatis has traditionally been used as a decoction in China, where the longest history of use has occurred. The use of isatis tinctures is fairly new although the Chinese and Japanese have been testing the activity of alcohol extracts for several decades. The plant is rarely used as a single tincture but is normally combined with other herbs. If you are going to use it as a single you might consider using it as a decoction as the

Chinese have traditionally done. (I do not think that capsules or tablets are an effective form for the herb; there is no clinical data on them and I have not seen good effects in practice from their use.)

In traditional Chinese medicine the roots and leaves are considered to be different medicines and are used for slightly different things (see the section on traditional Chinese medicine, page 276). Most people in the United States who do make tinctures are using the root; nevertheless, the leaves are exceptionally potent and are considered to be more specific for upper respiratory infections than the root—they are the most antiviral part of the plant.

I agree with a number of practitioners who have found the leaf better for acute conditions, and the root for chronic. The root seems to be better at modulating immune dynamics, increasing immune response, and moderating inflammation—just helping to tone down and even out everything while increasing immune potency. It, as well, does have a number of the potent antiviral compounds in it, so I like to include it, with the leaves, in formulations. It is also the most antibacterial part of the plant. The best tincture formulation, in my opinion, comes from a mixture of the root and leaves: one part root, two part leaves. The roots are sometimes hard to dig if you are wildcrafting them, however the aerial parts can easily be wildcrafted and both root and leaves can be bought online.

The leaves need to be dried before tincturing; do not use them fresh unless you are making dye. In addition, they need to be heated (as does the root) to better extract the polysaccharides. (The Chinese do things for thousands of years for a reason.) Also: You need soft water, that is, acidic, anywhere from a pH of 1 to 6, in order to more effectively extract the bioactive alkaloids from the plant. Most of the constituents are soluble in water, so you won't need much alcohol, mostly just enough to bring out a few alcohol-soluble constituents and to stabilize the tincture so it won't go bad when stored. Studies have found that high-alcohol-content extractions are not as effective as water extracts for treating virus infections; alcohol/water combinations are better than pure alcohol or pure water for extracting the constituents needed to treat bacterial infections.

TINCTURE

Use an herb:liquid ratio of 1:5, with the liquid being 25 percent alcohol. Use two parts leaves, one part root. (Tastes terrible.)

Take (for example) 5 ounces dried root (ground well) and 10 ounces dried leaves (also ground well). That makes 15 ounces of herb. You will need five times that of liquid, that is, 75 ounces. Of that liquid, 25 percent (one-quarter of it) will be alcohol, the rest will be water. You will need, then, 56 ounces of water, 19 of alcohol.

Add the ground-up herbs to a cooking pot, mix in the 56 ounces of water, bring to a boil. *If you do not have soft water, or if you do not know, you will need to add 1 tablespoon of vinegar as well to help extract the alkaloids.* Cover the pot and boil for 30 minutes. Then let it cool to room temperature. You can put the covered pot in the sink with cold water to speed this up. (Don't let it tip over; just an FYI on that one.)

When the mix is cool, pour it all into a large jar with a lid, and add the 19 ounces (liquid measurement) of alcohol. Put the lid on and let it sit for 2 weeks. Shake it every once in a while.

When done, pour off the liquid, and squeeze the marc (the herbs) to extract as much liquid as you can. Then throw the pressed herbs away (in the compost or garden of course). Bottle and label. As for dosage:

As a preventive: 30 drops up to 6x daily.

In acute conditions: 1 teaspoon up to 10 times daily.

Note: Again, I would not normally use this herb as a single, but only in combination with other herbs such as lomatium or licorice.

DECOCTION

The Chinese dosage is quite high, as usual, compared to Western approaches. Root decoctions: 10–30 grams (1/3–1 ounce) of the root boiled for 30 minutes; 1 cup drunk 3x daily for a max of 3 weeks. Leaf decoctions: 9–15 grams; 1 cup drunk 3x daily (in acute conditions 60–120 grams).

CAPSULES OR TABLETS

Again, I am not convinced this is a useful way to take this herb; all indications from the research are that the herb is most effective if heated in water and a decoction made. Nevertheless, if you must: 200 mg 3x daily. In acute conditions: 2 grams daily. Again, the leaves are better for viral influenzas.

Side Effects and Contraindications

Leaf: Occasionally nausea, rarely vomiting. Root: Rarely allergic reactions, urticaria, cyanosis of the face, dyspnea—but these were from intramuscular injections; there is no evidence of this from oral ingestion.

However caution should be exercised in long-term use. Normally, you would not take isatis for longer than 3 weeks. This should be sufficient to deal with anything you have, especially if you have combined the herb with other antivirals. You should avoid using the herb as a single medicinal if you are presenting with a subjective feeling of cold without fever. The herb *may* induce a deep chill with overuse (longer than 3 weeks). Under some circumstances, longer use can lead to feelings of weakness, light dizziness, and an odd feeling in the bones. Stopping the herb will correct the condition within a few days. Isatis should not be used by people on dialysis or those experiencing renal failure—high doses or long-term use may negatively affect the kidneys.

Herb/Drug Interactions

Synergist with antibiotics and viral vaccines, increasing the activity of both. The herb may interfere with tests for measuring total bilirubin content.

Habitat and Appearance

Isatis is native to southeastern Russia and China but has hitchhiked widely with people and is now common throughout Europe, northern Africa, Japan, the United States, and Canada. Isatis is mainly a northern hemisphere plant. It was a major agricultural crop a great many places, planted widely for several thousand years, due to its use as an indigo dye for cloth—but it escaped. (Italy has now begun growing it again as an important agricultural crop—a source of natural, nonpolluting indigo dye.)

Isatis is invasive nearly everyplace it gets transplanted—yet another potent invasive medicinal for emerging infections. The plant is especially invasive in the western United States, up into Canada, and around the Great Lakes region. It can be found wild in California, Colorado, Idaho, Illinois, Montana, Nevada, New Jersey, New Mexico, New York, Oregon, Utah, Virginia, Washington, Washington, D.C., West

Virginia, Wyoming, British Columbia, Ontario, Quebec. It is considered invasive in Canada and at least eight western states. A few isatis plants even traveled as passengers on the first Chinese space mission—one giant leap for plantkind.[8]

Like many of the potent antimicrobials that we need for resistant infections, this one is growing all around us, insisting we notice it. Nevertheless, as an invasive, it has been targeted as a plant to be terminated with extreme prejudice. Plant purists (phytoaryans) of various stripes exist in many locations and spend a great deal of their time working to eradicate isatis. They tend to exhibit phytohysteria at the simplest provocation and will speak badly of the plant if you bring it up in conversation—forgive them, for they know not what they do.

Isatis is a biennial (usually) though it can annualize if the climate forces it to or even straggle on as a perennial for a while. The plant begins as a basal rosette and looks something like a cross between the dandelion rosette and that of broccoli. It does have some small, rounded teeth on the first-year leaves that the second-year leaves don't have, but there isn't the same kind of toothiness as that belonging to dandelion leaves. The leaves in first-year rosettes are up to 7 inches in length with a cream-colored midrib. They are broadest at the tip and taper to a point at the base. I think the plant looks more broccoli-like in color and texture the second year; the stalk it sends up reminds me quite a lot of that pile of semiedible stuff you have left after you take the broccoli heads off for cooking, or at least a well-watered plant does. (Unfortunately, to my tongue, the tincture tastes not like broccoli but a bit like spoiled cabbage—interfering with my enjoyment in taking it . . . a lot—and I work to mask it with other herbs such as yerba santa. It helps. A little.) Isatis growing in more semiarid locations tends to get a bit woody and darker in color as it ages, the stem being, like the ripe seeds, a bit purplish/brown in color. The second-year leaves clasp the stem rather than having a slender stem (petiole) attachment.

The flowering stalk that it sends up the second year, to my eye, looks a bit like broccoli when it has gone to seed—the agriculturally grown isatis more so. The flowering stalk can be from 1 to 4 feet in height; it puts out clusters of yellow flowers that look to my eye a great deal like those of St. John's wort. They are bright yellow. It flowers in May/June and begins to set seed immediately. The flowers continue growing up compounded racemes, the seeds following closely behind, developing

from the earlier flowers that have already matured. It takes about 8 weeks from the beginning of flower-stem growth to the setting of the first seeds.

If environmental conditions don't support seed production the second year, the plant can continue as a rosette for several years until they do. That plant, not surprisingly, tends to be bigger, both roots and rosette.

The seed pods (each containing one seed) are flattish and remind me of rolled oats, in spite of their green color when young. They turn a dark purplish-brown when ripe. The pods hang off the flowering stalk in their thousands, lined up like paratroopers about to exit a plane over enemy-controlled territory. Each plant can put out as many as 500 seeds a season; later generations tend to spread out from that first plant in a sort of expanding zone of conquest, extending from a common center. It doesn't take long to dominate the landscape. The seeds can remain viable for years in the soil. Once freed from the seed pod, *every* seed will germinate. (It can't be bargained with. It can't be reasoned with. And it absolutely will not stop. Ever.)

The plant loves alkaline, semiarid soils and it does very well in them. It spreads rapidly in such soils, and doesn't need disturbed soils to extend into a new locale; it crowds out normal vegetation just fine. The plant seeds produce potent chemicals that inhibit competing plants; even its own seeds won't germinate until the chemicals are leached out of the soil after the parent plant dies the second winter.

Isatis is especially invasive in ecologically disturbed areas (even if they appear sound to the eye), especially where there has been overgrazing by cattle. The rate of spread is intense, one site in Montana reporting an increase from infestation in 2 acres to over 100 in 2 years. It reduces cattle grazing capacity by about 40 percent on infested range—part of its ecological function. Even sheep and goats don't like to eat it and that is saying something. It is intensely bitter the older it gets.

Radishes are also a member of the Brassicaceae family and the root of isatis is indeed sort of radish-like, a bit like a cross between a daikon radish and a carrot, except for the color. The first-year root tends to be a bit fuller, more plump, and the root bark (tannish-brownish with a hint of gold) a bit lighter in color. The plants send out lateral roots in the upper foot of soil to tap surface water as well as sending down a taproot

to drink from deeper sources. The second-year roots go much deeper, up to 5 feet (3 feet is more common). The root bark the second year is a bit darker. When sliced the inner bark is a light cream, the core darker, the same color as the outer bark. The roots are fleshy, generally 1 to 3 inches in diameter and, unless garden-grown, hard to dig up except for the top layers. They like tough soil.

Cultivation and Collection

Isatis *loves* semiarid, alkaline, average-to-poor soils with enough water to ease its thirst but not to overhydrate it. However, if you garden-raise it and water it well, it will grow fleshy and fat and look much more like a broccoli plant than otherwise. The plant has very low nitrogen requirements. It propagates easily from seeds sown in sunny locations. Sow in fall or spring. It seeds itself readily once established. Survives to –30°F (–34° C). Doesn't like the shade; needs sun. Will grow from sea level to 7,000 feet or so. Tolerates salty air and soil. *Isatis tinctoria* is often one of the earliest plants to emerge in spring (self-motivated, early riser).

Usually, *only* the leaves and roots are used medicinally. The stems are discarded, the flowers ignored, the seeds overlooked. The seeds are most likely highly medicinal but for some reason no one has explored their medicinal uses. (They contain a large amount of diverse fatty acids and are very high in the anticancer compounds glucobrassicin, neoglucobrassicin, and glucobrassicin-l-sulfonate. They also contain the glucosinolate precursors to the anti-inflammatory compound tryptanthrin.) The leaves and roots have somewhat different actions, so harvest and store them separately. (See the "Preparation and Dosage" section, page 265, for more.)

The root should be harvested in the fall of the first year or the spring of the second. Clean and slice the roots while they are still fresh, as you would carrots. Layer them on a tray and dry out of sunlight in a warm location.

Harvest the leaves from the first-season and second-season plants prior to flowering (if possible). The plant will releaf as it is harvested. When the plant is cut or the leaves damaged, later leaves can produce up to 65 times as much glucobrassicin. The leaves contain a number of important chemical precursors when they are harvested fresh; *they need to be dried at heat* for the chemicals to convert to their final form. The

Properties of Isatis

Actions

As a broad-spectrum antiviral, isatis is directly virucidal, inhibits viral replication, inhibits virus attachment to cells, inhibits hemagglutination, inhibits viral neuraminidase (equivalent to Tamiflu in potency), inhibits RANTES. It potentiates the effectiveness of viral vaccines and is an immune stimulant, anti-inflammatory, antipyretic, antinociceptive, antiallergenic, tyrosinase inhibitor, antioxidant, antifungal, antibacterial, antiparasitic, antileukemic, antitumor, potent urease inhibitor, potent cross-class serine protease inhibitor, butyrycholinesterase inhibitor, lipoxygenase inhibitor, antiendotoxin, dioxin antagonist (including against TCDD or 2,3,7,8-tetrachlorodibenzodioxin, the most potent).

Active Against

Isatis is a very broad antiviral herb. It is active against influenza viruses A and B (various strains of H1N1 as well as H6N2, H7N3, H9N2), SARS coronavirus, Coxsackie virus (B2, B3, B4), rubella virus, avian infectious bronchitis virus, respiratory syncytial virus, human adenovirus type 3, measles, mumps, varicella virus (chicken pox/shingles), Epstein-Barr, hepatitis B, herpes simplex virus 1, cytomegalovirus, hemorrhagic fever with renal syndrome (HFRS) virus, porcine reproductive and respiratory syndrome virus, swine pseudorabies virus, Newcastle disease virus, goose parvovirus, and porcine parvovirus.

It has some antimicrobial actions as well, against *Staphylococcus aureus*, *Toxoplasma gondii*, *Plasmodium falciparum*, *Leishmania* spp., *Pseudomonas aeruginosa*, *Trichophyton schoenleinii*, *Aspergillus niger*, *Candida albicans*, *Trichophyton simii*, *Macrophomina phaseolina*, *Bacillus pasteurii* (a.k.a. *Sporosarcina pasteurii*), leukemic and liver cancer cells, and possibly other cancers. Alcohol/water extracts of *Isatis microcarpa* (dried) leaves are active (in vitro) against *Bacillus subtilis*, *B. sphaericus*, *Staphylococcus aureus*, *Pseudomonas* spp., *E. coli*, *Salmonella* spp., *Aspergillus niger*, *A. flavus*, *Fusarium oxysporum*, *Alternaria tenuis*, *Microsporum fulvum.* Water extracts of the root are active against *Staphylococcus* spp., *Bacillus subtilis*, *E. coli*, *Salmonella typhi*, *Streptococcus* spp., and *Haemophilus influenzae.*

The isothiocyanates in the plant, especially phenethyl isothiocyanate, are potently inhibitory of *Clostridium difficile*. (Some monographs on isatis list a number of other organisms against which the herb is supposedly effective, e.g., *Neisseria* spp. The data they cite comes from Chinese texts that don't

identify which plants were tested—see the section on traditional Chinese medicine, page 276.)

Use to Treat

The herb is strongly specific for influenza (all strains irrespective of source), SARS, all primary respiratory virus infections, viral pneumonia, meningitis, pseudomonas lung infections, scarlet fever, sore throat, laryngitis, tonsillitis, Epstein-Barr (especially with acute-onset sore throat), gastroenteritis, hepatitis, bacterial conjunctivitis (as eyedrops), leukemia, chicken pox, shingles—generally, any viral infection including encephalitis.

Isatis is strongly active against paramyxoviruses, which include respiratory syncytial virus, mumps, measles, and Newcastle disease virus. Although not tested, it seems to be broadly active against other members of this group (rinderpest, canine distemper, metapneumovirus, Hendra virus, Nipah virus, morbillivirus) and is being used in veterinary practice with good effect for canine distemper, rinderpest (before eradication), and Newcastle disease. The herb is widely used in China for viral hepatitis but I have located only two tiny studies on its activity in this regard.

The herb is also widely used for encephalitis in China with apparently good results, though I can find nothing testing the antiviral activity of the herb against any encephalitis viruses. However, the herb is strongly active against the rubella virus, which is a member of the Togaviridae family of viruses, which also includes a number of encephalitis viruses. There are some clinical studies of the herb being used for encephalitis B (Japanese encephalitis virus) in China with very good outcomes (see the “Scientific Research” section, page 277, for more). And the herb is high in kaempferol, common in many plants, that is specifically, and strongly, active against the Japanese encephalitis virus. Its history of use in China for these viral diseases strongly supports the herb’s use in these conditions. I would use it as an adjunct for *any* encephalopathy of viral origin.

Other Uses

The plant has a very long history as a dye plant for making an indigo dye for cloth. There is a very good description of how to make the dye from fresh plants by Teresinha Roberts at http://www.woad.org.uk/html/extraction.html. The same dye was sometimes used for tattooing and also body paint by the Picts and the Bretons (Mel Gibson in that movie).

Finding It

Plant it, you won't regret it (though antiphytoimmigrants probably will if they find out). It will supply potent medicine for you and your family indefinitely as it reseeds easily once established. Or if you live in the right region you can wildcraft it; the invasive plant societies will love you for it. Seeds are available from Strictly Medicinal Seeds (https://strictlymedicinalseeds.com).

You can get bulk isatis root, powder, cut and sifted, or concentrated from 1st Chinese Herbs (https://1stchineseherbs.com). They carry the leaf as well. You can also get a very nice tincture of the root from Sage Woman Herbs (https://www.sagewomanherbs.com).

most important end-product chemicals are tryptanthrin and indirubin. Tryptanthrin is a potent anti-inflammatory compound that strongly inhibits prostaglandin and leukotriene synthesis—it is also potently antiparasitic against toxoplasmal, malarial, and leishmanial parasites. It is found in much higher levels in dried leaves than in fresh. Indirubin is potently anti-inflammatory as well, although in different ways. It is strongly cytotoxic to leukemia cells and is very virucidal. Indirubin is three to five times higher in the dried leaf than in the fresh.

Traditionally, the leaves are harvested, then allowed to dry in the sun for several days, then brought inside to finish drying. The most tryptanthrin is produced when the plants are dried at around 100°F (40°C).

Bag both leaves and roots, separately, in plastic when completely dry. Store in plastic tubs out of the sun. Leaves will last several years, roots much longer. If you can't store this way, replace the leaves yearly, the roots every other year.

A Eurasian rust fungus has been imported by the phytopolice to try to kill the plants off. Check any plants you harvest and skip those with the rust fungus present. You will know it when you see it: The leaves look sick, turning brown, spotted, shriveling.

Plant Chemistry

More than 65 nonvolatile plant compounds have been identified in the leaves of isatis: alkaloids, flavonoids, fatty acids, porphyrins, lignans, carotenoids, glucosinolates, and cyclohexenones. And

another 70 volatile compounds as well: aliphatic hydrocarbons, acids, alcohols, aldehydes, esters, aromatic aldehydes, ethers, furans, isothiocyanates, thiocyanates, sulfurated compounds, nitriles, terpenes, sesquiterpenes—the usual suspects. The isothiocyanates account for about 40 percent of the total volatile fraction.

Isatis also contains indican, isatin, isatisine A, indirubin, bisindigotin, kaempferol, indigotin, epigoitrin, isatinones A and B, trisindoline, salicylic acid, syringic acid, benzoic acid, gamma-linolenic acid, indolin-2-one, anthranilic acid, 3′-hydroxyepiglucoisatisin, epiglucoisatisin, various flavone C-glucosides, various sphingolipids, mannitol, various glucopyranosides, indolinone, indigo, alpha-linolenic acid, cytidine, hypoxanthine, uridine, xanthine, guanosine, L-pyroglutamic acid, sinigrin, uracil, beta-sterol, daucosterol, o-aminobenzoic acid, glucobrassicin, neoglucobrassicin, glucobrassicin-l-sulfonate, along with several hydroxycinnamic acids and, as usual, a whole bunch of other stuff. There is 20 times more of the cancer-preventing glucobrassicin in isatis than its relative broccoli.

Traditional Uses

Isatis has been used for millennia in Asia as a medicinal and has been cultivated since Neolithic times elsewhere for its use in textiles, body paints, inks, and medicine. It was widely cultivated throughout Europe until the early twentieth century when chemical dyes replaced the need for natural indigo sources. The name of Glastonbury, a town in Somerset, England, is thought to mean "place where woad grows." Many Neolithic sites contain isatis remnants, including the cave of l'Audoste in Bouches-du-Rhône in France, and the Iron Age settlement of Heuneburg in Germany. Isatis seed impressions have been found on many examples of ancient pottery. Egyptian mummy wrappings were sometimes dyed with the plant. Dye shops with the remains of isatis plants have been found in ancient Viking settlements. The author of the Lindisfarne Gospels used a woad-based pigment for his blues. Deep blue dye plants were very rare in the ancient world and the color was highly prized.

Since woad is biodegradable and renewable it is beginning to be commercially grown again to make both ink and dye (for inkjet printers and small craft dyers).

AYURVEDA

Isatis is traditionally used in Ayurvedic practice but I can find little on it. Used as a digestive tonic and for GI tract problems.

TRADITIONAL CHINESE MEDICINE

Isatis has been used for millennia in China. The leaves of the plant are referred to as daqingye, the root as banlan'gen; they are considered to be close but somewhat different medicinals with slightly different actions. However there is a problem in extrapolating from Chinese studies of the herb (a point rarely made when citing the Chinese studies). The problem is that four different plants (all from different genera) are referred to as daqingye in Chinese medicine, and two different ones as banlan'gen. While the plants are used interchangeably with each other, the problem is that the early Chinese clinical trials with the plants did not differentiate species in a number of instances. Later studies are much better.

The leaves (daqingye) are used as a bitter, cold herb, anti-inflammatory, detoxicant, for reducing fever, and for removing heat from the blood. It is considered specific for fever, colds and flu, maculae, papulae, pharyngolaryngitis, parotitis, encephalomeningitis, encephalitis B, erysipelas, carbuncle. It is considered good for headache and sore throat. The root is considered to be bitter, cold, with latent-heat-clearing properties, antipyretic, detoxicant, and anti-inflammatory, and for clearing heat from the blood. It is used for erysipelas, macular eruption due to pathogenic heat, loss of consciousness, hemoptysis, pharyngitis, mumps, conjunctivitis.

The Chinese used the leaf decoction to good effectiveness in treating the SARS outbreak there several years ago. It is now widely used for influenza and viral pneumonia, hepatitis, mumps, encephalitis, and gastroenteritis.

WESTERN BOTANIC PRACTICE

Isatis has a long history of use in Europe, at least as far back as the fifth century BCE. Hippocrates recommended the plant for treating wounds, ulcers, and hemorrhoids. Galen and Pliny recommended it as well. In the late Middle Ages it was used for snakebites, wounds, and inflammation. The American Eclectics didn't use it much, mostly as a vulnerary and

styptic. It has only just recently emerged into Western awareness as a medicinal, mostly for its antiviral properties. As yet, it is still little understood by American herbalists in spite of its being an invasive in the United States, and those who do use the plant generally use it the wrong way. They use the root as the main medicinal instead of the leaf, which is more active, especially against viral infections. Most use the plant as tablets or capsules even though it is not very active in those forms—water extracts, for example, are significantly more effective in stimulating immune responses than both high-concentrate alcohol extracts and the herb in solid form.

Many American herbalists, regrettably, don't know the plant at all.

Scientific Research

There have been a number of good clinical trials in China on the use of the herb for various things; I am not going into any depth on the ones that don't make a distinction as to which plant was used. Trials where the plant (leaf) is not clearly identified were conducted with upper respiratory infections, influenza, mumps, measles, infectious hepatitis, and infectious lymphocytosis. Trials where the root is not identified occurred with chicken pox, encephalitis B, hepatitis, mumps, influenza, infectious mononucleosis, herpes simplex, herpes zoster, pityriasis rosea, verruca plana, cerebral meningitis, diphtheria, and fulminant conjunctivitis.

Given the many later studies (in vitro, in vivo, human) that show that isatis is active against many of these organisms and/or conditions, there is good reason to believe that it was isatis that was used in some or all of the studies.

Isatis tinctoria leaf was used to treat patients with encephalitis B (Japanese viral encephalitis). Headache and other symptoms were sharply reduced, and the mortality rate was decreased in both mild and serious cases—in critical patients, Western intervention was needed along with the herb in order to prevent death.

Sixty people with rubella were randomly assigned to two groups. One received a combination formula of isatis root, milkvetch root, and basket fern, while the control group received ribavirin—both for 20 days. The isatis formula and ribavirin were effective for both groups; the isatis group responded more quickly to treatment.

Twenty healthy people experienced induced contact dermatitis; they were then treated with a variety of isatis extracts as well as pure tryptanthrin. The isatis extracts were more effective than the tryptanthrin in resolving the dermatitis.

A randomized, double-blind, parallel study was conducted with 200 people suffering from bacterial conjunctivitis. Isatis root eyedrops were used (versus levofloxacin) to treat them. The drops were administered six times daily; 90 percent were cured.

Twenty patients with head or neck cancer were split into two groups in order to test isatis root for the treatment of radiation-induced mucositis. The first group received normal saline, the second gargled, then swallowed an isatis root decoction. Those receiving the decoction had significantly reduced severity

of mucositis and anorexia and less swallowing difficulty. This study echoes other reports that the root decoction heals ulceration in and regenerates mucous membranes.

Purified extracts from isatis—indirubin and meisoindigo (an indirubin metabolite)—were used to successfully treat chronic myelogenous leukemia.

In vivo trials with isatis leaf in rats found that the herb was highly effective in treating chronic pseudomonas lung infection (similar in its dynamics to cystic fibrosis). The herb reduced the incidence of lung abscesses, decreased the severity of macroscopic pathology in lung tissue, and altered the inflammatory response in the lungs from an acute inflammation dominated by polymorphonuclear leukocytes to a less-intense chronic type inflammation dominated by mononuclear leukocytes.

In vivo trials with mice found that isatis enhanced the protectiveness of viral vaccines (foot and mouth disease). In other trials, extracts of isatis leaf reduced induced inflammation in mice. Both topical and oral ingestion were effective; purified tryptanthrin extracts were not effective. (Unusual extracts were tested: supercritical CO_2 and dichloromethane.) An in vivo study with mice found that dichloromethane extracts of isatis leaf were effective as an anti-inflammatory in the treatment of arthritis. And extracts of isatis leaf were found to inhibit allergen-induced airway inflammation and hyperreactivity in mice.

Isatis root extracts were found to be highly protective of mice after total-body irradiation, modulating inflammation and reducing tissue damage. And when endotoxins from Gram-negative bacteria were injected into rabbits, administration of an extract from isatis root (o-aminobenzoic acid) reduced fever and destroyed 84 percent of the endotoxins. Deaths dropped from 70 percent to 20 percent.

Isolated fatty acids (compound K) from *Isatis tinctoria* leaf prolonged survival of cardiac allografts in alloantigen-primed mice. Compound K, when combined with tacrolimus, significantly inhibited heart transplant rejection in mice.

An isolated constituent of isatis, indirubin, and an indirubin metabolite, meisoindigo, are used as a combination therapy in the treatment of myelogenous leukemia in China. They have been found to be antiproliferative and cytotoxic to cancer cells, and in clinical use they have extended survival times considerably, inducing hematologic remission.

Scores of studies have been conducted on the antiviral, antibacterial, antifungal, and cytoinhibitory actions of both the leaf and root. Crude extracts of isatis (and various isolated constituents) have been found more effective than ribavirin in their antiviral actions on Coxsackie viruses. They are as effective as Tamiflu (in vitro) in inhibiting viral neuraminidase. (Neuraminidases are enzymes that are essential for viral entry into the host cells.) Tamiflu acts as it does because it inhibits the enzyme that allows influenza viruses to enter cells to replicate.

Several compounds in the plant have been found to be potent urease inhibitors. Urease is an enzyme found in many bacteria, yeast, and fungi. A urease inhibitor is, in essence, an antimicrobial against microbes that need that enzyme to function (such as some of the mycoplasmas).

Tryptanthrin (especially), gamma-linolenic acid, and an indolin-2-one derivative are highly anti-inflammatory, inhibiting COX-2, 5-lipoxygenase (5-LOX), the expression of inducible nitric oxide synthase (iNOS), human neutrophil elastase, and the release of histamine from mast cells. Indirubin is a potent inhibitor of cyclin-dependent kinase 5 (CDK5), glycogen synthase kinase 3ß, and inflammatory reactions in delayed-type hypersensitivity.

Licorice

Licorice is an unusual medicinal. It is potently antiviral, moderately antibacterial (but fairly strong against a few bacterial species such as *Staphylococcus* and *Bacillus* spp.), moderately immune potentiating, and a very potent synergist. So, where to put it? (Throws dart.) Ahhh, the antivirals.

Licorice should also be considered a primary synergist plant. *It should rarely be used alone or in large doses for extended periods.*

Family: Leguminosae

Species used: Primarily *Glycyrrhiza glabra*. There are 18 or 20 or 30 species in the *Glycyrrhiza* genus. (So, you're saying taxonomy is a *science*?) They are native to Europe, North Africa, Asia, Australia, North and South America. All species have been used medicinally but the two most common are *G. glabra*—the European licorice—and *G. uralensis*—the Chinese. (Though the Chinese primarily use *G. glabra* now, and grow it extensively, because it generally contains the most glycyrrhizin.)

The Russian licorice *G. echinata* is often used in that region and other sweet licorices such as *G. inflata* and *G. eurycarpa* are used wherever they grow. The American licorice *G. lepidota* is rarely used these days in spite of its wide native range but was frequently used as a medicinal by the indigenous peoples in the Americas. (And, yeah, they used it just the same way.)

In this section I will talk mostly about *G. glabra* as it is the most commonly used medicinal species, referring to it as "the plant" or "licorice," sometimes as "it." If I talk about another species' actions, I will usually list it by name.

Common names: Licorice in the West, gan cao in China, mulathi in India, kanzou in Japan, where it is a prominent herb in Japanese kampo (traditional) medicinal practice.

Part Used

The root. The leaves have similar but much milder actions than the root.

Preparation and Dosage

Used as tincture, as tea, in capsules. Again: This herb is best used with other herbs in a combination formula.

One of the primary things to keep in mind when using licorice is that the higher the glycyrrhizin content, the more antiviral the herb will be. If using the herb as an antiviral you *should not* use deglycyrrhized licorice.

There is a lot of variety in the glycyrrhizin content of licorice roots. It varies depending on species as well. In the Japanese pharmacopoeia the glycyrrhizin content of the root has to be at least 2.5 percent, i.e., 25 mg per gram of root. In the Chinese (as well as under WHO guidelines) it has to be 4 percent, that is, 40 mg per gram of root. Unfortunately, in the United States growers don't test the glycyrrhizin (a.k.a. glycyrrhizic or glycyrrhizinic acid) content of their harvested roots. So there is no way to know how much is in there. I rarely am a fan of such testing but in this case I think it warranted for two reasons: 1) It will be easier to create proper dosages of the herb in practice and 2) it makes it easier to determine the likelihood of side effects and how to dose the additives that, if taken with the herb, will reduce or eliminate the chance of those side effects.

Glycyrrhiza uralensis generally has less glycyrrhizin than *G. glabra* though tests have shown that Mongolian varieties of *G. uralensis* run higher, from 27 to 58 mg/g though the low end is more common. I would not use *G. uralensis* for treating viruses unless that was all you had; in fact, most species roots run under 25 mg/g. *G. glabra* really is the way to go on this one.

Because the Chinese actually have to create 4 percent product by law, I would tend to buy imported roots if you really want to make sure you have at least that level of glycyrrhizin. They do sometimes import bad product into the United States but assuming that they have not in this case, if you buy from a Chinese herb supplier such as 1st Chinese Herbs (https://1stchineseherbs.com) you would theoretically be getting around a 4 percent root.

To begin, the roots you use must be 3 years or older (4 years if you are using *G. uralensis*). The glycyrrhizin content of younger roots is very low compared to the older roots.

There are a number of beliefs about how the root should be prepared. Some people feel that an infusion or decoction, or even a concentrated decoction, of the root (with the later addition of 20 percent alcohol to stabilize it) produces the best extract. I am not so sure. Studies in China and Japan have found that the glycyrrhizin is best extracted in a water and alcohol blend, not in water. In fact a mix of 50 percent alcohol and 50 percent water produced the most efficient extraction in all the studies I have read. So, I am going to take a wild guess and go with that here.

TINCTURE

Use the dried root, in a 1:5 herb:liquid ratio, with the liquid being 50 percent alcohol and 50 percent water. In general, the glycyrrhizin extracts better in lower-pH water (somewhat acidic), so you might want to add a tablespoon of vinegar to your water.

Dosage: 30–60 drops up to 3x daily. In acute conditions: ½ teaspoon 3–6x daily, blended with other herbs, and generally for a maximum of 6 weeks at this dose and only if you take the additional supplements described in the "Side Effects and Contraindications" section (page 283).

A company called Standard Process makes a standardized 1:1 tincture that is very reliable as regards the glycyrrhizin content. Some sources sell it by the 500 ml bottle, that is, about 16 ounces. It will last awhile; the pricing is actually reasonable at that size. Average dosage is 2.5 ml daily, which will give 75 mg glycyrrhizin. Dosage for acute viral infections would be 2.5–5 ml 3x daily (see the discussion of Japanese, Chinese, and WHO guidelines at left). Note: This brand is not easily found but you can find it if you google "licorice high grade 1:1." If you actually do want a reliable product as regards glycyrrhizin content, I would suggest this one.

INFUSION

Combine ½ to 1 teaspoon of powdered root with 8 ounces water, simmer for 15 minutes, uncovered, then strain. Drink up to 3 cups a day. In acute conditions, drink 1 cup every 2 hours.

DECOCTION

The traditional preparation in Japan (standard now in the Japanese pharmacopoeia) is as follows: 6 grams powdered root in 500 ml (about 16 ounces) water; bring to a boil, uncovered, and let boil moderately until the liquid is reduced to 250 ml. (This will be fairly mucilaginous.) Then add enough water to bring the volume up to 1,000 ml. Drink throughout the day. Tests in Japan found that this preparation will have about 50 mg/g of glycyrrhizin. (I assume here that the powdered root they used conformed to the Japanese standard of 2.5 percent glycyrrhizin.)

CAPSULES OR POWDER

Take 4,000 mg (i.e., 4 grams) daily in three divided doses. Note: ¼ teaspoon of the powder is about 2,000 mg. *However* . . . Chinese doses run high, as they tend to do, up to 9 grams daily. Oddly, the WHO monograph lists the dosage range as 5–15 grams daily, somewhat higher. Assuming that you are getting a 4 percent glycyrrhizin content in the root, that will give you 200–600 mg of glycyrrhizin daily, which is the WHO suggested limit. The EU standards suggest that people not consume any more than 100 mg of glycyrrhizic acid per day. In Japan glycyrrhizin intake is suggested to be kept to 200 mg per day. So, as usual, you have a range to choose from. *If* you are struggling with a severe viral infection for which this herb is specific, especially if it is severe encephalitis, there is no reason, keeping the contraindications in mind, to not use the higher WHO dose during limited treatment of 4 to 6 weeks' duration. Again, please keep in mind the side effects and contraindications.

Note: You can get licorice root standardized to either glycyrrhizin or glycyrrhic acid content if you look for it. Douglas Labs makes a 500 mg capsule standardized to contain 12 percent glycyrrhizin per capsule. This will give you 60 mg glycyrrhizin per capsule. For acute viral infections, if you are using this brand, take 1 or 2 capsules 3x daily. Note: Make *sure* you don't accidentally buy the deglycyrrhized stuff by accident; they make both kinds. It is a fairly easy brand to find online.

There are a number of different brands that have licorice root standardized for anywhere from 12 to 25 percent glycyrrhizic acid. I would take these similarly during acute viral infections, looking to get up to 600 mg daily of glycyrrhizic acid.

Side Effects and Contraindications

Generally, licorice is nontoxic, even in high doses. However, long-term use, especially if you use the herb as a single (rather than in combination), and most especially if you use large doses, can cause a number of rather serious side effects. Even the use of a tea over several years, due to the rather good range of effects the herb has, can do it, and every now and then it does. (This makes antiherb proponents *very* excited.)

Note: *This herb should rarely be used in isolation or in large doses or for long time periods*—that is, longer than 4 to 6 weeks. (However, see the comments in the next paragraph.) The side effects can be severe: edema, weak limbs (or loss of limb control entirely), spastic numbness, dizziness, headache, hypertension, hypokalemia (severe potassium depletion)—especially in the elderly. Additional problems are decreases in plasma renin and aldosterone levels, and at very large doses decreased body and thymus weight and blood cell counts. Essentially, this complex of symptoms is a condition called pseudoaldosteronism, which licorice can and indeed does cause if you take too much of it for too long. However . . .

Taking licorice along with some other supplements *can* reduce or even eliminate the tendency of the herb to produce pseudoaldosteronism. There is an intravenous form of glycyrrhizin commonly used in China that contains 40 mg aminoacetic acid (glycine), 2 mg L-cysteine, 1.6 mg sodium sulfite, and 4 mg monoammonium glycyrrhizinate (glycyrrhizin) per 2 ml vial. Normal dosing is 40–60 ml IV and up to 100 ml. The oral therapeutic dose is as high as 200 mg daily. This combination eliminates pseudoaldosteronism as a side effect. You can add both glycine and L-cysteine to your protocol to limit the potential for pseudoaldosteronism if you are taking large doses of licorice for extended periods. (Glycine, minimum 2,000 mg daily; L-cysteine, minimum 500 mg daily.) The addition of potassium (5,000 mg daily) will also help prevent the hypokalemia. Again: Licorice should be taken in combination with other herbs—this reduces the tendency for side effects by itself. And, if you do need to take largish doses of licorice, even with other herbs, for severe viral infections, please add these supplements to your regimen and carefully monitor for side effects.

Because of licorice's strong estrogenic activity it will also cause breast growth in men, especially when combined with other estrogenic

herbs. Luckily all these conditions tend to abate within 2 to 4 weeks after licorice intake ceases. Caution should be used, however, in length and strength of dosages.

A number of studies have found that large doses of licorice taken long term during pregnancy have detrimental effects on the unborn children. Low doses are apparently safe. Again, this plant should not be used in large doses or for lengthy periods of time *especially if you are pregnant*.

The herb is contraindicated in hypertension, hypokalemia, pregnancy, hypernatremia, and low testosterone levels. However, for short-term use in those conditions (10 days or less), in low doses combined with other herbs, it is very safe.

Herb/Drug Interactions

The plant is highly synergistic. It is also additive. It should not be used along with estrogenic pharmaceuticals, hypertensive drugs, cardiac glycosides, diuretics such as thiazides, loop diuretics, spironolactone, amiloride, corticosteroids, hydrocortisone.

Alternatives: *Taverniera cuneifolia,* endemic to northeastern Africa and southwestern Asia, has a very similar chemical profile, also contains a large amount of glycyrrhizin, and can be used similarly to commercial licorice.

Habitat and Appearance

The *Glycyrrhiza* genus is a member of the pea family with the usual pea-type leaves—a bunch of oval leaflets running along a central stem. The plants are perennials, can grow to 6 feet in height, and bush out to 3 feet. The plants produce spikes of the usual pea-family flowers during the summer. They range in color from yellowish to blue to purple in the various species. The plant sends out both roots and rhizomes, the roots thick and fleshy, up to 4 inches in diameter, going as deep as 3 feet. The creeping rhizomes spread out from the primary root, up to 26 feet in length, often sending up shoots of new plants far from the original. The roots and rhizomes of the cultivated species are light in color, the wild species darker. The inside of the root of most species is yellowish, and, in the commercial species at least, quite sweet. The native American species is not very sweet, though a lot of sources say it is (I first tasted it in 1987,

still waiting for that sweet taste to emerge on my tongue). The American species, though low in sweetness, possesses many of the same medicinal actions, according to most sources, as the more prominent medicinal species. (Though, since glycyrrhizin is considered to be the primary active constituent *and* is the source of the sweet taste in licorice, I am confused by that assertion.) I've not encountered any of the other, less common species in practice.

The licorice flowers mature into clusters of spiky brown seed capsules about the size of a grape (at least in the American species—the only one I have seen).

The genus ranges from semiarid desert to lush, wet climes such as Yorkshire, England, and from sea level to 8,500 or so feet in altitude. When wild, the plants often like growing along waterways in sandyish soil. The American species is endemic throughout Canada and most of the United States excluding the Southeast. *G. glabra* is cultivated in many places in the Americas but has escaped and can be found here and there in California, Nevada, and Utah. I can't find a record of any wild species in mid- to southern Africa (though it is most likely grown there) but the genus seems to have spread pretty much everywhere else. If you look around you will probably find a licorice native in your ecorange someplace.

Cultivation and Collection

The plants grow fairly easily from root cuttings; the seeds are more demanding. The seeds need to be stratified for several weeks, then scarified and soaked for 2 hours in warm water before sowing if you want an easy germination. Treated seeds will germinate at about an 80 percent rate, untreated at around 20 percent. Once started, the plants are pretty intent on remaining and spreading wherever they want to. Make sure you want it where you plant it—you won't be able to get rid of it if you change your mind. A few places here and there consider it an invasive because, well, it is.

Both the European and Chinese varieties warrant planting in the wild and letting them go; they are well able to look after themselves if released from captivity. As they are a major medicinal, the more they spread, the better off we will be.

The plants like a free-draining friable soil with a pH between 6 and 7 but they can take on a greater range than that and do quite well. They are drought tolerant and like the sun but do need a bit of water; they often grow wild along streambeds, where they are very tenacious.

It takes a few years for the plants to establish themselves (3 years is a good minimum period of time; earlier than that and the glycyrrhizin content in the roots is too low) but once they do, you will be able to harvest from them pretty much forever. You will rarely, if ever, be able to dig the entire root system of an established plant, so it will continue to grow and spread from what is left. Commercial growers generally achieve somewhere between 15 and 50 tons per 2.5 acres of roots once the plants have matured. The older the plants and the deeper the dig, the bigger the yield. The plants produce a lot of root mass. You can get enough medicine for an entire family from just one established plant, pretty much forever.

If you are growing *Glycyrrhiza glabra* harvest the roots after 3 years. The glycyrrhizin content is highest in August/September. The larger the roots, the higher the content. (The greatest concentration of glycyrrhizin in this species' roots, when it was grown hydroponically, was produced by the use of a quarter unit of Hoagland solution.)

If you are growing *G. uralensis* (the other major species used as medicine), harvest the roots of 4-year-old plants and older in mid-July, which is when the glycyrrhizin content is highest for this species. The glycyrrhizin content begins to fall in early August, becoming very low by November. It begins to rise again in March, reaching its peak between late June and mid-July.

Once you've harvested them, dry the roots out of the sun. The larger roots should be cut into smaller sections before being dried. The larger the root diameter, the higher the glycyrrhizin content.

In general, studies have found that wild plants are higher in glycyrrhizin than domesticated plants. *G. uralensis* tends to have much less glycyrrhizin than *G. glabra*. Normally it runs 1 to 4 mg/g but some studies have shown it to be as low as 0.52 percent by weight of the root.

Plant Chemistry

There are hundreds of compounds in licorice, many of which have been intensively studied. These include triterpenoids, polyphenols,

Properties of Licorice

Actions

As a major broad-spectrum antiviral, licorice prevents viral replication across a wide range of viruses and inhibits viral growth, viral uptake, neuraminidase in numerous influenza strains, virion-associated RNA-dependent DNA polymerase, casein-kinase-II-mediated activation of HIV-1 enzymes (including HIV-1 protease and reverse transcriptase), viral antigen expression of human cytomegalovirus, and protein-kinase-A- and casein-kinase-II-mediated phosphorylation of the ICP27 regulatory protein of HSV-1. It inactivates virus particles, strongly inhibits viral cytokine cascades, stops the ballooning degeneration of fused cells, modifies the intracellular transport and suppresses sialylation of hepatitis B virus surface antigen, inhibits RANTES secretion, lowers lipid bilayer membrane fluidity, thus stopping the virus-induced development of membrane pores through which the viruses can enter host cells. It is strongly virustatic, somewhat virucidal.

The herb has other actions as well:

Adrenal cortex stimulant
Adrenal tonic
Analgesic
Antibacterial
Anticancer/tumor inhibitor
Antihemolytic
Antihyperglycemic
Anti-inflammatory
Antioxidative
Antispasmodic
Antistressor
Antitussive
Antiulcer
Cardioprotective
Demulcent
Estrogenic
Expectorant
Gastric secretion inhibitor
Hepatoprotective
Immunomodulant
Immunostimulant
Laxative (gentle)
Mucoprotective
Prevents biofilm formation
Protects from effects of radiation exposure
Smooth muscle relaxant
Stimulates pancreatic secretions
Synergist (potent)
Thymus stimulant
Tyrosinase inhibitor
Xanthine oxidase inhibitor

As an immunostimulant, it stimulates interferon production, enhances antibody formation, stimulates phagocytosis. As an immunomodulant, it will reduce interferon-gamma levels if they are high and upregulate them if they are low.

Licorice is a fairly potent synergist. It has been found to potentiate the action of antituberculosis drugs, increasing positive outcomes in treatment. It potentiates the action of oseltamivir against resistant influenza strains. It reduces toxicity and potentiates other medications in the treatment of

Continued on next page

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rheumatoid arthritis. Licorice potentiates the effect of the neuromuscular blocking agent paeoniflorin, enhances the solubility of compounds from other plants (during tincturing) by a factor of up to 570 (e.g., the sapogenin isoliquiritigenin and the saikosaponins from ginseng), and increases the immune-stimulating action of other herbs such as *Echinacea purpurea* significantly.

It takes 4 to 8 hours (depending on what and how it is taken) for the glycyrrhizin to reach maximum serum concentration after oral ingestion; then it is slowly excreted and eventually eliminated entirely about 72 hours after ingestion. (Most of it is gone after 24 hours.) It stays in the body a long time.

Active Against

Licorice is broadly antiviral. It is active against a wide range of viruses through multiple mechanisms. It strongly inhibits the ability of many viruses to create the membrane pores through which the viruses then enter cells. This slows or even ends the viral infection right there. For other viruses, it is directly virucidal, and for others it stimulates the host immune system specifically to attack the invading virus. Licorice, and its constituent glycyrrhizin, are especially effective against enveloped viruses, and this covers a wide range: herpesviruses, poxviruses, hepadnaviruses, flaviviruses, togaviruses, coronaviruses, hepatitis D, orthomyxoviruses, paramyxoviruses, rhabdoviruses, bunyaviruses, filoviruses, and retroviruses. It is not active against all viruses in these groups but it is against many of them. Chinese skullcap and licorice in combination should be considered the main antivirals to use for any viral infection.

The viruses that licorice has been found effective for, irrespective of mechanism, are influenza A (various strains, H1N1, H2N2, H5N1, H9N2, novel H1N1, oseltamivir-resistant novel H1N1, and so on), SARS-related coronavirus (FFM-1, FFM-2—multiple isolates), respiratory syncytial virus, parainfluenza virus 3, Japanese encephalitis virus (multiple strains), tick-borne encephalitis, West Nile encephalitis, yellow fever, dengue, viral pneumonia, avian infectious bronchitis virus, enterovirus 71, rotavirus, adenovirus type 3, Coxsackie B3, Newcastle disease virus, vaccinia virus, vesicular stomatitis virus, HIV-1, cytomegalovirus, herpes simplex 1 and 2, hepatitis (A, B, C, E, and most likely D), varicella zoster, Epstein-Barr, poliovirus (wild and vaccine types 1, 2, 3), measles, Chandipura virus, pseudorabies virus, vovine immunodeficiency virus, murine retrovirus, and porcine reproductive and respiratory syndrome virus.

Glycyrrhizin has been found to inhibit cellular infection by 11 different flaviviruses, including some of the most damaging: dengue, Japanese encephalitis, tick-borne encephalitis, and yellow fever.

The herb does have a good range of other antimicrobial actions as well, against:

Arthrinium sacchari
Bacillus coagulans
Bacillus megaterium
Bacillus stearothermophilus
Bacillus subtilis
Candida albicans
Chaetomium funicola
Clostridium sporogenes
Enterococcus faecalis
Enterococcus faecium
Enterotoxigenic *E. coli*
Haemophilus influenzae
Helicobacter pylori
Klebsiella pneumoniae
Mycobacterium tuberculosis
Plasmodium spp.
Salmonella paratyphi
Salmonella typhi
Salmonella typhimurium
Sarcina lutea
Shigella boydii
Shigella dysenteriae
Staphylococcus aureus
Streptococcus lactis
Streptococcus mutans
Streptococcus sobrinus
Toxocara canis
Trichophyton mentagrophytes
Trichophyton rubrum
Vibrio cholerae
Vibrio mimicus
Vibrio parahaemolyticus

Use to Treat

Influenza (all types), respiratory viral infections, pneumonia (viral or otherwise), meningoencephalitis, SARS, any viral encephalitis, and as an adjunct in all antiviral herbal combinations. It is a synergist with other herbal medicines, increasing their potency, adds immune-boosting activity, and has numerous other supportive actions specific for many different types of viral infections.

Use licorice also to treat oral bacterial problems, for gums and mucous membranes, as an adjunct for bacterial infections, especially of the GI tract and respiratory tract, especially if there is cramping or ulceration.

Note: Licorice should be used in combination rather than alone. (See the "Side Effects and Contraindications" section, page 283.) I would not recommend this plant be used as a single medicinal.

Other Uses

As a sweetener. *Glycyrrhiza glabra* is also a potent plant remediator for reclaiming saline-heavy soils.

Finding It

You can buy very good organic licorice root from Pacific Botanicals (https://www.pacificbotanicals.com), my preferred source for high-quality grown herbs. Regrettably there is no way to determine the glycyrrhizin content of their product. (I still like it though; it *feels* good.) 1st Chinese Herbs (https://1stchineseherbs.com) has Chinese-grown *G. glabra*, which, while not organic, is definitely supposed to be at least 4 percent glycyrrhizin by weight. Seeds can be had from Strictly Medicinal Seeds in Oregon (https://strictlymedicinalseeds.com) or from Richters (https://www.richters.com), an international seed merchant.

Note: Some of the licorice in commerce comes from eastern Europe (which possesses some of the highest levels of soil and air pollution in the world). It makes no sense to buy potentially contaminated herbs to use for their broad-spectrum immune and liver actions.

polysaccharides, essential oils, flavonoids, saponins, and so on. The primary constituent that everyone talks about is glycyrrhizin, which, supposedly—if you believe stuff on the internet—can make up to 24 percent of the root by weight. I have not been able to verify this, even after looking at several hundred studies. The WHO monograph on the plant indicates that the root ranges only from 2 to 9 percent glycyrrhizin and I tend to believe it. (One of my Chinese materia medica references puts the high number at 14 percent.) The largest concentration I can find in any journal papers from China or Japan on direct study of plants in the field is 6 percent. That was in Uzbekistan, where it ran from 4 to 6 percent by weight in the root. Plants in Spain have run from 0.4 percent to 4.4 percent; in Italy 1.6 to 3 percent. In one paper, for their research, the authors bought licorice root that contained 7.64 percent glycyrrhizin from a supplier in Italy. That is the highest value I have seen that could be verified (so far).

In Japan, the root must have at least 2.5 percent, in China 4 percent, to be considered strong enough for medicine. If 24 percent were at all common, if that figure were even real, it seems that those cultures would be looking at higher figures for their traditional practice, considering that they both consider glycyrrhizin to be the active ingredient in the plant and both use the purified extract medicinally. So, no, not 24 percent. I think the WHO monograph is more accurate.

The glycyrrhizin content can vary considerably depending on species used, where it grows, whether domesticated or wild, and when it was harvested. In general, wild *G. glabra* plants are considered to have the highest glycyrrhizin content.

While there are some minor distinctions in the terms, glycyrrhizin is considered to be a synonym for the terms glycyrrhizinic acid and glycyrrhizic acid.

Some other constituents that people find exciting are glabrin A and B, glycyrrhetinic acid, glycyrrhetol, glabrolide, glabridin, glycidipine, isoglabrolide.

Traditional Uses

Licorice has been used as a food plant and medicinal for between four and five millennia. The genus name, *Glycyrrhiza,* is Greek in origin, *glykys* meaning "sweet" and *rhiza* "root." The root's main constituent, glycyrrhizin, is 50 times sweeter than sugar, as seemingly every article on licorice repeats, ad nauseam. All licorice species have been used as medicine wherever they have grown and by every culture that has had access to them.

AYURVEDA

Variously known as mulathi, yasti-madhu, jasti-madhu, madhuka, mithiladki, and so on. The plant is considered cooling, tonic, demulcent, expectorant, diuretic, and a gentle laxative. It's used for treating poisoning, ulcers, diseases of the liver, bladder, and lungs. It is specific for any inflammation in the mucous membranes anywhere in the body. It is used for cough, sore throat, hoarseness, fevers, and as a general tonic in debility from long-term disease conditions, especially those that are pulmonary or of the GI tract. It is considered a synergist, a specific additive to other herbal formulations.

TRADITIONAL CHINESE MEDICINE

Known as gan cao in Chinese medicine, licorice has been used in China for 3,000 years or so. The herb is considered sweet and mild, to regulate the function of the stomach, to be qi tonifying, lung demulcent, expectorant, latent-heat cleansing, antipyretic, detoxicant, anti-inflammatory, spleen invigorative, and it is a synergist in many herbal formulations.

It is used in pharyngolaryngitis, cough, palpitations, stomachache due to asthenia, peptic ulcer, pyogenic infection, ulceration of the skin, hepatitis, encephalitis B, measles, and all types of respiratory infections.

WESTERN BOTANIC PRACTICE

The ancient Egyptians used licorice as a major medicinal; the plant has often been found in their tombs. The Greek Theophrastus in the third century BCE noted the plant's use for asthma, dry coughs, and respiratory problems. The Romans called the plant *liquiritia*, which was eventually corrupted to the word *licorice*. It was a primary medicine in ancient Rome for coughs. It was used throughout Europe as a primary medicinal and although harvested in the wild originally, it has been a main agricultural crop for over a thousand years.

The American Eclectics used it intensively, as did most medicinal practitioners in the Americas. The Eclectics used it for coughs, catarrhs, irritation of the urinary passages, diarrhea, and bronchial diseases. It was an early agricultural medicinal, grown by most people in their medicinal gardens. The indigenous tribes of the Americas used the indigenous species similarly, that is, for sore throat, chest pains, swellings, coughs, stomachache, fevers, toothache, skin sores, spitting blood, and as an antidiarrheal and a general tonic.

Scientific Research

The medicinal species have been intensely studied for years; there are over 1,900 citations on PubMed alone. This look will be brief, as a full monograph would run hundreds of pages. And first, given the nature of *this* book . . .

ANTIVIRAL DYNAMICS, MEMBRANE FLUIDITY

Licorice has strong impacts on viruses across a broad range. Licorice and its (strongest?) constituent glycyrrhizin act against a wide range of viruses, specifically by modulating membrane fluidity in both host and viral cells—the herb lowers membrane fluidity significantly.

Enveloped viruses (herpesviruses, poxviruses, hepadnaviruses, flaviviruses, togaviruses, coronaviruses, hepatitis D, orthomyxoviruses, paramyxoviruses, rhabdoviruses, bunyaviruses, filoviruses, and retroviruses) have a viral envelope surrounding them, covering their protein capsids (i.e., their viral shell). The viral envelopes are generally composed of glycoproteins that identify and bind to receptor cells on the surface of host cells the viruses want to enter. Once the proper receptors are identified, the viruses bind to them, fuse with the host cell, create a pore in the cell, and enter the host cell. Voilà! Infection. The viruses take over the cell they have entered,

reproduce, burst it open, then spread to other cells.

Licorice and its constituents act by inhibiting the ability of enveloped viruses to fuse with host cells, create pores in the host cell membrane, and enter them. It does this by significantly reducing the membrane fluidity of both the host cell and the virus. As little as a 5 percent reduction in host cell membrane fluidity will reduce HIV infection by 56 percent for instance. An increase of 5 percent will enhance infectivity 2.4-*fold*, more than doubling it.

Interesting speculation: Excess cholesterol in the bloodstream serves to reduce host cell fluidity; the most interesting studies on the effects of licorice and glycyrrhizin on viral infection occurred because glycyrrhizin is similar in shape to cholesterol. The researchers postulated that it might act in a number of instances *because* it was reducing membrane fluidity. This stimulates speculation: Are cholesterol-lowering drugs affecting membrane fluidity across the board in those using them, thus increasing viral infections in that group? (Cholesterol-lowering drugs do affect sterol levels in the body and do reduce, in some circumstances, the levels of steroid hormones that the body produces, especially if combined with a low-cholesterol diet.)

Glycyrrhizin is uptaken fairly quickly into the cell (due most likely to its cholesterol-like shape). It diffuses rapidly across the membrane and concentrates on the inner membrane surface (and possibly within the membrane itself), where it causes the cell membrane to become more rigid, significantly reducing the movement of compounds through the membrane. Due to its nature glycyrrhizin is also uptaken by the viruses and incorporated into their viral envelope, which also becomes more rigid. At human body temperature the effects on cell fluidity are significant. Once this occurs, a virus's ability to fuse with a host cell and create a pore in the membrane through which to enter the cell is inhibited.

The degree of inhibition is dose dependent. The more licorice or glycyrrhizin, the more inhibition that occurs. Glycyrrhizin also stops the ballooning degeneration that occurs in virus-infected/fused cells. Tests on influenza viruses, vaccinia virus, herpes simplex virus 1, Newcastle disease virus, measles, HIV-1, and SARS have found that it acts in exactly this way with all those enveloped viruses. (It uses different mechanisms with polio, another enveloped virus type.) Of note: Interferon, whose production is stimulated in the body by licorice, also alters membrane fluidity, as well as being directly antiviral. So, licorice acts through multiple mechanisms on membrane fluidity.

Unfortunately, because of improper use and understanding, much of the licorice root in the United States is deglycyrrhized, that is, the glycyrrhizin is removed. Glycyrrhizin is felt to be the constituent that causes most of the side effects of licorice (see the discussion of side effects on page 283). Please note: Deglycyrrhized licorice is worthless as an antiviral (though it does still work for GI tract problems). If you pay attention to the proper use of the herb and its side effects (really! see the side effects section) and use the plant with awareness you really should not have any trouble with the herb. Oddly enough, the Japanese insist that their use of purified glycyrrhizin has produced very few side effects in clinical practice (yes, there have been some). As one researcher noted: "An advantage of GL [glycyrrhizin] is that it is a broad anti-viral agent with few side effects. Although data about the safety of long-term usage and high doses of GL still need to be collected, the best example of safety is a long history of safe use in clinical settings in Japan."[9] Only in the West would they *remove* what is considered by many to be the primary active constituent of the herb.

Note: In general, licorice is rarely used *by itself* for treatment of disease in the countries that do use herbs as part of their health care system, so there are not many studies on its use *by itself*. Glycyrrhizin is, however, used a lot as a potent antiviral (with other actions as well) and there have been a number of studies on it. In Japan, for instance, glycyrrhizin is widely used in clinical practice, has far fewer side effects than pharmaceuticals, and is extensively studied.

PHARMACOKINETICS

Usually, researchers study isolated glycyrrhizin versus licorice root extract pharmacokinetics, with sometimes a variation here and there. Glycyrrhizin is hydrolized (i.e., *changed*) by human intestinal flora by the bacterial enzyme glucuronidase to glycyrrhetinic acid (GA), which is the primary form of glycyrrhizin that is active in the body. Glycyrrhizin is not normally detected in plasma at any time but GA is and is one of the main compounds tested for when looking at plasma concentrations and the pharmacokinetics of licorice.

Rats and rabbits biologically process the herb and its constituents a bit differently. In rats the amount of GA in plasma is a bit less if they are given the herb extract rather than the purified compound. In rabbits that is reversed. Nevertheless, the use of the root extracts themselves do allow most of the glycyrrhizin to enter the bloodstream.

People's bodies work a bit more like rats in this instance. Licorice extract produces a slightly lower glycyrrhizin (glycyrrhetinic acid) profile than the pure extract itself. Peak plasma concentration with the purified extract takes about 6 hours; with the root extract it is 8 hours. Both then decline slowly over the next 24 hours. The level of other licorice constituents such as glabridin are a bit different. They can be detected in plasma within an hour and reach peak within 4 hours. Then they, too, decline slowly over the next 24 hours.

The constituents in licorice do cross the blood-brain barrier. In rats, glycyrrhetinic acid, the metabolite of glycyrrhizin, can be found in plasma, the brain, and the cerebral spinal fluid after oral administration. The amount of glycyrrhizin (glycyrrhetinic acid) in human plasma increases if licorice extracts are taken with Chinese skullcap (which they should be for treating viral infections).

The constituents in licorice root are altered into potent metabolites by gut bacteria and again by the liver, which sends them back into the GI tract in the bile, where the gut bacteria again alter them. Around and around. This is part of the reason that the clearance time of licorice constituents is so long. It is also why licorice is so good for treating viral liver disease; the constituents concentrate in the liver.

In treating viral diseases that are acute, if the amount of the herb and its constituents is increased to a relatively high level and you keep taking them every day, every 3 to 4 hours, then the amounts in the body stay high, essentially bathing the body in those specific compounds. Many of these are uptaken by cells in the body, decreasing their fluidity, which inhibits viral infection. At the very least, the herb needs to be taken until all the virus particles are eliminated, several weeks at minimum. The continual presence of the constituents in the body, especially in the brain, also reduces inflammation and protects neurons from damage, especially during encephalitis infections.

COMPOUND SYNERGY

Studies with licorice have shown, as they invariably do with every plant studied in this fashion, that the constituents of licorice are highly synergistic. For example, licorice extract is strongly reductive of nitric oxide (NO) and inducible nitric oxide synthase (iNOS) in the body. That is, it lowers inflammation by reducing oxidation, one of the reasons it is good for encephalitis, for instance. *However, glycyrrhizin, by itself, has no effect on NO or iNOS.* Further, a licorice extract with the glycyrrhizin removed has, as the researchers put it, "significantly attenuated" impacts on NO and iNOS; that is, it barely reduces inflammation. But if they add the glycyrrhizin, which has no effect on NO or iNOS itself, back into the extract, the impacts on NO and iNOS return to their previous levels.

ANTIVIRAL ACTIONS

Licorice prevents viral replication across a wide range of viruses, inhibits viral growth, and inactivates virus particles. In vitro studies have found that licorice inhibits influenza A uptake into cells and inhibits RANTES secretion by bronchial cells infected with influenza A. In vitro, glycyrrhizin is more active against SARS-associated coronavirus than ribavirin, 6-azauridine, pyrazofurin, and mycophenolic acid.

In vivo studies found that glycyrrhizin significantly reduced morbidity and mortality in mice infected with lethal doses of influenza virus (H2N2). There were no survivors in the control group; all the licorice-treated mice survived. Pulmonary consolidations and virus titers in the lung tissues of the licorice group were significantly lower than in the tissues of the control group. When splenic T cells from the licorice mice were transferred to nonlicorice mice, survival rate increased to 100 percent.

Another in vivo study found that glycyrrhizic acid inhibited influenza virus and Newcastle disease virus growth in embryonated eggs.

Glycyrrhizin is strongly protective of mice with induced herpes simplex encephalitis. Mortality was significantly reduced (from 60 to 25 percent), and glycyrrhizin directly inhibited the virus replication in vivo and markedly reduced the expression of iNOS, significantly alleviating autoimmune reactions to the disease.

Glycyrrhizin has been found to be 10 times more potent in reducing the infectivity of hepatitis A viruses than ribavirin. Both licorice and glycyrrhizin have been found to irreversibly inactivate herpes simplex viruses.

Glycyrrhizic acid cream, applied six times daily in people with acute oral herpetic infections (HSV1), resolved pain and dysphagia within 24 to 48 hours.

Three HIV patients were given IV glycyrrhizin six times over 1 month. HIV p24 antigen was present in all at the beginning of the trial; by the end it had either decreased or become negative.

In infants infected with cytomegalovirus who were given IV glycyrrhizin, liver enzymes normalized and the virus disappeared sooner than in controls. Oral glycyrrhizin worked similarly.

NEUROPROTECTIVE/CNS ACTIONS

Stronger Neo-Minophagen C, a glycyrrhizin-containing preparation used in Japan for treating hepatitis, showed potent neuroprotective effects after induced middle cerebral artery occlusion in the postischemic rat brain. Motor movement, neurological deficits, and infarct volume all improved.

(Whole licorice preparations are more effective along this line than purified glycyrrhizin.)

Glycyrrhizic acid is strongly neuroprotective in postischemic rat brains via anti-inflammatory activity through the inhibition of HMGB1 phosphorylation and secretion.

Albendazole and diammonium glycyrrhizinate (DG) were used to treat eosinophilic meningitis in mice infected with *Angiostrongylus cantonensis*, a parasitic roundworm that normally causes this condition in people and animals. Albendazole is a pharmaceutical used to kill the worms, DG is a form of glycyrrhizin. When DG was added to the treatment, survival time increased, mortality was reduced, neurological dysfunction was significantly reduced, weight loss decreased, levels of IgE, IL-5, and eotaxin all decreased.

Glycyrrhizin was found to reduce secondary inflammatory processes in mice after spinal cord compression injury. NF-κB, NO, iNOS, and Bax were reduced. Bcl-2 was increased.

An aqueous extract of licorice given to mice (150 mg/kg) significantly improved learning and memory. It also reversed the amnesia induced by diazepam and scopolamine.

IMMUNE IMPACTS

Licorice and glycyrrhizin enhance interleukin-10 (IL-10) production in the body. IL-10 is also known as cytokine synthesis inhibitory factor; it is an anti-inflammatory cytokine. It essentially downregulates other cytokines and blocks NF-κB activity. In other words, if an infectious organism begins a cytokine cascade by initiating NF-κB activity (common), increasing IL-10 will begin to normalize cytokine levels in spite of what the bacteria or viruses are doing. Another component of licorice root, isoliquiritigenin, also has potent effects on cytokines, especially NF-κB. Specifically it blocks the induction of VCAM-1 (vascular adhesion molecule-1), E-selectin, and PECAM-1 (platelet endothelial cell adhesion molecule-1). It interferes with THP-1 monocyte adhesion to TNF-α-activated endothelial cells, and abolishes many of the cytokine effects of TNF-α. It does this by blocking the nuclear translocation of NF-κB, essentially acting as an upstream cytokine cascade blocker in bacteria- or virus-initiated inflammatory processes.

A double-blind, repeated-within-subject, randomized trial with *Echinacea purpurea*, *Astragalus membranaceus*, and *Glycyrrhiza glabra* found that licorice increased CD25 expression on T cells. It also increased CD69, CD4, and CD8 expression on T cells.

SORE THROAT TREATMENT

Forty adults about to undergo elective lumbar laminectomy were split into two groups. One received water as a preoperative gargle, the other water with licorice. The use of licorice gargle performed 5 minutes before anesthesia was effective in reducing or eliminating the incidence and severity of postoperative sore throat in patients.

SYNERGY IN TUBERCULOSIS TREATMENT

Licorice enhances outcomes in the treatment of tuberculosis: A randomized, double-blind, placebo-controlled study with 60 people with sputum positive pulmonary tuberculosis was conducted. They were split into two groups, one

taking placebo, the other licorice—in addition to their regular therapy. Sputum conversion was seen in 80 percent of the licorice group, 70 percent of the placebo group. Fever was relieved in all of the licorice group, 80 percent in the placebo group. Cough was relieved in 96 percent of the licorice group, 81 percent of the placebo group. GI side effects were seen in 20 percent of the placebo group, none of the licorice group. ALT and AST levels were raised in 6 percent of the licorice group, 30 percent of the placebo group. Elevated uric acid in serum was observed in 3 percent of the licorice group, 16 percent of the placebo group.

HEPATITIS

A single compound, an interferon stimulator, from licorice was used to treat patients with subacute hepatic failure. The survival rate was 72 percent compared to 31 percent in those who received traditional therapies.

In 13 cases of infectious hepatitis treated with licorice, the icterus index normalized in 13 days, urinary bile pigments were negative in 10 days, marked reduction of hepatomegaly took 9 days, pain over the liver disappeared in 8 days.

Glycyrrhizin has been used in Japan for more than 60 years in the treatment of hepatitis C. In several clinical trials it has been found to significantly lower AST, ALT, and GGT concentrations while reversing histologic evidence of necrosis and inflammatory lesions in the liver. And glycyrrhizin improves clinical picture, improves liver function, and reverses viral infection during chronic hepatitis B infection.

OTHER STUDIES

Atopic dermatitis. A licorice gel was used to successfully treat atopic dermatitis in a double-blind clinical trial, with 30 people in each group. The gel significantly reduced erythema, edema, and itching over the 2-week trial.

Aphthous stomatitis. Bioadhesive patches containing licorice were used to control the pain and reduce healing time in recurrent aphthous ulcer. Licorice patches caused a significant reduction in the diameter of the inflammatory halo and necrotic center compared with placebo. (There have been three of these trials, all successful.)

Pharmaceutical side effects. In a comparative trial, licorice, when used along with spironolactone in the treatment of polycystic ovary syndrome, significantly reduced the side effects compared to spironolactone when used alone.

Peptic ulcer. Licorice was found in a trial with 100 people with peptic ulcer (86 of whom were unresponsive to conventional treatment) to be effective: 90 percent experienced good effects, 22 were cured, 28 were significantly improved.

Lichens planus. In a clinical trial of lichens planus, 66 percent of people who took glycyrrhizin were cured.

Miscellaneous. There have been a number of trials using licorice in combination with other herbs. It reduced risperidone-induced hyperprolactinemia in patients with schizophrenia. Reduced hyperuricemia in vegetarians. Was effective in the treatment of advanced pancreatic and other gastrointestinal malignancies. Was successful in the treatment of 138 cases of intestinal metaplasia and 104 of atypical hyperplasia of the gastric mucosa.

Lomatium

Family: Apiaceae, the carrot family

Species used: There are 70 or maybe 80 species in this genus—trying to pin a taxonomist down is like gluing feathers on a donkey so it can fly. They are indigenous to the United States (lomatiums, not taxonomists) and grow from the Mississippi River region throughout the West, Southwest, and Northwest. A number of them can be used medicinally but information on the genus and its medicinal uses is very sparse. *Many of the species are exceptionally rare and are endangered so be highly conscious if you are wildcrafting. Only harvest if the species is very common in your area or is not endangered.* The most commonly used lomatium is *Lomatium dissectum* but several others can be used identically and grow in enough abundance, here and there, to be harvested and are (usually) not considered endangered: *L. ambiguum, L. bicolor, L. cous, L. foeniculaceum, L. grayi, L. macrocarpum, L. nudicaule, L. orientale, L. simplex, L. triternatum.*

L. dissectum, L. cous, L. bicolor, L. foeniculaceum, L. macrocarpum, and *L. orientale* have the widest range, some of them extending into Iowa, Minnesota, and Missouri. *L. nudicaule,* though confined to the Northwest, has a particularly long history of medicinal use and is considered by some to be stronger than *L. dissectum.*

There are, just to make things harder, two varieties of *L. dissectum, L. dissectum* var. *dissectum* and *L. dissectum* var. *multifidum.* The former is more prevalent east of the Cascade mountain range, the latter west. The former likes more rain and lower altitudes, the latter likes it semi-arid and grows as high as 7,000 feet. I have generally used *multifidum*; it appears to be somewhat stronger in its medicinal effects. Almost no source you order from will be able to tell you which variety of *dissectum* they are selling you.

Synonyms: The *Lomatium* genus used to be the genus *Leptotaenia.* It was reclassified during World War II—shortly before it was inducted. *Lomatium dissectum,* for example, used to be *Leptotaenia dissecta.* For some reason *Lomatium dissectum* was also once called *Ferula dissoluta, ferula* meaning "rod" and *dissoluta* meaning "debauched, unrestrained by convention or morality," ergo, debauched rod (there's a really good

joke here but . . .). In any event, it appears that botanists and taxonomists are closely related, perhaps from an ancient debauchery.

Common names: Lomatium, biscuit root, cough root, Indian consumption plant, desert parsley, Indian parsnip. *Lomatium dissectum* is sometimes known as fernleaf biscuitroot.

Biscuit root, desert parsley, and lomatium are the general common identifiers and will usually be combined with a descriptive to create a particular species' common name in a geographical area, e.g., northern Idaho biscuit root, Bradshaw's desert parsley, California lomatium.

Parts Used

The root is normally what is used, but the seeds are highly active. They often contain considerably more constituents than the root and may be used instead (though hardly anyone does).

Preparation and Dosage

Lomatium is generally used as a tincture (at least these days).

TINCTURE

Fresh root: 1:2 herb:liquid ratio. Chop the wilted root as finely as possible with a very sharp knife, place in a jar, add the tincturing medium (70 percent grain alcohol, 30 percent water), cover, and let macerate 2 weeks. Dosage: 10–30 drops up to 5x daily. In acute conditions, take 10–30 drops each hour.

Dry root: Same as for the fresh root, except powder the root and use a 1:5 herb:liquid ratio.

Fresh seeds: Same as for the fresh root.

Dry seeds: 1:3 herb:liquid ratio, with 50 percent alcohol. Dosage: 1 dropperful 3–5x daily or once per hour in acute conditions.

Common influenza tincture blend: Combine equal parts of lomatium, red root, licorice, and pleurisy root (*Asclepias tuberosa*) tinctures. In acute conditions with debility (bedridden, lingering loss of energy, failure to thrive, pneumonia) take up to 1 teaspoon 6x daily. You should make up 16 or so ounces to keep on hand. When the flu hits you won't want to make it. (I do, however, prefer the more extensive formulation described in Chapter 2; see page 41.)

INFUSION OR DECOCTION

You can try it, it worked for the indigenous peoples. I have tasted and used the tea myself and its impact on the tongue is nearly as strong as that of the tincture. Somehow, in spite of not being water soluble, the aromatics are indeed getting into the tea. (It is prepared by pouring hot water over the root and leaving it covered for an hour or so. This does help keep the aromatics in there.)

Add 1 teaspoon of the powdered root to 6 ounces hot water, cover, and let steep.

Influenza decoction blend: Robin Seydel's influenza blend (via Northwest herbalist Ryan Drum): Combine 1 ounce each of lomatium seeds, lobaria lichen, licorice root, Oregon grape root, echinacea blossoms, red clover blossoms, and rowan berries. Grind all the herbs into a powder (or as close as you can get). Add to 1 quart hot water, and boil for 2–4 minutes. Remove from the heat, cover, and let steep for an hour. Then strain, add lemon juice and honey to taste, and drink hot, 1 quart per day.

STEAM INHALANT

Pour boiling water over some of the freshly chopped root or seeds (which should be in a hot-liquid-tolerant container). Drape a towel over your head and the container, and breathe in. This works a treat for influenzal infections. Take the tincture, too.

Side Effects and Contraindications

Pregnancy and a nasty rash. (Actually, that should be "Nasty rash and pregnancy"—you won't get pregnant from taking the tincture.)

From what is known, the plants are exceptionally nontoxic. Injections of isolates of the plants at 2.5 percent of the weight of mice were not toxic in any way—no observable differences at all. *However*, the plant is contraindicated in pregnancy *and* about 1 percent of people using the herb get a rather nasty rash. The rash appears to be associated only with *Lomatium dissectum*; the use of other species has apparently not produced it.

As far as anyone knows, this *is* an allergic reaction to the plant but it is a rather unusual one—there is no itching, no discomfort other than the visual. Herbalist Michael Moore reports that he found it occurring

only with the fresh root tincture and only then if the tincture was taken as a single, i.e., not blended with anything else.

The rash usually begins within 8 hours of taking the tincture. It can cover the whole body—warm baths often make it worse. The rash is a deep to dark red, even purplish. It can cover the entire body (most people stay home to avoid encountering the human subspecies *Homo staris*). There is rarely, if ever, any itching or discomfort. It's just there. *Nothing* will make it better, not steroids, not Benadryl, not calamine lotion, not Pepcid, not herbs, not herbal washes, not doctors or hospitals. So save your money and skip the emergency room visit—besides physicians don't know squat about plant medicines, or their side effects. (Though they are often highly prejudiced against them.) It's grin-and-bear-it time. The rash will disappear within a week or so after its initial occurrence.

To avoid the rash use lomatium as part of a mixture, not singly, and use the dried root, not the fresh root, for tinctures. Oh, and don't spend a lot of time looking in the mirror.

Herb/Drug Interactions

None known.

Habitat and Appearance

The lomatiums are called desert parsley for a reason; their leaves and stems often look very similar to those rarely purchased parsley bunches you see in the grocery store and sometimes buy and use a tiny bit of for an experimental recipe before the rest decays into a dessicated phytomemory (often found months later) in the bottom of the vegetable drawer in the refrigerator. Some of the species are small, like that parsley bunch, while others are much larger; *L. dissectum* is one of the giants.

Like most of this family, *L. dissectum* begins as a fernlike bunch of basal leaves that, as the plant matures, sends up a rather substantial flowering stalk. The fernlike leaves of this species may be up to a foot long, with a dozen or so leaves spreading out from the root core. The flowering stalks can be from 2 to 5 feet in height. They are usually leafless. The plant flowers in early spring, the seeds mature in late summer or early fall.

The roots are 3 or so inches in diameter and up to 2 feet long (*L. nudicaule*: 6 inches in diameter, up to 3 feet long); they're big. Herbalist Michael Moore describes the root in his typical fashion:

> The root is fleshy, thick at the top, lumpy, and irregular, like a mutant cross between a carrot and rutabaga, with odd parts left over. The skin is pearly grey, with many oil glands spread throughout the variously cream- and yellow-colored flesh.[10]

Most lomatiums grow in well-drained, sandy or rocky soil—basically a certain kind of deserty-sandy-dryish-hard-to-get-to, hard-to-dig-in, old volcanicy or decomposed granity terrain and soil. There are a few exceptions—such as *L. nudicaule*. But herbalist Ryan Drum comments that *that* particular plant was most likely brought to the island on which he lives in Puget Sound by the Salish who settled there. The plant only grows near old Salish settlements on extensive beach sand flats with few other plant competitors, a terrain similar to the deserty homeland it was transplanted from.

The lomatiums are semiarid plants of the Great Plains, deserts, high mountains, and northwest United States. That's where you will find them.

Cultivation and Collection

The lomatiums don't cultivate easily though a few agricultural groups are insisting they are indeed growing the plant and at least one commercial company describes its tincture as being from organically grown roots. Still, germination of the seeds is reportedly difficult. The plant *could* grow in similar terrain any place on Earth but it would take work to get it to take in a new location. Transplanting may be a better option.

If you are wild-harvesting, you need to be aware of a couple of things. The first is the terrain: Lomatium likes it tough. *Lomatium dissectum*, and its variants, likes to grow around impossibly difficult rocky outcrops and the roots are tenacious. Find some in soil and skip the rocky ones. The second is the *nature* of the root itself: It appears in different forms depending on its age, the time of year, and which species it is from. Michael Moore comments:

> In the spring the roots ooze milky aromatic sap; by fall the sap is more resinous and balsamic; but in any case, the sticky bitter aromatic sap and the soft, fibrous flesh (not at all woody) differentiate this big Lomatium from the others

> of the genus. The Lomatium clan is huge, with eighty closely related species in the western states. For many years the large size of a few of these plants (most Lomatiums are quite small) caused botanists to classify them apart in the genus Leptotaenia. The larger Lomatiums, by whatever name, are an amorphous group, and I have found several strikingly varied stands whose roots are typical of *L. dissectum* in their morphology and constituents, which act like *L. dissectum* when tinctured, have identical constituents as measured by TLC (think-layer chromatography), but don't particularly look like *L. dissectum*. I have found these plants in such varied locations as Modoc County, California, northwestern Wyoming, and the front range of Colorado. . . . Most of the other large Lomatiums that grow in the range of *L. dissectum* have sweet, starchy parsniplike roots and could not be confused with this bitter, aromatic, oily- and waxy-rooted giant.[11]

Moore lists the species that he has found to be usable as *L. dissectum, L. multifidum, L. eatonii,* and *L. occidentalis*. Ryan Drum comments that *L. nudicaule* is just as good if not better. *Lomatium grayi* and *L. dissectum* overlap in their range and the two are often confused.

In any event, the root you are harvesting must be strongly aromatic, bitter, and oily if it is to be antiviral. Normally this is seen *only* in mature plants. If it is not, then it probably won't work as a medicinal though it will work as a rather good food source.

You can harvest the roots at any time of the year. They are easier to identify in the spring, a bit easier to dig after spring rains.

Dry the roots for a few days, then cut them up (they are a bit too resinous if you don't let them dry a little first). *Make sure you cut them up.* Generally they are sliced into wheels as you would slice a carrot. If you dry them whole, you will need a chain saw to reduce their size later. (Just an FYI on that one.) Once the root circles are well dried store them in well-sealed plastic bags inside a well-sealed plastic tub out of direct sunlight in a coolish location. They will last several years. At least.

Moore likes the spring roots for making a fresh root tincture, and the fall roots for dried root tincture. I tend to prefer spring roots in this family; they are much bigger in my experience and, I feel, more potent. That is because the roots of all perennials grow throughout the winter, albeit very slowly. By spring they are full of stored nutrients and tend to be juicy and fat. By fall they have used those stores to send up stalks and set seed. Fall roots are stringier, a bit dryish and beef-jerkyish.

The big lomatiums are perennials; it takes them years to mature—often as much as a decade before they can be harvested. *Many sources*

Properties of Lomatium

Actions

Analgesic
Antibacterial
Antifungal
Antimicrobial
Antiseptic
Antispasmodic
Antiviral
Expectorant
Mucous membrane tonic

Active Against

There have been few studies on the activity of the lomatiums against microorganisms and virtually none on their activities against viruses. The two most comprehensive studies occurred in 1948 and 1949 in the United States. Both focused on the antibacterial activities of extracted aromatics of the root, one isolated through steam distillation, the other through ethyl acetate extraction and filtration. The range of activity was fairly broad. Fractions of the steam distillate were found active against the following:

Achromobacter lacticum
Agrobacterium spp.
Aspergillus spp.
Bacillus spp.
Candida albicans
Clostridium spp. (mildly)
Coccidioides immitis
Corynebacterium diphtheriae
Diplococcus pneumoniae
Eberthella typhosa
Escherichia coli (mildly)
Fusarium spp.
Haemophilus influenzae
Histoplasmosis capsulatum
Micrococcus tetragenus
Microspermum trichoderma
Mucocus capsulatus
Mucor culmorum
Mycobacterium spp.
Mycoderma spp.
Neisseria spp. (mildly)
Pestallotia funera
Proteus spp.
Pseudomonas spp.
Pythium debaryanum
Rhizoctonia spp.
Serratia marcescens
Shigella spp.
Staphylococcus spp.
Streptomyces griseus
Trichophyton spp.

In the ethyl acetate extracts study, several different forms were used at varying degrees of strength. They were found active against the following:

Bacillus subtilis
Corynebacterium diphtheriae
Diplococcus pneumoniae
Escherichia coli (mildly)
Micrococcus aureus
Neisseria catarrhalis
Proteus vulgaris (mildly)
Pseudomonas aeruginosa (mildly)
Serratia marcescens (mildly)
Streptococcus pyogenes
Vibrio comma (a.k.a. *V. cholerae*)

The ethyl acetate extracts were not active against *Klebsiella pneumoniae*, *Salmonella schottmuelleri*, *Aerobacter aerogenes.*

Both studies found the steam and ethyl acetate isolates much more active against Gram-positive organisms than Gram-negative (as usual). Even at concentrations of 10^{-3} the Gram-positive organisms were completely inhibited. Some of the Gram-negative organisms were, however, highly susceptible to the isolates, e.g., *Vibrio comma*, *Neisseria catarrhalis.*

There have been no in-depth studies on the antiviral properties of lomatium, however a few researchers looking a bit more broadly report the plant roots to be active against rotavirus and HIV as well as *Bacillus subtilis*, *Staphylococcus aureus*, *Colletotrichum fragariae*, *Mycobacterium tuberculosis*, *Pseudomonas aeruginosa*, *Trypanosoma cruzi*, and *Propionbacterium acnes.*

In spite of the lack of viral studies, I, and many others, have found the plant highly active against most viral and bacterial respiratory infections, including pneumonia. I haven't found anything better for serious, debilitating influenza, avian flu, swine flu, West Nile, or incapacitating pneumonia. In some instances the people were bedridden, very weak and debilitated; in others, ER physicians had diagnosed them with severe pneumonia. In all cases focused treatment (i.e., high, frequent doses) worked well, though in the more debilitated instances, it took weeks to turn it around. Normally, people begin to show improvement within a day or two.

Use to Treat

Upper respiratory viral infections, all influenza strains, SARS, viral encephalitis, pneumonia. Lomatium is most effective, in my opinion, if combined with other herbs such as red root, licorice, pleurisy root. Nevertheless, it is very potent by itself and can be used as a single (however, see the "Side Effects and Contraindications"section, page 300). The taste is intense though you can get used to it.

Clinicians have reported good success in using the plant for other viral infections, for instance, Epstein-Barr and cytomegalovirus (in chronic-fatigue-like situations), hepatitis C (lowering viral load), and HIV. Some (Michael Moore) have used it in the treatment of shigella and other bacterial infections with good success.

There is a tendency to look at the plant as a systemic antibacterial due to the early studies. I think the plant is better thought of as a systemic antiviral, especially for respiratory infections, most especially the emerging influenza strains. See the section on Western botanic practice, page 309.

Continued on next page

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Other Uses

The roots of many lomatiums, before they attain maturity, are highly edible (though the taste may take some getting used to). The roots have traditionally been eaten fresh, steamed, roasted, boiled, in soups and stews, canned, or dried to crispy, and very tasty, sticks (if the roots are small enough). Larger immature roots or the sweeter varieties were peeled, pounded into cakes, and dried in the sun, hence the "biscuit" part of the common name. Lewis and Clark compared the taste of the dried cakes to bread. Most lomatium species were apparently used similarly.

Finding It

You can order the sliced and dried roots rather easily online. I prefer Pacific Botanicals (https://www.pacificbotanicals.com) for most herbs that I have to order in bulk; their quality is supreme.

put the minimum period of growth before the plants can be harvested for medicine at 20 years. Some of the lomatiums in the wild are very old indeed—several hundred years; old-growth plants are not just trees.

Ryan Drum notes that the young *L. nudicaule* roots are sweet and starchy, very tender and highly edible, much like a plant candy. Only the older plants, when matured, will present with the typical oily, resinous, aromatic medicinal constituents needed in this species. Numerous other sources indicate that the same is true of *L. dissectum* and its variants—the plants need somewhere near a decade at minimum before they begin generating their volatile, and potently antiviral, oils and become useful for medicine.

If you harvest a root and it is sweet and starchy, then eat it but don't use it for medicine. However, all indications are that the seeds can be used no matter how old the plant is; they possess a similar chemistry to the root. This is borne out in part by the traditional uses of the seeds of *L. nudicaule* by the Salish in northwestern Washington State. They

used *only* the seeds for medicine (the root was a food staple) and their traditional uses of the seeds are for the same range of problems that the roots are now used for in conventional practice.

Plant Chemistry

One of the main problems with scientific studies on the lomatiums (there haven't been many) is that researchers rarely make any distinctions between older plant roots that are highly aromatic and younger plant roots that are not. (It took highly schooled plant specialists *decades* to realize that some plants they had listed as perennials—or biennials—were in fact unique monocarpic plants, *Frasera speciosa* being an example. That plant may take anywhere from 20 to 100 years before it sends up a stalk and flowers and once it does, it dies. These specialist guys, they are a bit slow. And no, they didn't think to look at any of the Native American data on that plant.)

Comparison studies on the lomatiums should only occur with plants of similar ages; the differences in the percentage of compounds found in various subspecies of *Lomatium dissectum* could easily be from differences in the age of the roots tested. The current chemistry studies, which are nice to have, are nevertheless suspect in some of their conclusions about the species studied due to the failure of the researchers to indicate the age of the plants or to be certain the plants were of a similar age before comparisons.

Nevertheless, many members of the *Lomatium* genus (and the carrot family of which it is a member) have a number of similar chemistries, e.g., apiol, a calcium antagonist widely present in the carrot family and strongly present in the seeds and roots of *Lomatium californicum* and *Ligusticum hultenii*. The antiasthmatic and antispasmodic compound Z-ligustilide is common in many of the lomatiums as well.

Most of the chemical studies on the lomatiums have been conducted by butterfly researchers since butterflies are strongly attracted to this genus (the researchers are looking for the exact chemical attractants so they go deep). The nearest to a comprehensive look at the chemistry of the main medicinal species has occurred with the essential oils of two variants of *Lomatium dissectum*. The roots, seeds, and stems all were found to contain a number of terpenoid hydrocarbons, among other things.

The seeds of *Lomatium dissectum* var. *dissectum* were found to contain 113 compounds; the seeds of *Lomatium dissectum* var. *multifidum* contained 138. The leaves and upper stems of variety *dissectum* contained 137 compounds; those of variety *multifidum* contained 173.

The root of variety *dissectum* had 68 compounds; oddly, the study doesn't reveal the number of root compounds of variety *multifidum*. Variety *dissectum* is particularly rich in esters, specifically the 2-methylbutyrates; the primary compounds found are: phellandrene, limonene, beta-caryophyllene, palmitic acid, E-beta-ocimene, linolenic acid, octanol, octyl acetate, myrcene, 4-methylpentyl 2-methylbutyrate, alpha-bisabolol, cuparene, Z-S-hexenol, decyl acetate, longifolene, palmitoleic acid, Z-ligustilide, and E-2-methyl-3-octen-5-yne. The roots of the two closely related forms of *Lomatium dissectum* have very similar chemistries but they do differ, e.g., *Lomatium dissectum* var. *multifidum* has about 20 percent longifolene (a major constituent of many pines) in its complex of essential oils, while *Lomatium dissectum* var. *dissectum* has only about 3 percent.

Studies with other lomatiums have found similar compounds. The best in-depth look at species other than *dissectum* comes from Philip S. Beauchamp et al. (2009). They identified over 200 constituents in the plants. While all of the plants did not have the same constituents, they were similar in their compounds. The principal components of the six species (*brandegei*, *eastwoodiae*, *graveolens*, *howelii*, *junceum*, and *parryi*) are (in order or prominence): alpha-pinene, beta-pinene, camphene, alpha-phellandrene, alpha-terpinene, beta-phellandrene + limonene, Z-beta-ocimene, gamma-terpinene, p-mentha-2,4(8)-diene, linalool, dehydrosabina ketone, terpinen-4-ol, cryptone, alpha-terpineol, methyl chavicol, citronellol, methyl thymol, linalyl acetate, lavandulyl acetate, citronellyl acetate, beta-bourbonene, benzyl-2-methylbutyrate, beta-elemene, italicene, dodecanal, beta-caryophyllene, gamma-elemene, alpha-humulene, 7-epi-e,2-dehydrosesquicineole, gamma-muurolene, germacrene D, beta-selinene, bicyclogermacene, germacrene A, alpha-cadinene, germacrene B, epi-alpha-cadinol, alpha-muurolol. There are a number of highly antimicrobial tetronic acids in the root.[12]

Other species (*californicum*, *dasycarpum*, *grayi*, *lucidum*, *macrocarpum*, *nuttallii*, *rigidum*, *suksdorfii*, *utriculatum*) contain large amounts of similar constituents: falcarindiol, coniferyl ferulate, ferulic acid,

Z-ligustilide, senkyunolide, trans-neocnidilide, apiol, suksdorfin, osthol, chromones, sibiricin, macrocarpin, beta-phellandrene/limonene, decanal, dodecanal, bornyl acetate, germacrene D, alpha-humulene, bicyclogermacrene, alpha-pinene, beta-pinene, peucenin 7-methyl ester, beta-caryophyllene, Z-3-hexenol, palmitic acid, linoleic acid, E-2-hexenal, sabinene, terpinen-4-ol, myrcene, various coumarins, glycosides, and flavonoids. Z-falcarinol levels are particularly high in most lomatium species' roots.

The chemistry of the lomatiums, as with most plants, is complex and poorly understood. The seeds and aerial parts do have a great many more compounds than the roots, the seeds appearing nearly as active as antimicrobials as the roots. However, the roots do possess a number of compounds unique to them and are generally, in traditional practice and conventional herbal circles (though not in indigenous), considered to be the most potent antiviral parts of the plants.

Again: Only as the plant roots age do the major aromatics become highly concentrated in the roots; the seeds possess them from the beginning.

Traditional Uses

I have spread what would normally be in this section throughout this monograph. Read on.

AYURVEDA

Unknown.

TRADITIONAL CHINESE MEDICINE

Unknown.

WESTERN BOTANIC PRACTICE

All the lomatiums were known to the indigenous cultures that lived among them. All of them used the plants similarly: roots for food, seeds and roots for medicine (among other things). Lewis and Clark knew of the plants' uses for food, as they used them on their trek across the country once they were introduced to them. They almost certainly would have known of the plants' uses as medicine. Many of the trappers who lived in the region would have known as well. They didn't seem to have

written about them, however. Lomatium only came to the awareness of the medical community during the influenza epidemic of 1918–1919.

The Washoe tribe in Nevada used the plant to treat members of the tribe who had fallen sick with the disease. A report on their use of the plant was printed in the *Bulletin of the Nevada State Board of Health*, in January 1920, by a physician, Ernst Krebs. If you have read, in depth, the description of just what the 1918 influenza virus did in the bodies of those it infected and then compare the outcomes in practice from lomatium during the pandemic, the effects of the herb are considerable.

> The Indians gather this root in the late fall, November being considered the proper month for gathering. The root is used in the fresh or dry state. It is cut up and a decoction is made by boiling the root in water, skimming off the top and giving large doses of the broth. A pound of root is considered about the proper dose to treat a case of fever for 3 days, which is the longest time needed to break up a fever due to influenza or pulmonary disease, although the Washoes used it as a panacea. Whether a coincidence or not, there was not a single death in the Washoe tribe from influenza or its complications, although Indians living in other parts of the State where the root did not grow died in numbers. It was such a remarkable coincidence that the root was investigated by a practicing physician who saw apparently hopeless cases recover completely without any other medication or care of any kind.
>
> A preparation was prepared and employed in a great many cases among the whites, from the mildest to the most virulent types of influenza, and it proved itself to be a reliable agent in preventing pulmonary complications. Other physicians were induced to give it a trial with the same results. It is beyond the experimental stage, as its therapeutic action in this direction is established and beyond any doubt. The cases in which it has been used run into the hundreds. There is probably no therapeutic agent so valuable in the treatment of influenzal pneumonia and, as far as being tried, in ordinary lobar pneumonia if started early. Its action on coughs is more certain than opiate expectorants and its benefit is lasting. It acts as a powerful tonic to the respiratory mucus membranes. It is a bronchial, intestinal and urinary antiseptic and is excreted by these organs.[13]

Interestingly, the aromatic constituents in the root are only mildly soluble in water, are better soluble in alcohol, and are lost when boiled (making a decoction). Given the traditional indigenous preparation methods, there are obviously a number of other compounds in the root, water soluble and heat resistant, that are, inescapably, strongly antiviral.

For a while, the reports of the efficacy of the plant stimulated study of its actions but the emergence of pharmaceutical antibiotics stopped research on the plant until Michael Moore began speaking about it in the early 1970s. It is now considered (among many herbalists) to be *the* primary antiviral medicine in the U.S. herbal community.

Scientific Research

Most of this section has been spread throughout the rest of this monograph, so, just a couple of notes here:

The lomatiums contain a large number of chemically unique coumarins (pyranocoumarins, furanocoumarins, prenyloxycoumarins, prenyloxyfuranocoumarins, and so on) that have been found in a number of studies to be powerfully antiviral against both RNA and DNA viruses ("remarkable" is how one research team put it). The compounds penetrate the viral coat and inhibit ribonucleoprotein-complex-associated activity among other things. They have been found exceptionally potent against influenza viruses (H1N1, etc.). *Ferula assa-foetida,* once considered a close relative of *Lomatium dissectum* (formerly *Ferula dissoluta*), is especially rich in the same compounds; they are more potent against influenza viruses than amantadine. (Specifically, they inhibit the M2 ion channel.) The compounds in lomatium are in fact some of the most potent M2 ion channel inhibitors known.

Lomatiums also have high levels of longifolene, which is an exceptionally active antimicrobial. It is as active as the pharmaceutical nifurtimox against *Trypanosoma cruzi*, which causes Chagas disease. It is primarily active against Gram-positive bacteria.

Honorable Mentions

There are a few other herbs that are showing potent antiviral actions that I think warrant closer examination over time. They are *Ampelopsis brevipedunculata, Forsythia suspensa, Sophora flavescens, Strobilanthes cusia,* and boneset (*Eupatorium perfoliatum*). The only one I will offer any depth on here is boneset. (And, yes, there are *lots* of other plants with antiviral actions; this is just a beginning. And yes, I do know about *Ocimum sanctum*.)

AMPELOPSIS BREVIPEDUNCULATA

Ampelopsis brevipedunculata, a.k.a porcelain berry, is an invasive, especially in the eastern United States, and is native to Asia. It's a woody vine that grows to 25 feet in length with an extensive root system. It mimics kudzu root in a lesser, porcelain berry sort of way, covering whatever it can as it spreads. It is on the lists of most phytoaryans; it is considered armed and dangerous, to be exterminated with extreme prejudice.

The herb is active against a number of viruses, including enterovirus 71, hepatitis, and varicella zoster. The stem, roots, and leaves are all used, though the stem and roots are the most commonly used for

treating viral diseases. A water/ethanol extract of the roots and stems was found to be the strongest medicinal plant compound, of any tested, for treating four strains of enterovirus 71. The plant is traditionally used in Japan and parts of Asia as a major anti-inflammatory, hepatoprotective, and analgesic.

It has a number of immune modulation effects and has proved effective in treating liver fibrosis. It protects the liver from various toxic substances, is active against breast cancer cells, is a fairly potent antioxidant, is decidedly anti-inflammatory, and is analgesic. Because the plant is an invasive, its use really should be further explored.

FORSYTHIA SUSPENSA

Forsythia suspensa is one of the 50 fundamental herbs in Chinese medicine. It is a weeping forsythia, a large shrub, and quite pretty. It has escaped cultivation and is moderately invasive throughout the United States.

It is called either qing qiao or huang qiao in Chinese medicine, depending on whether the green or fully ripe yellow fruit is used as medicine. The unripe fruit is considered to be the strongest. (Rarely, the stem bark and leaves are used.) The dried fruit, whatever its ripeness, is used for fever, headache, restlessness, delirium, lymph gland enlargement, erysipelas, boils, and inflammations.

The herb is highly active against influenza A viruses, avian infectious bronchitis virus, cytomegalovirus, and respiratory syncytial virus. A number of the compounds in the plant have a fairly wide range of antiviral action (ursolic acid, oleanolic acid, quercetin, rutin, pinoresinol) and the plant really should be tested further. Studies on the leaves have found them to be stronger in their impacts than the flowers.

It is anti-inflammatory, antioxidant, vasorelaxive, antibacterial (against staph, *Helicobacter pylori*, *E. coli*), antiemetic, cytoprotective, and diuretic (antiedema).

SOPHORA FLAVESCENS

Sophora flavescens is also an herb used in traditional Chinese medicine, in which it is called ku shen. It is used for the treatment of viral hepatitis, viral myocarditis, gastritis, enteritis, cancer, and various skin diseases. The root is usually used. The plant is considered specific for jaundice,

dysentery, leukorrhea, itching, scabies, eczema, and dysuria when used in combination with other plants.

The herb has been found active against a range of viruses: hepatitis B, respiratory syncytial virus, Coxsackie B3, HHV-6, coronavirus, influenza A (H1N1), rotavirus, herpes simplex virus 1 and 2, HIV, and various cancers. It is a potent neuraminidase inhibitor and also has some antibacterial (staph, enterococci, resistant and nonresistant) and antifungal (*Aspergillus niger*) actions.

STROBILANTHES CUSIA

Strobilanthes cusia is a plant that deserves more attention as an antiviral in that it contains many of the same compounds (e.g., tryptanthrin, indigo, indirubin) as isatis. (One of the plant's common names is actually Assam indigo and it was once used, as isatis was, as a source of indigo dye.) And, not surprisingly, it is a pretty good antiviral, too. In Chinese medicine it is called da-chang-yeh. The roots and leaves are used.

The plant is active against a number of viruses including influenza A (H1N1) strains, hepatitis B, herpes simplex virus 1, Sindbis virus, eastern equine encephalitis virus, Getah virus, and tobacco mosaic virus. Studies *in planta* found it increased innate resistance in plants to virus infections.

The plant contains some fairly potent compounds that target the subgenomic RNA of alphaviruses and alphavirus-like RNA viruses. This viral group, when it infects people, causes rashes and arthritis and encephalitis.

The plant has also shown antiplasmodial and antifungal activity.

In Chinese medicine, the plant is used for influenza, epidemic cerebrospinal meningitis, encephalitis B (Japanese viral encephalitis), viral pneumonia, and mumps. It is used for sore throats, aphthae, inflammatory diseases of the skin, and to reduce fever. The plant is considered antinociceptive, anti-inflammatory, and antipyretic.

Because the plant has so many of the same potent antiviral compounds as isatis and because it is widely grown throughout the world as an ornamental, its antiviral actions should be studied in more depth. One hope: Because the plant is in a different family, maybe it won't taste like isatis, i.e., spoiled cabbage.

Boneset

There have been extremely few studies on boneset; most of them are very old and what newish ones there are mostly quote nineteenth-century texts. Recently Mareike Maas, in Germany, has been doing some very good in-depth research on the plant, much of which is very hard to get in English. Her work is showing that the plant has a much wider range of antiviral actions than was formerly understood. I suspect the plant is going to be a more useful antiviral than previously suspected.

Family: Compositae

Species used: There are 36, or 60, or pi? species in the *Eupatorium* genus, taxonomists being troublesome again. Nearly all are native to the Americas, *Eupatorium cannabinum* being an exception. Many of the species in the genus are medicinal, and some do have a very similar range of action. However, this is the one I know best, so *Eupatorium perfoliatum* it is.

Common names: Boneset, common boneset, throughwort, agueweed, feverwort, sweating plant—but no one has used those last three names since 1885. (And it's pronounced "A-gyew-weed," not "aaagh-weed," big fella.)

Parts Used

Aerial parts, in flower or just before flowering, depending.

Preparation and Dosage

The herb is bitter and about as much fun to drink as a tea made from earwax. Honey helps considerably . . . and if you have the kind of flu where you can't taste anything. Generally, the herb is taken as tea or tincture but few take the tincture directly on the tongue. Too bitter.

TEA

Cold tea: Combine 1 ounce of herb with 1 quart boiling water, and let steep overnight. Strain and drink throughout the day. The cold infusion is better for the mucous membrane system and as a liver tonic. If you want to help fevers, you need to take the tea hot.

Hot tea: Combine 1 teaspoon herb with 8 ounces hot water, and let steep 15 minutes. Take 4–6 ounces up to 4x daily. Boneset is only diaphoretic when hot and should be consumed hot for active infections or for recurring chills and fevers.

TINCTURE

Fresh herb in flower: Use a 1:2 herb:liquid ratio, with 95 percent alcohol. Dosage: 20–40 drops in hot water up to 3x daily.

Dry herb: Use a 1:5 herb:liquid ratio, with 60 percent alcohol. Dosage: 30–50 drops in hot water up to 3x daily.

For acute viral or bacterial upper respiratory infections: Take 10 drops of tincture in hot water every half hour up to 6x daily.

For chronic conditions: When the acute stage has passed but there is continued chronic fatigue and relapse, take 10 drops of tincture in hot water 4x daily.

Side Effects and Contraindications

For some reason the phytohysteria surrounding elder is not present with this herb. Boneset is an emetic when taken in large doses, so an early sign that you may be taking too much is *nausea*. Generally, the cooler the tea the less nausea. However, the tea really must be taken hot to help fevers. The herb *may* be contraindicated in pregnancy but no one really seems to know why. *Sometimes* some people have an allergic reaction to plants in this family (chamomile, feverfew, ragwort, tansy), so if you are allergic to those, careful with this one.

Herb/Drug Interactions

None noted.

Habitat and Appearance

The plant is pervasive in the eastern half of the United States and Canada, from Texas, Oklahoma, North Dakota, and so on eastward. However, every place I've seen it grow has been wettish, humid, with good soil.

Boneset grows up to 3 feet tall, they say. I've never seen it get that big, but most of my experience of the plant has been in the tiny state of Vermont. Two feet seems about average, just as with hominids. The

plant grows in a straight stalk, the leaves going north-south, then east-west, then north-south again. The leaves continue on through the stalk, hence *through*wort; it basically looks like the opposing leaves were glued together at the wide end and the stalk just punched through them. Once seen, never forgotten.

Cultivation and Collection

The plant is a perennial and likes full or partial sun in moist to wet conditions, on the edges of swamps, along streams, in wet meadows, in marshlands, basically anyplace mosquitos like to breed except maybe old tires. It spreads by seed; there are a lot of sources on the internet.

If collected at flowering and allowed to dry the plant will usually go to seed as it dries. It should only be collected in flower (August or September) if being tinctured fresh and *right now*. If you are going to use it as a tea, it should be picked just prior to flowering, hung upside down in a shaded place, and allowed to thoroughly air-dry. If you pick it in flower and try to dry it then, it will go to seed as it is hanging there, like a bat, upside down, waiting to come suck your blood late in the night, and there will be a mess.

Plant Chemistry

Methylglucuronoxylan, astragalin, eufoliatin, eufoliatorin, eupatorin, euperfolin, euperfolitin, euperfolide, euccannabinolide, eupatoriopicrin, hyperoside, rutin, polysaccharides, a number of guaianolides, and a bunch of other stuff. Many of those are sesquiterpene lactones, common in the eupatoriums. There are a number of caffeic acid derivatives in the plant, at least five flavonoid glycosides, and a number of dicaffeoylglucaric acid derivatives that are considered unusual. (And if David Hoffmann is reading this that means: depsides of hydroxycinnamic acids with hexaric acids.) These unusual compounds are particularly high in the flowers.

Traditional Uses

AYURVEDA

Nope, but there are other eupatoriums used in this system.

Properties of Boneset

Actions

Analgesic
Antibacterial (mild)
Anti-inflammatory
Antiviral
Cytotoxic
Diaphoretic
Emetic (mild)
Febrifuge
Gastric bitter
Immunostimulant (increases phagocytosis)
Mucous membrane tonic
Peripheral circulatory stimulant
Smooth muscle relaxant

Active Against

There hasn't been a lot of testing of this plant as an antimicrobial. It is mildly active against some Gram-positive bacteria such as *Staphylococcus aureus* and *Bacillus megaterium*. It is stronger in its actions against malarial parasites (*Plasmodium* spp.), making it essentially a midlevel antiplasmodial herb. It has pretty good activity as an antiviral against influenza A (H1N1). Although not tested for it, I do believe this species is active against some, if not all, serotypes of dengue.

The eupatoriums do have a range of action against viruses but there has been much too little work on them. *Eupatorium patens* is active against dengue-2, HSV-1, and HSV-2; *Eupatorium articulatum* against HSV-1 and vesicular stomatitis virus (VSV); *Eupatorium glutinosum* against VSV; *Eupatorium bunifolium* against herpes simplex viruses. Most of these eupatoriums have been used similarly to boneset.

Note: Because of the common name *boneset* some people think this eupatorium good for setting bones. Others, however, insist that the name came from a common, ancient name for dengue, breakbone fever, and that the herb has never been used for setting bones and, more, indigenous peoples never used it for that either. (Feelings run high.) I have taken various sides in this over the years but did think it highly amusing a number of years ago when a well-respected indigenous herbalist (from the United States) who had used the herb for over 50 years (and who had learned its use from *her* teacher when she was very small) informed a rather self-satisfied group of herbalists that she used the herb primarily for setting broken bones (as a compress/poultice) and had done so all of her life. (So, now, I read fiction novels and don't think about why it is called what it is called.)

Continued on next page

Continued from previous page

Use to Treat

Influenza, dengue fever, malaria, all viral infections with intermittent fever (hot, then cold, then hot, then . . .), and aches and pains. Comment: I consider the plant a useful adjunct botanical for intermittent viral infections (flu, malaria, dengue), not a primary treatment botanical. It will really help lower fevers. It will help with the aches and pains of viral infections. It *will* make you sweat (if taken hot as a tea, as it should be).

Finding It

Fields and streams in the eastern United States, the internet, herb stores here and there. Strictly Medicinal Seeds (https://strictlymedicinalseeds.com) sells the seeds.

TRADITIONAL CHINESE MEDICINE

No, but there are other eupatoriums used in this system.

WESTERN BOTANIC PRACTICE

The plant, indigenous to North America, has been used by native peoples for millennia, specifically for intermittent fevers and chills, with pain in the bones, weakness, and debility. The American Eclectics used it for intermittent (i.e., malarial), typhoid, and remittent fevers, for general debility, pneumonia, cough, epidemic influenza, colds, catarrh, and pains accompanying those conditions. It was one of their primary remedies.

Scientific Research

The sesquiterpene lactones in boneset have a large range of actions. They are highly immunostimulatory and very active against cancers. One study found the herb itself to be potently cytotoxic, in essence comparable to the strength of the pharmaceutical chlorambucil.

The specific lactone active against the malarial parasite is considered to be a dimeric guaianolide. It has a range of antiplasmodial actions but is strongest against *Plasmodium falciparum*. The action is mild (compared to herbs such as cryptolepis) but if the plant is added to a traditional antimalarial that is strong, such as cryptolepis, the effects are mutually supportive. A homeopathic formulation of boneset was found to significantly

inhibit plasmodial replication (60 percent inhibition). And an in vivo study with mice found that the homeopathic preparation of the herb did inhibit plasmodial parasites but not completely.

South and Central American healers have been using homeopathic preparations of boneset to try and minimize the impacts of dengue fever outbreaks. In Rio in 2008, a homeopathic preparation containing phosphorus (30c), *Crotalus horridus* (30c), and *Eupatorium perfoliatum* (30c) was given to nearly 160,000 people. The incidence of the disease, compared to the same time period the year before, fell by 93 percent. In comparison, in areas not using the homeopathic preparation the disease incidence increased 128 percent. (Note: A few other, much smaller studies with the homeopathic of eupatorium itself for the common cold and dengue fever—though the people who were ill were not actually tested for dengue [the researchers guessed based on symptoms]—did not find any usefulness from the preparation.)

Clinical trials have shown that boneset stimulates phagocytosis better than echinacea, is analgesic (at least as effective as aspirin), and reduces cold and flu symptoms. In mice it has shown strong immunostimulant activity and cytotoxic action against cancer cells.

The herb is also anti-inflammatory for lipopolysaccharide-stimulated macrophages, primarily by inhibiting NO and iNOS expression. CSF-3, IL-1α and ß, and the chemokines CCL2, CCL22, and CXCL10 are all inhibited. TNF-α is moderately inhibited. Indications are that it inhibits NF-κB.

Again, despite boneset's long use and potent reputation little research has occurred with the plant.

Antiviral Supplements

While there are a number of supplements that do help during viral infections (vitamin C, vitamin B complex) zinc seems the most crucial, while monolaurin has shown some good antiviral activity in vitro and users report help from its addition to viral protocols.

Zinc

Zinc has been found active against a number of viruses (alphaviruses) and supportive in treatment for others (influenza, HIV). Studies have found that zinc supplements can triple the survival rate for children with pneumonia, for example, and that it significantly reduces the duration of the common cold.

Dosage: 10–25 mg daily depending on age and weight; 25–40 mg daily during acute episodes.

MONOLAURIN

Monolaurin is one of the major constituents of coconut oil and one of the factors in "the coconut oil miracle." It has been found, in vitro, to

be active against measles, HIV, HSV-1 and HSV-2, hepatitis, Epstein-Barr, infectious bronchitis virus, rubella virus, Newcastle disease virus, dengue (four serotypes), lymphocytic choriomeningitis, vesicular stomatitis virus, visna virus, cytomegalovirus, influenza viruses, pneumonovirus, and respiratory syncytial virus. Caveat: Most of these in vitro studies are hard to find; many of the assertions come from people connected with products, or are merely repetitions of other sources. There are very few in vivo studies, and even fewer with people. However, the constituent is considered safe (it's on the FDA's "Generally Recognized as Safe" list) and it has shown some very good effects against HIV in in vivo studies. I have seen some people experience good healing with it.

Dosage: 2–4 grams per day.

Supporting Lymph Function

During many viral infections, especially those that affect the lungs, the lymph system can become overloaded, the nodes severely inflamed, the spleen enlarged. Herbs that stimulate lymph action are very important to use. My favorite one is red root.

Red Root

Family: Rhamnaceae

Species used: *Homo dissertationus* has determined that there are 50 or 60 or 4^3 species of *Ceanothus* in the Americas. The genus doesn't grow anyplace else, at least not *natively*, though it is an ornamental throughout most of the world in one species shape or another.

Most species can be used medicinally; the most common are *C. velutinus*, *C. cuneatus*, *C. integerrimus*, *C. greggii*, and *C. americanus*. All species are apparently identical in their medicinal actions. My personal favorite is *Ceanothus fendleri*, a.k.a Fendler's ceanothus, which grows in my region and which I have been using for over 25 years. The important part is the color of the bark—see the "Cultivation and Collection" section on page 322.

Common name: Red root mostly, but in the old days it was supposedly called New Jersey tea (so they say; I never heard anyone say that phrase, at least when referring to something that does not inebriate).

Part Used

The root or inner bark of the root.

Preparation and Dosage

Red root can also be used as a tincture, tea, strong decoction, gargle, or capsules (but really, I think the tincture is best).

TINCTURE

Dry root, in a 1:5 herb:liquid ratio, with 50 percent alcohol. Dosage: 30–90 drops up to 4x daily.

TEA

1 teaspoon powdered root in 8 ounces water, simmer 15 minutes, strain. Dosage: Drink up to 6 cups daily.

STRONG DECOCTION

1 ounce herb in 16 ounces water, simmer slowly for 30 minutes, covered. Dosage: 1 tablespoon 3–4x per day.

GARGLE

In cases of tonsillitis or throat inflammation gargle with strong tea 4–6x per day.

CAPSULES

Take 10–30 "00" capsules per day if you must.

Side Effects and Contraindications

No side effects have been noted; however red root is contraindicated in pregnancy.

Herb/Drug Interactions

Should not be used with pharmaceutical coagulants or anticoagulants.

Habitat and Appearance

The various species in this genus seemingly grow everywhere in North and Central America, from Canada to Guatemala, from sea-level coastal scrublands to pine forests at 9,000 feet or higher. They can grow in hot, humid locations and semiarid desert areas. They are widely divergent in appearance, too, from tiny deciduous ground covers (up to 12 inches tall) to large evergreen bushes (to 9 feet tall) to "small" trees 25 feet in height. Their foliage ranges from tiny leathery leaves to large broad softies. Some species' branches have "spines," some don't. They do all have leaves though, so identification should not be a problem.

The flowers grow in tufted clusters and are intensely fragrant. (Yummy to my nose—at least with *C. fendleri*.) The seed capsules are identical on all species I have seen, three-lobed triangular things that, again in all species I have seen, turn a reddish color, the exact color of the root bark (and tincture), when mature. That and the flowers, once you have seen them, are the easiest ways to identify the genus.

Cultivation and Collection

This genus has been intensively cultivated and there are scores if not hundreds of cultivars and hybrids. Adding to the confusion, the plants mix with abandon in the wild and . . . well, basically they just have sex whenever and with whomever they wish and the result is a very variable genus. In any event, you can get a large number of types if you wish to grow the species yourself. The genus should grow in just about any geographical location and it has made a home in other places than the Americas by masquerading as an ornamental; it is common in the UK and the EU. It will soon (I hope) escape into the wild, where it will be found to be invasive, for in ceanothus habitat, there are some two million seeds produced per acre once the plants establish themselves. They are propelled under great force out of the capsules (to extend their range) and can remain viable for centuries. I really love these guys.

The plants are propagated by seed or cuttings. The seeds need to be scarified first (show them horror films?) and then stratified. They are usually soaked in water for 12 hours followed by chilling for 3 months—mimicking winter.

The roots/inner root bark should be harvested in the fall or early spring—whenever the root has already had a good frost. The inner bark

Properties of Red Root

Actions

First and foremost red root is a lymph system stimulant and tonic. It is anti-inflammatory for both the liver and spleen. It is also an astringent, mucous membrane tonic, alterative, antiseptic, expectorant, antispasmodic, and exceptionally strong blood coagulant.

Finding It

North and Central America, the internet, herb stores. In gardens nearly everywhere. It is commonly planted throughout the UK and the EU—though the herbalists in those regions, for the most part, have not yet cottoned on to that fact. It will grow well in those regions, or . . . you can sneak into a garden some night and dig some. Just be really, really quiet.

Alternatives: Poke root (*Phytolacca americana*) is an excellent alternative. Dosage however should be one-third that of red root. As well, poke itself contains some fairly potent antiviral compounds in the fruit, root, and so on. The pokeweed antiviral protein has been shown to cure mice of HIV infection and is apparently active against a wide range of viruses. I have a feeling about poke . . . it may turn out to be a much more important medicinal than we have thought it to be.

Cleavers will have some of the same effects but the dosage should be four times that of red root. The fresh juice of the plant is best. Cleavers, additionally, strongly inhibits elastase (by about 60 percent) and is useful for bacteria that use elastase as part of their infection strategy.

of the root should be a bright red and this color should extend through the white woody root as a pink tinge after a freeze. The root must look like this to be actively medicinal. If you get the roots in the late spring, summer, or early fall, they will be white throughout with just a hint of pink in the inner bark. They just will not work like that. It takes that cold snap to stimulate the production of the chemical constituents that you need the plant for.

Caution: The root is extremely tough when it dries. It should be cut into small 1- or 2-inch pieces with plant snips while still fresh or you will regret it. Really. Trust me on this one thing.

Store the cut and dried roots in plastic bags in large plastic bins in a cool place and they will last you for years.

Plant Chemistry

Betulin, betulinic acid, bacteriohopanetetrol, ceanothic acid, ceanothenic acid, ceanothine, ceanothamine, ceanothane, americine, integerressine, integerrenine, integerrine, methyl salicylate, a lot of tannins, flavonoids, flavonol glycosides, flavonones, dihydroflavonols. The leaves have a somewhat different profile, but I won't include it here as the root is what we are dealing with. The plant is fairly high in protein, iron, copper, zinc, magnesium and very high in calcium. The roots are nitrogen fixers and possess nitrogen-filled nodules.

Traditional Uses

Red root is an important herb in many disease conditions in that it helps facilitate clearing of dead cellular tissue from the lymph system. When the immune system is responding to acute conditions or the onset of disease, as white blood cells kill bacterial and viral pathogens they are taken to the lymph system for disposal. If the lymph system clears out dead cellular material rapidly the healing process is enhanced, sometimes dramatically. The herb shows especially strong action whenever any portion of the lymph system is swollen, infected, or inflamed. This includes the lymph nodes, tonsils (entire back of throat), spleen, appendix, and liver.

AYURVEDA

Nope.

TRADITIONAL CHINESE MEDICINE

Not remotely.

WESTERN BOTANIC PRACTICE

Red root has a very long history in the Americas. The indigenous cultures used the plant for a wide range of complaints from arthritis

to influenza, primarily as an astringent. The early American herbalists picked it up and the Eclectics then developed the use of the plant considerably, using it as an astringent, expectorant, sedative, antispasmodic, and antisyphilitic. It was used specifically for gonorrhea, dysentery, asthma, chronic bronchitis, whooping cough, general pulmonary problems, and oral ulcerations due to fever and infection. Its primary use, however, was for enlarged spleen and, to some extent, enlarged liver.

Scientific Research

There hasn't been much study on the plant, however, and really nothing looking in depth at its actions on the lymph system, including the spleen, though there are some nice hints here and there.

In recent years there has been a minor amount of exploration on the antimicrobial actions of red root. Several of the root compounds have been found active against various oral pathogens including *Streptococcus mutans*, *Actinomyces viscosus*, *Porphyromonas gingivalis*, and *Prevotella intermedia*. The flowers are active against *Staphylococcus aureus* and a couple of candida species; the roots probably are, too.

Betulin and betulinic acid, which are fairly prominent in the root, have a broad range of actions, both in vivo and in vitro: antiplasmodial, antiviral, anti-inflammatory, anthelmintic, antioxidant, antitumor, immunomodulatory. Ceanothane is a fairly strongly antistaphylococcal, antiplasmodial, and antimycobacterial. These various actions are going to have some effect on bacterial and viral diseases but exactly what and how much is not clear.

There is some evidence that red root's activity in the lymph nodes also enhances the lymph nodes' production of lymphocytes, specifically the formation of T cells. Clinicians working with AIDS patients, who have historically low levels of T cells, have noted increases after the use of red root. It is especially effective in reducing inflammation in the spleen and liver from such things as excessive bacterial garbage, white blood cell detritus in the lymph, and red blood cell fragments in the blood in diseases like babesiosis. There is evidence, clinical, that it has broad action throughout the lymph system and helps reduce not only the spleen but also the appendix when inflamed and that it stimulates lymph drainage as well in the intestinal walls.

A number of human trials have occurred using the herb as a tincture extract (usually 10–15 ml per person). The trials focused on heavy bleeding including excessive menstruation, and the plant was found to be a powerful coagulant and hemostatic in all studies. A marked reduction of clotting time was noted.

In one study, a single oral administration of 3.5–7 ml of a hydro-alcoholic (tincture) extract of ceanothus (species *americanus*) resulted in an interesting effect: At low doses accelerated blood clotting occurred within 10–20 minutes after administration. However, at higher doses coagulation *decreased* 1 hour after administration. This raises interesting speculations about the herb's range of actions.

In vivo studies have shown marked hemostatic activity and hypotensive action. In vitro studies have also found a strong reverse transcriptase inhibition and a broad antifungal activity.

7

STRENGTHENING THE IMMUNE SYSTEM

It is the body which ultimately controls infections, not chemicals. Without underlying immunity, drugs are meaningless.

—Marc Lappé

One of the great lessons from the AIDS epidemic is the realization, among the medical establishment, of the necessity for a healthy immune system. Among those with infections such as tick-borne encephalitis, influenza, Lyme, mycoplasma, and bartonella (as examples) researchers have constantly noted that the healthier the immune system, the less likely one is to be infected and, if infected, the less severe the course of the disease.

The immune system is an "organ" just as our lungs and livers are and there are things you can do to keep the immune system healthy. Regular touching is one of them, such as receiving Swedish massage on a weekly or monthly basis. Certain foods do help immune health as well. Some of the best foods that support immune health are:

- **Yogurt.** Regular intake does result in fewer sick days. The body's white blood cell count increases substantially and the GI tract bacterial community remains very healthy, which also helps. Kefir can also be used.
- **Oats and barley.** Farm animals given a mix of the two have many fewer infections, including those from influenza. (And yes, in spite of rumors to the contrary, we actually are animals, too.)

- **Garlic.** Although not as strong an antibiotic as I had formerly thought, regular garlic intake does boost immune function—in one study, those taking garlic were much less likely to catch colds and flu.
- **Selenium-rich foods** have been found to help clear influenza infections from the body. Selenium is found highest (in descending order) in Brazil nuts, fish (tuna, cod, halibut, sardines, flounder, salmon), poultry (chicken and turkey), sunflower seeds, shellfish (oysters, mussels, shrimp, clams, scallops), meat (liver, beef, lamb, pork), eggs, mushrooms, whole grains, wheat germ, onions, garlic, asparagus, broccoli, tomatoes. One ounce of Brazil nuts (usually just called "nuts" in Brazil) will supply 544 mcg of selenium—you don't need many; one Brazil nut can supply a whole day's supply of selenium. To give a comparison, tuna fish contains 68 mcg per ounce, cod 32 mcg per ounce, turkey 27 mcg, sunflower seeds 23, oysters 22, and so on.
- **Chicken soup.** Yes, it does work.
- **Black tea.** It significantly increases the immune system's interferon levels. Green tea will also be of benefit.
- **Zinc-containing foods.** Zinc is an essential mineral, especially in immune function. It enhances the actions of many of the immune system's actors, including T cells. Zinc is highest in oysters, wheat germ, liver, seeds (highest in sesame, tahini, pumpkin, squash, and watermelon seeds), roast beef, dark chocolate and cocoa, lamb, peanuts, garlic, chickpeas. To give you an idea of levels: Oysters concentrate zinc (and copper as well). One medium oyster contains about 13 mg of zinc, 3 ounces of wheat germ contains 17 mg, calf liver has about 12 mg per 3 ounces, sesame seeds contain about 8 mg per 3 ounces, and so on.
- **Mushrooms.** But not the usual store-bought variety. Shiitake and maitake can both be used in cooking, and they are both very good for raising immune function, primarily due to their high levels of polysaccharides. Their polysaccharides raise immune function considerably when taken as a regular part of the diet.
- **And of course, in the diet,** ginger, broccoli and other members of that family (all have some of the same properties as isatis), red bell peppers (which are, gram for gram, higher in vitamin C than anything else on the planet), and oregano.

And then there are the medicinal herbs.

Immune Herbs

These are my favorite three herbs for optimizing immune function. All three are tonic herbs, can be taken in large quantities, and help the immune system respond to any adverse events that may occur. They tend to act as adaptogens, that is, substances that alter the body's responses to stressors (either internal—think "illness"—or external—think "my job") in such a way as to maximize healthy functioning. If you have low energy, or a low-functioning element of the immune system, these will raise it. If you have an overabundance of energy (stressed out) or an overactive immune system, these will lower or calm function.

Of special note: These herbs also have some activity against viruses, including influenza and encephalitis viruses, making them nicely synergistic with the herbal antivirals in this book. They also very specifically reduce the cytokine cascades many of these viruses initiate *and* raise just the right immune markers necessary to reduce the viral invasion of the body. They are very good herbs. They are: astragalus, cordyceps, and rhodiola.

Astragalus

Family: Leguminosae

Species used: This is a huge genus of some 3,000 species, prevalent throughout the world. The primary species used is *Astragalus membranaceus,* a.k.a. *A. membranaceus* var. *mongholicus,* a.k.a. *A. mongholicus.* Sigh . . . now that the number of species in this genus has been, almost, settled, the number of variants is in question. (*Yes,* this one is *Astragalus membranaceus* but it looks funny. I found it in Mongolia, therefore . . .)

There is not much information on whether any of the other species in the genus can be used similarly. Most sources say not. However the Chinese are doing some good work with different species and finding a range of antibacterial, antiviral, anti-inflammatory, analgesic, and some immunomodulatory actions in them that are similar to those of the main medicinal species. The species they are looking at are *A. adsurgens, A. aksuensis, A. brachystachys, A. siculus, A. strictus, A. verrucosu,* and *A. verus,* so there are quite a few out there that are possibly good medicinals.

Synonyms: *Astragalus propinquus* is, in some circles, a synonym for *A. membranaceus*. However, a number of sources now insist (cue shocked expression) that *this* is the correct name for the plant. And of course, *Astragalus mongholicus* is just the aromatic reproductive expression of a woody perennial of the genus *Rosa* by any other name.

Common names: Astragalus (English), huang-qi (Chinese).

Part Used

The plant is a perennial with a long fibrous rootstock. The root, which is the part used for medicine, is often found thinly sliced and dried (a traditional preparation in Chinese medicine) and most closely resembles a yellow (medical) tongue depressor. Bulk quantities of the powdered or coarsely ground organic root are commonly available through herbal suppliers to Western botanic practitioners.

Preparation and Dosage

Many astragalus formulations are standardized, though I'm not sure that the literature really supports standardization with this herb.

The root is sometimes standardized for 7,4′-hydroxy-3′-methoxyisoflavone-7 (or just hydroxy-3′-isoflavone-7) but the reasons are not entirely clear for doing so. No literature exists that I can find that lays out why in fact this particular constituent was singled out and not the astragalosides. (Astragaloside IV, for instance, is one of the primary active ingredients of the plant in heart disease. It increases exercise tolerance, reduces chest distress and dyspnea, and optimizes left ventricular function.) The methoxyisoflavone constituent for which the plant is often standardized is an anabolic-type compound that enhances strength and muscle formation and may have some protective actions in upper respiratory infections and on digestive function. Data on its functions are somewhat unclear and hard to come by; I have been unable to locate any clinical or laboratory studies on the constituent—though they must exist somewhere. Their rarity stimulates speculation. A number of manufacturers, however, seem to have cottoned on to this and are now standardizing for astragalosides.

The whole root contains constituents that are essential for treating carditis and enhancing immune function. And, indeed, the majority of the Chinese studies—clinical and laboratory—were with the whole herb.

The herb may be taken as tea, powder, capsules, tincture, or in food.

TINCTURE

Tincture preparations vary considerably in their herb:liquid ratio, from 1:2 and 1:3 up to 1:5, and with alcohol concentrations ranging from 25 to 60 percent. There doesn't seem to be a lot of data on why nor what is the best tincture preparation procedures. However, there is some good evidence suggesting it be done this way . . .

In general, many of the most potent actions of the plant come from its polysaccharides, and polysaccharides are most efficiently released from the root cells by hot water. This is, in part, why many traditional uses of astragalus involve cooking it or using it as a tea. So, if you are making an extract of, let's say, 5 ounces of astragalus powder, you would then use anywhere from two to five times that amount of liquid. Many of the manufacturers whose products I think are good use from 40 to 50 percent alcohol for their astragalus tinctures in either a 1:3 or 1:5 tincture ratio. For this example, let's do it this way . . .

Start with 5 ounces of astragalus root powder and 25 ounces of liquid (this makes it a 1:5 ratio). The liquid should be half water and half pure grain alcohol, which will give you a 50 percent alcohol extraction medium. You would be using 12.5 ounces of water. Combine the root with the water *only* in a pot, and bring it to a boil (starting with cold water). As soon as it comes to a boil, turn off the heat and cover. Let it steep overnight. In the morning, put the whole mess in a jar, add the alcohol (12.5 ounces), and tighten the lid. Leave for 2 weeks, shaking when you remember to do so. Then decant.

As for dosages:

As a tonic: 30–60 drops up to 4x daily.

In chronic illness conditions: 1 teaspoon 4x daily.

As a preventive (from viral infection): 1 teaspoon 4–6x daily.

In acute conditions: 1 teaspoon 4–6x daily, generally every 3 hours.

TEA

Put 2–3 ounces of herb in 1 quart of hot water, let steep for 2–3 hours, strain, then drink throughout the day.

POWDER

In chronic conditions: 1 tablespoon 3x per day.

In acute conditions: 2 tablespoons 3x per day.

Your body's own bile and stomach acids will extract the constituents. You can go higher on these doses if you wish. The Chinese use very large doses of the powdered root, from 15 to 60 grams per day, essentially ½ to 2 ounces per day.

FOOD

Astragalus has been used for centuries as an additive to meal preparation. The sliced root is placed in soups and removed before eating or a strong infusion of the root is made and used to cook rice or as a stock for soups.

IMMUNE-ENHANCING BROTH

Robyn Landis and K. P. S. Khalsa share a tasty recipe (below) for an immune-enhancing astragalus broth in their book *Herbal Defense* (Warner Books, 1997).

Immune-Enhancing Broth

INGREDIENTS

- 3 cups water or vegetable broth
- 1 ounce astragalus (five "tongue depressor" lengths of the sliced root)
- 1 bulb (5–10 cloves) fresh garlic, sliced or whole
- Salt and pepper to taste

To make:

Combine the water, astragalus, and garlic and simmer for several hours, until the garlic is soft. Season with salt and pepper to taste. Consume all the broth if you feel an infection coming on, or take a cup or two several times during the week to prevent infection. Consume the cooked garlic separately, leave in the broth, or use as a spread on toast.

Immune-Enhancing Rice

INGREDIENTS

4 cups water, plus more as needed
1½ ounces sliced astragalus root
2 cups brown rice

To make:

Combine the water and astragalus, bring to boil, and simmer for 2 hours, covered. Remove from the heat and let stand overnight. Remove the astragalus, and add enough water to bring the broth volume back up to 4 cups. Add the rice, bring to a boil, then reduce the heat and simmer, covered, until done, approximately 1 hour. Use this rice as you would any rice, as a base for meals throughout the week.

Side Effects and Contraindications

No toxicity has ever been shown from the regular, daily use of the herb nor from the use of large doses. The Chinese report consistent use for millennia in the treatment of colds and flu and suppressed immune function without side effects.

Astragalus is contraindicated, however, *for some people*, in certain kinds of late-stage Lyme disease because it can exacerbate autoimmune responses in that particular disease. For others it can alter the Th1/Th2 balance and reduce the autoimmune dynamics. Whether or not it acts as a modulator seems to depend on individual reactions to the herb; I haven't been able to find a reason why, for some people, it exacerbates their condition and for others it does not.

Herb/Drug Interactions

Synergistic actions: Use of the herb with interferon and acyclovir may increase their effects. The herb has been used in clinical trials with interferon in the treatment of hepatitis B; outcomes were better than with interferon alone. It has also shown synergistic effects when used with interferon in the treatment of cervical erosion; antiviral activity is enhanced.

Drug inhibition: Use of the herb with cyclophosphamide may decrease the effectiveness of the drug. Not for use in people with transplanted organs.

Herb/Herb Interactions

Synergistic with echinacea and licorice in the stimulation of immune function.

Habitat and Appearance

There are over 3,000 species of astragalus in the world, 16 of which grow in the United States. The leaf structure looks like that of a typical member of the pea family. It is a short-lived, sprawling perennial and grows up to 4 feet in size.

The medicinal astragalus is native to northeast China though it has been planted a great many other places, including the United States. Wild populations are still rare in the West though astragalus is under wide cultivation as a medicinal in the United States and escape to the wild will occur sooner or later.

Cultivation and Collection

Astragalus is started from seeds in the early spring indoors. The seed coat needs to be scored with something like sandpaper prior to planting. Growers (e.g., Elixir Farm in Missouri) have found that it prefers a sunny location with, as the Elixir Farm website notes, "deep, sandy, well-drained, somewhat alkaline soil. It does not like mulch or deep cultivation. The crowns of the emerging plants are very sensitive to compost and respond well after they have gained some momentum in the spring." Not surprisingly, given the plant's medicinal actions, it is highly resistant to insect damage, crown rot, mildew, and drought.

The plant grows larger and more woody each year, with the roots harvested beginning in the fall of the third year or spring of the fourth. Spring and fall harvests occur in China. The root is generally considered too weak a medicinal if harvested prior to that time.

Properties of Astragalus

Actions

Adaptogen
Antibacterial
Antihepatotoxic
Antiviral
Cardioprotective
Diuretic
Enhances function in lungs, spleen, and GI tract
Hypotensive
Immune enhancer
Immune modulator
Immune restorative
Immune stimulant
Tonic

Astragalus is an immune potentiator and modulator. It strongly regulates interferon-gamma and interleukin-2 levels. If interferon-gamma levels are high, it is strongly active in lowering them. Enhances CD4+ counts and balances the CD4:CD8 ratio. Astragalus is specific for immune atrophy and enhances function in the spleen and thymus.

Active Against

While not specifically an antimicrobial herb, astragalus does possess some antimicrobial actions. Most important for this book, it does have antiviral activity. It is active against influenza A (H1N1, FM1), human adenovirus type 3, herpes simplex 1, Coxsackie virus B3, infectious bursal disease virus, cytomegalovirus, Punta Toro virus, Japanese encephalitis virus, porcine parvovirus, hepatitis B and significantly reduces the effects of canine distemper virus in vivo. The herb is strongly protective against infection with Japanese encephalitis virus and bunyavirus if taken prophylactically. Most of its actions come from antivirus stimulation of the immune system.

The herb does have some other antimicrobial effects. It is active against *E. coli*, *Arbiter aerogenes*, *Proteus vulgaris*, *Staphylococcus aureus*, *Salmonella enteritidis*, *Shigella dysenteriae*, *Campylobacter*, *Streptococcus hemolyticus*, *Diplococcus pneumoniae* (a.k.a. *Streptococcus pneumoniae*), *Aeromonas hydrophila*, and *Candida albicans.*

Use to Treat

All viral infections as an immune adjuvant. Many people are beginning to view the herb as a primary immunomodulator to prevent viral infection and illness. It is specific for that purpose.

It is also specific for treating myocarditis from Coxsackie B3 infection. It is specific for reducing the impacts of Japanese encephalitis virus. It is specific as a preventive for reducing the likelihood of infection from Lyme bacteria and reducing the severity of the disease.

Finding It

Herb stores everywhere and the internet.

Plant Chemistry

Astragalosides 1 through 7, astraisoflavin, astramembranagenin, astrapterocarpan, beta-sitosterol, betaine, formononetin, GABA, isoastragaloside (1, 2, and 4), isoliquiritigenin, linoleic acid, linolenic acid, soyasaponin I, kumatakenin, choline, glucuronic acid, 4′-hydroxy-3′-methoxyisoflavone-7, a couple of dihydroxydimethylisoflavones, 3′-hydroxyformonentin, calcium, folic acid, copper, iron, magnesium, manganese, potassium, sodium, zinc.

Traditional Uses

Astragalus, first mentioned in the 2,000-year-old Chinese text *Shen Nong Cao Jing*, is considered to be one of the superior tonic herbs in Chinese medicine. The plant has become one of the primary immune herbs used worldwide over the past four decades.

AYURVEDA

Five species of astragalus are used in the materia medica of India, none of them this species. They are minor herbs, used primarily as emollients.

TRADITIONAL CHINESE MEDICINE

Astragalus has been a major herb in Chinese medicine for between 2,000 and 4,000 years. It is one of the 50 fundamental herbs in Chinese medicine. Its traditional uses are for spleen deficiency with lack of appetite, fatigue, and diarrhea. It is specific for disease conditions accompanied by weakness and sweating, stabilizes and protects the vital energy (qi), and is used for wasting diseases, numbness of the limbs, and paralysis. Other uses are: for tonifying the lungs, for shortness of breath, for frequent colds and flu infections; as a diuretic and for reduction of edema; for tonifying the blood and for blood loss, especially postpartum; for diabetes; for promoting the discharge of pus, for chronic ulcerations, including of the stomach, and for sores that have not drained or healed well.

WESTERN BOTANIC PRACTICE

The herb was not used to any extent in Western botanic practice until the tremendous East/West herbal blending that began during the 1960s. It is now one of the primary immune tonic herbs in the Western pharmacopoeia.

Scientific Research

A considerable amount of scientific testing has occurred with astragalus, including clinical trials and both in vivo and in vitro studies. PubMed now lists over 5,700 citations for studies with astragalus and this does not include the many Chinese studies that have never been indexed for it. The Chinese database CNKI now has over 16,000 entries on the herb. What follows is merely a sampling.

IMMUNE FUNCTION

Most of the clinical studies and trials regarding immunostimulation have been focused on the use of astragalus in the treatment of cancer and/or as an adjunct to chemotherapy to help stimulate chemo-depressed immune function. A number of other studies have examined its immune effects with a range of different conditions.

The herb has been used with children suffering tetralogy of Fallot after radical operation to correct the condition. Tetralogy of Fallot is a complex of four heart abnormalities that occur together, generally at birth. Surgery is used to correct it. Astragalus was found to decrease abnormal levels of IgG, Igm, C3, C4, CD8+, and CD19+ while increasing levels of CD4+ and CD56+. The ratios of CD4:CD8, CD3:HLA-DR, and CD3:CD16 normalized between the second and third weeks of use. IL-6 and TNF-α both began decreasing in the first week and by the fourth week were in the normal range.

When astragalus was used in the treatment of herpes simplex keratitis levels of Th1, including IL-2 and IFN-γ, increased and Th2 levels, including IL-4 and IL-10, decreased, showing that the herb modulated Th1 and Th2 levels. This same kind of effect has been found in the treatment of numerous cancers. For example in a study of 37 lung cancer patients astragalus was found to reverse the Th2 status normally present in that condition. Th1 cytokines (IFN-γ and IL-2) and its transcript factor (T-bet) were enhanced and Th2 cytokines were decreased.

A clinical study with 63 people suffering serious abdominal traumatic injury found that the addition of astragalus to the treatment regimen significantly increased cellular immunity.

In clinical trials with a number of different cancers and congestive heart conditions, astragalus has been found to increase CD4+ levels, reduce CD8+ levels, and significantly increase the CD4:CD8 ratio. The plant has been found to have a broad immunostimulatory effect. Use of the herb with cancer patients undergoing chemotherapy found that white blood cell counts improved significantly (normalizing). The herb has been found to be specifically useful in preventing or reversing immunosuppression from any source: age, bacterial, viral, or chemical. It enhances phagocytosis and increases superoxide dismutase production from macrophages.

RESPIRATORY INFECTIONS

Eighty-eight children with recurrent respiratory infections were split into two groups. One received astragalus, the other did not. The children were followed for 1 year. Those in the astragalus group had significantly fewer occurrences.

HEART DISEASE

There have been numerous clinical trials with the herb for treating heart disease. The herb has been found specific for inhibiting Coxsackie B infections, both as an antiviral and as a heart protector. It will reverse damage to the heart in a number of conditions. With respect to Lyme carditis probably the most important of its impacts are those on left ventricular function, angina, and shortness of breath. While it is not completely protective for atrioventricular (AV) block it does improve electrophysiological parameters and ameliorates AV block to some extent.

In a trial of astragalus for 2 weeks with 19 people with congestive heart failure, 15 people experienced alleviation of symptoms of chest distress and dyspnea, and their exercise tolerance increased substantially. Radionuclide ventriculography showed that left ventricular modeling and ejection function improved, and heart rate slowed from 88.21 to 54.66 beats/minute.

In another trial, 43 people suffering from myocardial infarction were tested with astragalus. Left ventricular function strengthened. Superoxide dismutase activity or red blood cell levels increased, and lipid peroxidation of plasma was reduced.

In a study with 366 cardiac patients astragalus was found to be effective when compared to lidocaine and mexiletine (which were not found effective). With astragalus the duration of ventricular late potentials shortened significantly.

In the treatment of 92 patients suffering ischemic heart disease, astragalus was more successful than nifedipine. Patients were "markedly relived" from angina pectoris. EKG test results improved 82.6 percent.

ANTI-INFLAMMATORY ACTIVITY

Astragalus has been found to possess anti-inflammatory activity by inhibiting the NF-κB pathway and blocking the effect of IL-1ß in leukotriene C production in human amnions. The constituent astragaloside IV inhibits increases in microvascular permeability induced by histamine. The whole herb decoction has been found to reduce capillary hyperpermeability. It is strongly inhibitive of TGF-ß as well.

NEUROLOGICAL ACTIONS

Astragalus was found to improve anisodine-induced impairment of memory acquisition and alcohol-elicited deficit of memory retrieval. After use of the herb, these issues were reduced. The plant has been found to exert potent antioxidant effects on the brain, helping to prevent senility.

In one study, 106 newborns with neonatal hypoxic ischemic encephalopathy were separated into two groups. One received oral astragalus granule for 7 months, the other nimodipine for 3 months, then pyritinol for an additional 4 months. There was better recovery in the astragalus group with less long-term

negative effects from the initial condition. The incidence of cerebral palsy was markedly reduced. (Another study used injection, with similar outcomes.) Studies on the use of astragalus injection in the treatment of cerebral palsy in children found that it significantly reduced symptoms.

FATIGUE

Astragalus has been found effective in alleviating fatigue in heart patients and in athletes. In one trial, 12 athletes were randomly separated into two groups, and six were given astragalus. Astragalus was found to positively influence anaerobic threshold, enhance recovery from fatigue, and increase fatigue threshold.

A double-blind, randomized, controlled trial with 36 adults with chronic fatigue found that a mixture of astragalus and Salviae Radix significantly decreased fatigue scores.

RENAL EFFECTS

In one study, injection of astragalus was found helpful in reducing negative parameters in patients with chronic glomerulonephritis. In another study astragalus injection was used in the treatment of renal syndrome of hemorrhagic fever. One hundred forty-six people were separated into two groups. One group received ribavirin, the other astragalus. Both groups received IV glucose. The course of the disease was shorter in the astragalus group; renal function was restored more quickly.

HEPATITIS

A number of trials have found the herb effective in the clinical treatment of hepatitis B and liver disease. Liver function is improved, the liver is protected from damage, and regeneration is stimulated.

Cordyceps

Family: Ummm, errrr, uhhhh, well, let's see . . . (Counts on fingers—can I use the thumb? Is it a finger? Or not?). Ophiocordyceps? (Taxonomists really are the most irritating of people.)

Species used: *Cordyceps sinensis* almost always though *C. militaris* is considered interchangeable (and by some, stronger), and many of the others in the genus are usable as well.

In total, there are 140 or 480 or 670 members of the *Cordyceps* genus or the *Ophiocordyceps* genus or the *Metacordyceps* genus, or the *Elaphocordyceps* genus, or all of them together . . . or something. (I read the whole 55-page peer-reviewed journal article—three times—but

despite having opposable thumbs and a degree in advanced basket weaving, I still can't follow it. Let's see, the seraphim are the ones that hang from the top of the cave . . . ?).

All of these (prefix)cordyceps mushrooms are endoparasitoids (as distinct from elastoparanoids, i.e., taxonomists). This 10-dollar word simply means that they are parasitic on other living organisms, mostly insects, though a few parasitize other fungi (*turning* on their own kind). The fungus invades and takes over the host's body, replacing its tissues with its own. The main medicinal species that most people use, *Prefix-or-not-cordyceps sinensis*, is a parasite on caterpillars, specifically the larvae of the ghost moth (which is why it is sometimes called the caterpillar fungus). The fungal spores invade the caterpillar (which lives underground), and they sprout into active mycelia (which spread throughout the caterpillar body via the circulatory system), eventually killing the caterpillar (which then mummifies). The mycelia ultimately fill the corpse, leaving the exoskeleton intact, and the mushroom sprouts from the body (via the head) the next summer, and, hey, we got medicine. (Yum!)

Cordyceps species of one sort or another are common throughout the world. Each species is a parasite of either a different arthropod or the one particular mushroom species it likes to parasitize. The range of insect hosts is large: beetles, moth and butterfly pupae and larvae, ants, spiders, grasshoppers, locusts, cicadas, centipedes, bees, and cockroaches, and probably more that no one has found out about yet. Each species of cordyceps has somewhat different medicinal actions, no doubt coming, in part, from what kind of host species it infects (and no, no one has studied this as yet either).

This particular species of *I-guess-it's-a-cordyceps* that we are talking about, the primary one used in medicine, is specific to the Tibetan plateau and the Himalayas in India, Nepal, and Bhutan. It is generally hand-harvested by the local people and is, at this point in time, tremendously expensive (a recent estimate I was given—in 2012—was US$1,600 to $2,000 per pound; prices are increasing about 20 percent per year). Cordyceps mushrooms provide a major source of income for people in those regions. Several hundred tons are harvested each year, making up about half the yearly income of the local peoples and about 10 percent of Tibet's GDP.

To lower the cost and to make the herb more available, the mycelia, in China, are now grown (fermented) in vats much like penicillin and other pharmaceuticals. Those manufactured in the West are usually grown on grains (*vegan* cordyceps?). All the commercial varieties of cordyceps you are likely to find are grown, not wild.

Synonyms: *Sphaeria sinensis, Cordyceps sinensis, Ophiocordyceps sinensis.*

Common names: Cordyceps, caterpillar fungus, yartsa (or yatsa) gunbu (Tibetan), keera jhar (India), dong chong xia cao (Chinese, and it translates as "worm in winter, herb in summer"), chong cao (Chinese again, but this term usually refers to species other than *C. sinensis*), tochukaso (Japanese), aweto (Maori, New Zealand), club mushroom (United States—we are a poetic people but we walk really softly).

Part Used

Grown varieties: the mycelium. Wild-harvested: the whole damn thing—caterpillar body, fruiting mushroom, and all.

Preparation and Dosage

Cordyceps needs to be viewed as a medicinal *food*, not a raw drug to be taken in minute doses. The Chinese tonic dosages are normally rather large, 3 to 9 grams per day, and during acute disease conditions they can go as high as 50 grams, nearly 2 ounces, per day.

If you think of the herb as a food, then eating 2 ounces, say, as you do of asparagus or potatoes, doesn't seem like all that much. In China, cordyceps is often added to soups and stews (just as astragalus is) as a food ingredient for chronic illness. Sometimes the Chinese decoct it in water and drink it as a tea; however traditional healers for millennia in Tibet and India (and in parts of China) used the herb only after soaking it in an alcohol/water combination, usually the local alcoholic drink. And in fact a number of the constituents are only extractable in alcohol.

The best way to use the herb is either as a powder preparation, taken directly by mouth (allowing the stomach acids and bile, etc., to extract for you), or as a tincture.

For acute viral infections, especially in the brain and CNS, and systemic mycoplasma, especially with brain/CNS involvement, I would

recommend you buy the powder in bulk from someone such as 1st Chinese Herbs (https://1stchineseherbs.com) and then take 3–4 tablespoons of the powder blended in water or juice three times daily.

The tepid U.S. dosages, 500–1,000 mg daily, are useless for any active disease condition.

CAPSULES

The Chinese brands, if you buy capsules, run around 900–1,000 mg per capsule and the suggested dose is 6,000 mg (6 grams) per day—just for a tonic dose. If you want to use the capsules for active viral infections in the brain and CNS I would double that.

TINCTURES

As a tonic: ¼–½ teaspoon 3x daily.

For active infections: ½–1 teaspoon 3–6x daily.

Note: If you are going to make your own tincture from cordyceps powder, then use a 50 percent alcohol solution in a 1:5 herb:liquid ratio. Add the cordyceps powder to the water *only*. Starting with cold water, bring the mixture to a boil, then cover and let steep overnight. *Then* add the alcohol and let it steep for a few weeks. This will more efficiently extract the polysaccharides from the root.

Some sources recommend taking cordyceps with vitamin C to help assimilation. There isn't anything in the scientific literature on this and the Asians used the herb (and noted its beneficial effects) for thousands of years before vitamin C was discovered, so . . . not sure where that urban legend came from.

Side Effects and Contraindications

There are no side effects noted in the literature. Up to 5 grams per kilogram of body weight per day have been used in rats long term with no side effects. That would be 350 grams—i.e., about 12 ounces or ¾ pound—in a person weighing 150 pounds. Double that dose was used with rabbits for 3 months with no side effects.

The only reported side effects I can find are occasional reports of dry mouth, nausea, diarrhea. One case of an allergic reaction that subsided when the herb was discontinued.

Rant

As usual taxonomists are creating trouble for everyone who accepted their formerly completely-accurate-and-no-doubt-about-it descriptions of the natural world. Remember all those lectures we tried not to sleep through, the notes we took that concretely identified parts of the world as this and not that, the tests that we passed (or didn't), and the degrees we got (or didn't) that proved how much we knew? Well, none of it, it turns out, had much to do with the real world. (Cue shocked expression.)

In previous years—well, centuries actually—plants, and ultimately most living organisms, were classified by the system that that irritating man Carl Linnaeus developed. He spent a lot of time looking up plant skirts and describing their sexual organs and physical appearance and then putting them into groups—as did his legions of obedient automatons, I mean, followers. The classification system used in most plant field guides is still oriented around the basic framework he laid down centuries ago.

But . . . with the advent of DNA analysis everything in the natural world is being relabeled, creating a huge shift in the human lens through which the natural world is segmented into its various boxes. In the old model, hippos and whales were very different animals. In the new one, they are each other's closest living relatives. The closest living relatives of birds are now crocodiles and alligators (you can tell by the feathers). The closest living relative of the hyrax, a guinea pig–like animal (weighing 8 pounds) is now the elephant (which is the only animal that can't jump—my mind's a junkyard). And the closest living relative of the taxonomist is the measuring tape. (Who knew?)

Close examination shows that this new lens is likely to be, ultimately, as unworkable as the old one. There really isn't a basic underlying reality upon which all other things rest that will allow us to conquer our fear of the wild (allowing us to feel in control of all the lesser-evolved organisms on the planet). And as some of the new generations of naturalists are beginning to say (and as Darwin himself said long ago) evolution is not an

Herb/Drug and Herb/Herb Interactions

Cordyceps sinensis is synergistic with cyclosporine A and the amount of the drug needed is lessened if cordyceps is taken. The hypoglycemic actions of the herb also reduce the dosage needs for those on antidiabetic medications. There is some concern as well that cordyceps might be synergistic or additive with antiretroviral drugs, thus affecting dosage requirements, but nothing has yet been reported in the literature.

escalator going from there to there (with us riding triumphantly on the top step) but in reality a tremendously tangled bush all woven about itself, every branch equidistant from the center (and all equally important). As those involved in deep, perceptual observations of the real world will find, this new DNA system of ours will itself be found to be flawed (because under every cause is another cause, ad infinitum), which will lead in time to a new classification system that will, again, make all our previous maps unworkable once more.

These classification systems, again, are only *maps*. And maps are not, and never have been, the real world. (Hmmm, the GPS says that this road *does* go through, where the hell did this swamp come from? Hey! Is that an alligator? I mean a bird?) Nevertheless . . .

Once upon a time, there was a large grouping of mushrooms called the cordyceps. (And no, they really aren't mushrooms but are in fact *ascomycetes* and yeah, tomato is a fruit—but no one cares.) And for hundreds of years there were scores, nay, hundreds of mushrooms in the genus. Then came a plague of DNA scientists upon the land and one of them, after much thought, putteth down his tools and he looketh upon the multitude and sayeth, "This is so wrong." And he taketh up his measuring tape and toucheth his chalk to the board and then he writeth for those among us who haveth ears . . .

"The Species *Cordyceps sinensis* of the Genus that we have known as *Cordyceps* of the Family called *Clavicipitaceous* is no more. It is casteth out and we place it now in the Family *Ophiocordycipitaceae* and we rename the Genus *Ophiocordyceps*. And the one that was formerly called *Cordyceps sinensis* shall henceforth be known as *Ophiocordyceps sinsensis*."

And drawing the sacred dagger along the ground he declared, "So mote it be." Then a great cry went up throughout the land in-the-year-of-our-DNA-scientist 2007 and thus was this thing done. Woe be to he that heedeth it not.

Still . . .

Habitat and Appearance

The most common cordyceps medicinal species are what are called club mushrooms by all the mushroom hunters I know, though some mycofanatics are given to Latinizing, often rolling the consonants trippingly across their tongues. They are generally brownish-to-orangish in color (the mushrooms, not the tongues, though if the tongues *were* brownish-to-orangish this medicine would help clear them up). They

look somewhat like a tiny club, narrowing at the bottom, widening at the top, up to 5 inches or so tall (the mushrooms, not the mycologists, though there was this one guy . . .). Basically a very tiny version of something Fred Flintstone might use. Normally they are a bit wrinkled along the sides. There are some other species that have a cap, like other mushrooms, but I have never seen one in person; they tend to be a bit rare in my part of the woods. *Cordyceps militaris* is the one most often found in the United States; it's the only one I have met, and collected, personally, in the Rocky Mountains at 8,000 to 10,000 feet. (*C. sinensis* develops at high altitudes between 10,000 and 16,000 feet, on and in prairies rather than in forests, and tends to be more brownish; *C. militaris* tends more toward orange.)

Most of the cordyceps species specialize in their preferred hosts but *Cordyceps militaris* (go figure) parasitizes the pupae and larvae of numerous moth species and, I have heard, beetles as well. All of the cordyceps tend to sprout from the head of whatever insect they infect. (What *is* this about anyway? Doctrine of signatures? Good for treating mental disorders?) *Cordyceps*, by the way, means "club head," *cord* being club, *ceps* head, while *y* is a query referent, e.g., why.

Most of the species that have been found exist in Asia (about 100 in China alone) but there are somewhere between 5 and 20 in the United States depending on how many digits the taxonomists are using to count them. The U.S. species commonly parasitize cicadas, beetles, and moth larvae and pupae. *Cordyceps cardinalis* for example is moderately common in the southern Appalachian mountains of the eastern United States (and also in southeastern Japan). It is closely related to *C. militaris* (or *C. pseudomilitaris*, which only pretends to be violent), the species that has the largest geographical distribution, having been found on all continents except Antarctica. (Antarctica means "no bears," another useless fact I can't get out of my brain.)

C. militaris grows throughout the United States and is especially common in the Rocky Mountains, the Carolinas, and along the East Coast, often in mountainous regions. It is the primary medicinal species that is easy to find wild in this country. It is used similarly to *C. sinensis* and there have been some decent studies on its effects (150 or so on PubMed, versus 300 on *C. sinensis*).

Cultivation and Collection

I have seen photos of *Cordyceps militaris* being intentionally grown on grain. In fact, the main method to develop fruiting bodies of cordyceps, rather than just the mycelium, is to use grains as a substrate—the first used, and still most common, is rice. There is also a company in Texas, called Unicorn Bags, that sells *C. militaris* spores with detailed inoculation information on how to use live pupae (that is the stage between caterpillar and butterfly). Not my thing really but if you are excited about it look them up (https://unicornbags.com). (First, grasp the pupa firmly, then take your hypodermic needle and . . .)

There is some speculation, but there is little research on it as yet, that the fruiting mushrooms grown on grain have different medicinal actions and chemistry than those found wild and this is true of the vat-grown mycelium as well.

Studies of the gross constituents show a high similarity between the grown and wild species and, when tested, the grown varieties do have very similar impacts in the body. The one in-depth study I have seen does show a variation in chemistry—the same compounds are in both but in differing quantities, the grown having much more of some, less of a few others. One other analysis found that there were some particular compounds in the insect-host-grown cordyceps that were not in the vat-grown. Those compounds tend to be named after the insect host itself, e.g., cicadapeptins. And those compounds do have medicinal actions themselves. Nevertheless, most studies have been with the grown varieties, not the wild, and they have been shown to have range of action very close to that of the wild species.

If you wish to harvest wild cordyceps, especially in the United States, you will most likely find *C. militaris.* I, personally, don't know any of the others in their wild state though some mycofanatics do know of them, find them, and utilize them with supposedly good results. *C. militaris* does have the best research outside of *C. sinensis*; I don't think there is any doubt that the two can be considered interchangeable in action. The Chinese, in practice, apparently agree, and some even think *C. militaris* is better. So, if you want to hunt the wild cordyceps, look for *C. militaris*. (Easiest way? Join the local mycological society and go hunting with them.)

The best time to harvest the mushroom is in the summer after a good wet winter or spring—depending on the local climate they can be found

Properties of Cordyceps

Actions

Adrenogenic
Antiasthmatic
Antibacterial
Anticonvulsant
Anti-inflammatory
Antimetastic
Antimicrobial
Antioxidant
Antipyretic
Antitumor
Antitussive
Bronchial regulator
Cardiotonic
Expectorant
Hepatoprotective
Hypoglycemic
Hypolipidemic
Immunoadaptogen
Immunomodulator
Insecticidal
Mitochondrial adaptogen
Nerve sedative
Neuroprotective
Renoprotective
Sleep regulator
Steroidogenic

Cordyceps is a rather potent immunoadaptogen. If immune activity is high, it reduces it; if low, it enhances it. When taken regularly, if the immune system is stressed by, say, a bacterial organism, the herb will stimulate the immune system in just the right way to respond to the stressor while lowering the levels of or inhibiting entirely the bacterial-induced cytokines that are generated.

As a mitochondrial adaptogen, it increases oxygen utilization in the mitochondria, stimulates ATP production by the mitochondria, and protects mitochondria from adverse events. As a hepatoprotective, it offers autoimmune protection, reduces fibrosis, reduces and inhibits cirrhosis, and protects against hepatitis B. As a renoprotective, it protects from toxicity, inhibits renal failure, and reverses glomerulonephritis. And as a cardiotonic, it is hypotensive, strengthens heartbeat, is antiarrhythmic, and improves myocardial ischemia.

Active Against

Cordyceps is not primarily an antibacterial but is rather a systemic tonic and adaptogen. Still it does have some antimicrobial actions. It is active against some viruses, a few strongly so—influenza virus (H1N1, H9N2), herpes simplex virus 1, HIV-1 protease, hepatitis B, Newcastle disease virus—and a number of other microbes such as *Mycobacterium tuberculosis*, *Plasmodium* spp., *Clostridium* spp., *Staphylococcus aureus* (resistant and nonresistant), *Enterococcus faecalis*, *Bacillus subtilis*, *Candida albicans*, and various cancers (breast, thyroid, kidney, bladder, prostate, lung, Leydig cell tumor, melanoma). Its antiviral actions make it a perfect immune adjunct for use in treating most major viral infections.

The herb, while not generally active against bacteria, is, however, highly protective of the human body when bacterial infections occur. For example, in one study, mice were fed either phosphate buffered saline (PBS) or *Cordyceps sinensis* mycelium for 3 days and then infected with *Streptococcus pyogenes.* The PBS group showed bacterial dissemination throughout their bodies, while those in the cordyceps group did not. Only 40 percent of the PBS group survived until day 8, while 70 percent of the cordyceps group were still alive at day 10. In addition the PBS group showed extensive skin necrosis, none in the cordyceps group did.

Survival was significantly increased if the cordyceps group received more cordyceps every other day. In fact, *all* of the cordyceps-treated group then survived while ALT and AST levels remained normal. Use of the extract, in vitro, against the same bacterial strain showed *no* direct antibacterial activity at all.

Use to Treat

Any respiratory viral infection, any inflammation in the brain or CNS—especially encephalitis and meningitis, fatigue and weakness, especially after long illness or in chronic infections, poor mitochondrial function, chronic wasting, unproductive cough from no known cause, joint inflammation, mental fog and confusion, low libido, lung infections, kidney infections, thick mucus in the lungs that will not move, immune dysregulation, dizziness, tinnitus, nocturia, cancer. It is especially effective for mycoplasma infections.

Finding It

You can get bulk powder and capsules from 1st Chinese Herbs (https://1stchineseherbs.com) as well as many other places on the internet. If you want to spend enormous amounts of money, you can also buy the wild-crafted mushroom itself. Or . . . you can join the local mycological society (find a fun one, usually it *won't* include guys with mathematically shaven beards) and learn to find it in the wild.

from April to August. I have found them only in the mountains, in pine forests, usually in July/August. Once you've located one, carefully dig the entire mushroom, including the host insect, which will be belowground or embedded in rotted wood (or something). Bag it separately from all the other mushroom species you have collected, take it home, and dry it on an open-air tray in the dark. Watch it carefully to make sure it does not decay as mushrooms are wont to do (though these generally are not as wet as most of the other types and so are less prone to decay once picked). When dry, store in whole form in plastic bags in plastic tubs, out of the sun.

Plant Chemistry

Three constituents are, at present, considered to be the major active chemicals in cordyceps: cordycepin (a.k.a. 3′-deoxyadenosine, a purine alkaloid and a derivative of adenosine), cordycepic acid (a.k.a. D-mannitol), and cordyceps polysaccharide. Some commercial formulations are standardized for cordycepic acid (usually 10 percent), others for 7 percent cordycepin or 0.1 percent adenosine (sort of the same thing). Most are made from cordyceps mycelium and will state as much on the label.

Vat-fermented cordyceps mycelium contains a lot more cordycepin than the wild mushrooms, 40 mcg per gram versus 5 mcg/g. Cordycepic acid varies in wild populations, comprising anywhere from 7 to 29 percent by weight depending on time of year, location, and so on. The fruiting bodies contain from 30 to 85 mg per gram of cordycepic acid; the mycelial content is much higher (which is part of the reason the whole caterpillar is harvested for medicine, not just the club mushroom itself).

Cordyceps, like most mushrooms, has a very high polysaccharide content. The main one is considered to be cordyceps polysaccharide and is primarily composed of D-mannose and D-galactose in a ratio of 3:5. It runs from 3 to 8 percent by weight of the harvested fungus. Most of the rest of the polysaccharides in the herb are simply labeled by identifiers such as P70-1, CPS-1, and so on. As with many mushrooms, there are a lot of them, 36 so far.

The fungus is very high in nucleotides, the molecular components of the nucleic acids RNA and DNA. The main ones are guanosine, adenosine, and uridine in that order. The nucleotides tend to be higher, often much more so, in vat-grown cordyceps mycelium than in the wild fungus.

There are various sterols. Ergosterol is a primary one, a precursor of vitamin D_2. It is much higher in the fruiting body itself (10 mg/g) than in the grown mycelium (1.5 mg/g). Others are sitosterol, daucosterol, and campesterol.

Cordyceps has very high levels of 18 different amino acids. The mycelial powders have the highest content. Glutamate, arginine, and aspartic acid are the highest.

The mushroom also has very high levels of fatty acids, in this order: linoleic acid, oleic acid, palmitic acid.

It also contains substantial quantities of 13 different minerals (and traces of 7 more), in this order: potassium, phosphorus, magnesium, calcium, sodium, iron, aluminum, zinc, manganese, silicon, boron, copper, selenium.

And, of course, vitamins: E, K, B_1, B_2, B_{12}.

There are a few other compounds in the fungus including cordymin, various aminophenols, some unusual cyclic dipeptides, various dihydroisocoumarins, cordypyridones A and B, various diphenyl ethers, myriocin, various polyamines (cadaverine, spermidine, spermine, putrescine and so on).

The constituents of *C. militaris* are very similar.

Note: Research is showing that some of the active compounds in the various cordyceps species are specific to the insect host upon which they form, e.g., cicadapeptins 1 and 2 that *Cordyceps heteropoda* creates from the chemicals in the cicadas upon which it develops. Again, this type of research is very new and very uncommon.

Traditional Uses

Cordyceps was first recorded in Tibet in the fifteenth century in the medical text *Mennag chewa rinsel* by Zurkhar Namnyi Dorje. Oddly enough, in spite of the fact that cordyceps first appeared in Tibetan healing texts, and continued to do so through the nineteenth century, it is rarely used as a medicine there. Those who do use it do so primarily as a liquid tonic that they take throughout the day for increasing vigor and strength and as an aphrodisiac. Generally, the liquid is prepared by placing four or five cordyceps mushrooms in arak (a rice or barley liquor) and leaving it to steep in a cool, dark place for 2 to 3 months (sometimes up to a year).

While rarely mentioned in Tibetan texts, and not considered all that important an herb in that tradition, its range of actions in that system are increasing the energy of the body, increasing and restoring semen, increasing kidney strength. It is considered specific for altitude sickness.

Though the Tibetans didn't find cordyceps to be a major medicine, the Chinese did. It has, since its discovery, been a major trade item with China, sometimes worth more than gold.

The herb came to Western prominence in 1994 when a Chinese track coach insisted that his team won so handily in the 1994 Asian Games in Hiroshima, Japan, having already broken world records in the 1,500-, 3,000-, and 10,000-meter events the year before, because he had them use the herb regularly as part of their training regimen.

AYURVEDA

In spite of cordyceps being indigenous in India, there is little, if any, mention of the herb in traditional Ayurvedic texts. There is some speculation that the herb "sanjivani" mentioned in the older texts is cordyceps, but it's a guess.

In India, the use of cordyceps primarily occurs in community herbal practice, not in formal Ayurvedic healing. It is commonly used among traditional healers in Sikkim, a landlocked Indian state in the Himalayan mountains that borders both Nepal and Tibet. It is recommended as a tonic for all illnesses, improving energy, appetite, stamina, libido, endurance and normalizing sleep. It is considered to be a longevity herb and specific for colds and flu, coughs, asthma, cancer, tuberculosis, diabetes, erectile dysfunction, BHP, jaundice, and hepatitis. Although occasionally prepared as a water extraction it is generally infused in an alcoholic liquor, as in Tibet.

TRADITIONAL CHINESE MEDICINE

Cordyceps is described variously as having a neutral property and sweet taste or as being sweet/acrid with a warm property. It acts on the lung and kidney channels, is lung nourishing, kidney vital essence and vital energy tonifying, hemostatic, and phlegm resolvent, that is, a mucolytic. It is generally prescribed for overall debility after sickness and for the aged. It is considered to be one of the three primary invigorating

medicinals in Chinese medicine along with Asian ginseng and deer antler.

It is specific for tonifying the lungs, arresting bronchial bleeding, dispelling phlegm, chronic cough, asthma, wasting, and tonifying the kidneys. It is also used for impotence, low libido, poor seminal emissions, aching of loins and knees, and as a tonic for spontaneous sweating, aversion to cold, tinnitus, chronic nephritis, general weakness, and sexual hypofunction.

WESTERN BOTANIC PRACTICE

Until 1994, none. Now, lots of interest, primarily based on the Chinese and Japanese research and cordyceps's reputation as a longevity herb and aphrodisiac. (We seek our youth and we will not be denied.)

Scientific Research

Most of the scientific studies have occurred since 1995, after the Asian Games, and the numbers of published articles are increasing every year. There were four studies published in 1995, by 2011 there were 80. And those are just the ones accessible through PubMed; there are scores more in the Chinese, Japanese, and Korean databases, most not translated into English. Of those 80, only 13 were not Korean, Japanese, or Chinese and about half of those 13 were not studying the medicinal actions of the plant. The Western world is still betting that those buggy whip orders will pick up again . . . any day now.

The majority of the studies I am citing were with *C. sinensis* or *C. militaris* herbs or their isolated compounds.

IN VITRO STUDIES

Cordyceps downregulates a number of inflammatory cytokines and upregulates others such as IL-10, TGF-ß, and IL-1ra that are specific for controlling overactive inflammation responses in the body. In underactive immune systems, it will upregulate cytokines to help the body deal with disease. In overactive immune circumstances it will downregulate them.

It downregulates or inhibits NF-κB, TNF-α, IL-1ß, IL-12, NO, iNOS, SOD, elastase, luceriferase, ERK, JAK-2 (Janus kinase-2), JNK, p38, PGE2, spleen tyrosine kinase (Syk), STAT-1, AP-1, MMP-3, MMP-9, and H_2O_2 hemolysis, and it scavenges hydroxyl radicals.

When cells are challenged by lipoproteins from the bacterial cytoplasmic membrane cordyceps strongly downregulates TNF-α, IL-12, and NO. In lipopolysaccharide-activated macrophages it inhibits NF-κB, NO, TNF-α, IL-1ß, IL-6, IL-12, IFN-γ, AP-1, COX-2, the phosphorylation of p38 MAPK and Akt, as well as inhibiting PGE2 levels and suppressing Syk/NF-κB, IKKε/IRF-3, and p38/AP-1 pathways.

Treatment with cordyceps or the cordyceps constituent cordycepin, or

adenosine, causes lipopolysaccharide-stimulated macrophages to return to their original inactivated shape. This dynamic is dose dependent and needs relatively high levels of the herb.

Cordycepin suppresses TNF-α and MMP-9 expression in human bladder cancer cells, inactivates the phosphoinositide 3-kinase (PI3K) pathway in LNCaP cells, and increases levels of TIMP (tissue inhibitor of metalloproteinase) 1 and 2, and thus downregulates MMP-3 and MMP-9 in prostate cancer cells.

Cordyceps possesses a potent sphingomyelinase inhibitor that inhibits the breakdown of sphingomyelin in the body, especially in the brain, making it specific for mycoplasma. It strongly inhibits hydrogen peroxide oxidation and activity against cells and actively protects the mitochrondria (reducing oxidative stress and mitochondrial depolarization). It acts as an intracellular antioxidant and is a strong hydroxyl radical scavenger. All these actions are dose dependent.

Cordycepin strongly inhibits lipopolysaccharide-activated inflammation in microglia cells. It significantly inhibits the production of NO, PGE2, and proinflammatory cytokines in the microglia. It suppresses NF-κB translocation by blocking IkappaBalpha degradation and inhibits phosphorylation of Akt, ERK-1 and ERK-2, JNK, and p38 kinase. A compound of cordyceps, coptidis rhizoma, and Chinese skullcap was shown to have powerful neuroprotective effects on lipopolysaccharide-activated microglial cells. It inhibited NO, iNOS, COX-2, PGE2, gp91 phox, iROS (intracellular reactive oxygen species), TNF-α, IL-1ß, and IkappaBalpha degradation. It upregulated heme oxygenase-1 and increased cell viability and mitochondrial membrane potential. The three-herb compound was found to strongly protect neural cells from toxicity.

Cordyceps is strongly modulatory on immune cells. In vitro it acts as an activator and maturation stimulant to monocytes and immature dendritic cells by stimulating the expression of costimulatory molecules and proinflammatory cytokines, enhancing dendritic-cell-induced allogeneic T cell proliferation and reducing the endocytic ability of dendritic cells. *However*, during lipopolysaccharide stimulation cordyceps suppresses the proinflammatory cytokines involved. It suppresses the lipopolysaccharide-induced, dendritic-cell-elicited allogeneic T cell proliferation and shifts the immune response from a potent Th1 to a Th2 dynamic. In the absence of infection, it potentiates Th1 immune activity. During active infection, it actively modulates the extreme upregulation of lipopolysaccharide cytokines and balances the overreactivity of the Th1 response.

Cordyceps has a lot of effects on airway epithelial cells. It acts to normalize cellular function in airway epithelia by normalizing ion transport. It blocks airway inflammation by blocking NF-κB production in airway epithelial cells. It significantly reduces epidermal-growth-factor-stimulated mucus hypersecretion in lung mucoepidermoid cells by downregulating COX-2, MMP-9, and MUC5AC gene expression through blocking NF-κB and the p38/ERK MAPK pathways. It strongly regulates the inflammation that occurs in the bronchii and regulates bronchoalveolar lavage fluids by doing do. It downregulates IL-1ß, IL-6, IL-8, and TNF-α. It is highly protective of epithelia and normalizes the function of the surface epithelium.

In rheumatoid arthritis synovial fibroblasts it inhibits IL-1ß-induced MMP-1 and MMP-3 expression. MMP-1 degrades fibrillar collagens, MMP-3 the extracellular matrix. It also inhibits MAPK activation, specifically p38 and JNK. It is a fairly potent inhibitor of p38 phosphorylation.

Cordyceps is highly protective of renal tubular epithelial cells in vitro. It is antiadipogenic. It is antiatherogenic by blocking MAPK, specifically ERK, JNK, and p38. It suppresses the expression of diabetes-regulating genes. It reduces platelet aggregation.

Cordyceps stimulates ATP generation by mitochondria and also antioxidant activity, and it modulates immune responses intracellularly. It protects mitochondria from ROS and enhances the mitochondrial antioxidant defenses. The effects are dose dependent.

IN VIVO STUDIES

Cordyceps militaris, grown on soybeans, was used to prepare a hot water extract that was then given to mice infected with influenza A virus. Significantly reduced virus titers in lung tissue were observed after 3 days when compared with mice not given cordyceps. A polysaccharide, presumed to be the most active antiviral agent, was extracted and given intranasally to mice infected with lethal strains of influenza A. Mortality dropped from 70 to 18 percent. The polysaccharide was determined to be a type of arabinogalactan, similar to those extracted from larch and juniper.

In rats, cordycepin attenuated neointima formation (a thickened layer of arterial tissue) in vascular smooth-tissue muscle cells by inhibiting ROS. Cordymin, a constituent of cordyceps, was found to be strongly anti-inflammatory in induced gastric inflammation in mice by inhibiting IL-1ß, TNF-α, and total oxidant levels. It was also found to be strongly analgesic. Cordyceps (*militaris*) extract suppressed induced acute colitis in mice and significantly reduced the production of inflammatory cytokines from macrophages and mast cells. NO, iNOS, and TNF-α were all strongly inhibited. Cordyceps extract inhibited airway inflammation in rats by blocking NF-κB. It significantly inhibited ovalbumin-induced airway inflammation in sensitized guinea pigs and rats that mimics the human condition of asthma. Likewise, *Cordyceps militaris* reduced airway inflammation in a mouse asthma model.

Lipopolysaccharide-injected mice, experiencing induced inflammation, showed a remarkable reduction of IL-1ß, TNF-α, iNOS, COX-2, and PGE2 when given an extract of *Cordyceps pruinosa.*

Cordyceps extract increased CD4+ and CD8+, IL-4, and IL-10 in mice, especially in mesenteric lymph node lymphocytes. Regular daily doses of cordyceps extract prevented disuse-induced osteoporosis in rats. And an extract increased glutathione levels, reduced oxidants, and lowered blood glucose levels in rats with streptozotocin-induced diabetes.

Cordyceps extract significantly improved learning and reduced memory impairment in mice. *Cordyceps militaris* extract (and cordycepin) protected hippocampal neurons in gerbils from ischemic injury. Cordycepin was found to be strongly protective of neurons against cerebral ischemia/reperfusion. It considerably lowered levels of MMP-3 in the brain, increased SOD, and decreased malondialdehyde, significantly reducing oxidation. In one study *Cordyceps sinensis* mycelium strongly protected rat neurons from ischemic injury by inhibiting NF-κB, PMNs (polymorphonuclear neutrophils), IL-1ß, iNOS, TNF-α, ICAM-1, and COX-2. A cordymin extract pretty much did the same thing in another study. In still another study cordyceps extract protected the brain from injury after middle cerebral artery occlusion-induced cerebral ischemia in rats. And in yet another study cordycepin prevented postischemic neuronal degeneration in mice.

Cordyceps sinensis extract significantly reduced renal ischemia/reperfusion injury in rats. Various forms of renal injury in rats were ameliorated by the use of several types of *C. cicadae* extracts.

Mice exposed to ionizing radiation experienced restored immune function from a polysaccharide of *C. sinensis* through modulation of the secretion of IL-4, IL-5, and IL-17. A butanol extract of *C. bassiana* was shown to inhibit induced atopic dermatitis in mice. And in hamsters, cordycepin was shown to prevent hyperlipidemia.

Other studies have shown cordycepin to be strongly steroidogenic. It stimulated testosterone production in mouse Leydig cells. And serum testosterone and sperm count and motility were strongly increased in rats after supplementation with *C. militaris*.

HUMAN CLINICAL STUDIES AND TRIALS

Cordyceps extract inhibits the proliferation and differentiation of Th2 cells and reduces the expression of related cytokines by downregulating GST-3 mRNA and upregulating FOXP3 mRNA and relieves chronic allergic inflammation by increasing IL-10 in the blood of children with chronic asthma.

In one study, 60 asthmatic patients were split into two groups. Thirty used an inhaler, the rest used *Cordyceps sinensis* (CS) capsules. IgE, soluble ICAM-1, IL-4, and MMP-9 were all lowered in the cordyceps group (though not as much as in those using an inhaler). Another study at the Beijing Medical University with 50 asthma patients found that the symptoms in the group treated with CS were reduced by 81 percent in 5 days versus 61 percent over 9 days in the pharmaceutical group.

There have been a number of other trials of the herb in the treatment of chronic obstructive pulmonary disease (COPD), asthma, and bronchitis that have not been translated into English. The herb was effective for all these conditions; it is especially indicated for COPD.

One trial split 65 renal dialysis patients into two groups. One group took cordyceps (330 mg) and ginkgo (230 mg) three times daily for 3 months. At the end of that period microinflammation, a problem in renal hemodialysis, was significantly lowered in the herb group. Levels of hs-CRP (high-sensitivity C-reactive protein), IL-6, and TNF-α were all much lower.

In one study with 51 patients suffering chronic renal failure, the use of 3–5 grams/day of CS significantly improved renal function and increased immune function. Another study with 57 people suffering gentamicin-induced renal damage split the subjects into two groups; one received CS, the other conventional pharmaceuticals. After 6 days those in the CS group had recovered 89 percent of their kidney function versus 45 percent in the other group.

Sixty-one people with lupus nephritis were split into two groups. One received 2–4 grams of cordyceps (before meals) and 600 mg of artemisinin (after meals) three times daily for 3 years. They were observed for an additional 5 years after treatment. Twenty-six had no recurrence, four had mild, and for one the herbs did not work.

A randomized trial of cordyceps in the treatment of 21 elderly patients (divided into two groups) found that cordyceps ameliorated aminoglycoside nephrotoxicity.

Cordyceps sinensis (CS) was used in the long-term treatment of renal transplant patients. Long-term survival was no different in the treated and untreated groups, however the incidence of complication was significantly lower in the CS group. The CS group needed much lower

doses of cyclosporine A and serum levels of IL-10 in the CS group were much higher. Another renal transplant study with 200 transplant patients showed the same outcomes.

Three separate studies with a combined patient population of 756 men and women who were experiencing reduced sex drive found that after 40 days 65 percent of those taking cordyceps reported improved libido and performance versus 24 percent of those taking placebo. In another study with elderly patients complaining of decreased libido, impotence, and other sexual malfunctions 3 grams/day of cordyceps was administered for 40 days. Increased sperm survival time, increased sperm count, and decreased numbers of malformed sperm were all found in the majority of males. Improvements in hypoleukorrhagia, menoxenia, and sex drive were reported in a majority of the women.

There have been a number of clinical studies of the herb in cancer treatment, along with chemo and radiation. In one study of 50 patients taking cordyceps, tumors reduced in 23. In another, after 2 months, most patients taking cordyceps reported improved subjective symptoms. White blood counts stayed at 3000/mm^3 or higher. The use of cordyceps during radiation and chemo has been found to counteract the negative immune effects of those procedures.

There have been a number of Chinese studies on using the herb for treating heart conditions, liver problems, hypercholesterolemia, and male/female sexual dysfunction but few of them have been translated into English. There have also been a few studying exercise tolerance and improvement, e.g., 20 adults aged 50 to 75, in a double-blind, placebo-controlled trial showed improved exercise performance while taking cordyceps. However, the main studies in the United States have been on exercise tolerance with young athletes, and they all showed no improvement. The dosages were extremely low.

The best overall look at the herb, its history, and its medical uses is probably John Holliday and Matt Cleaver, *On the Trail of the Yak: Ancient Cordyceps in the Modern World* (2004).

Note: To be effective for anything, cordyceps *must* be dosed appropriately. That means a minimum dose of 3 grams daily but the best results occur with 6 grams daily as the baseline, especially in acute conditions. The renal studies usually used from 3 to 4.5 grams. This dose range can also work for lung problems, except in truly acute conditions when it should be 6 to 9 grams (in mycoplasma treatment as well).

Rhodiola

Family: Crassulaceae

Species used: There is, as usual, confusion among those with advanced degrees in plant science as to just how many species of rhodiola there are: 36, or maybe 60, probably 90. It's like stamp collectors ("No, look at that tiny ink spot on the edge, that's what makes it rare."); I just want to scream.

The primary medicinal that most people use is *Rhodiola rosea*, but many of the related species are used medicinally in the regions in which they grow. Because of the interest in *R. rosea*, the genus is being intensively studied for activity: I have found medicinal studies of one sort or another on *R. crenulata, R. quadrifida, R. heterodonta,, R. semenovii, R. sachalinensis, R. sacra, R. fastigiata, R. kirilowii, R. bupleuroides, R. imbricata, R. rhodantha,* and *R. integrifolia.*

There have been some extravagant claims (easily found on the internet) that *only* Russian *Rhodiola rosea,* harvested near the Arctic Circle (presumably by fasting virgins as the northern lights first emerge over the rim of the Earth), contains the necessary active constituents for the herb to be useful. However, *all* the *Rhodiola rosea* plants, irrespective of where they grow or in what country, have nearly identical chemistry. They are all perfectly usable as medicine.

But please note: The exact chemical profile of the *R. rosea* plants themselves differs depending on time of year, time of day, and geographical location (this valley or *that* one) irrespective of whether they are harvested at the Arctic Circle in Russia by fasting virgins or not. In other words, you can pick *R. rosea* from this location in May and again in September and the chemical profile of the plant *won't* be the same. The same is true of every species in the genus—and of every medicinal plant on Earth. Part of the art of herbalism is being able to determine medicinal potency of the plants you are harvesting by using the most sophisticated scientific instrument ever discovered—the focused power of human consciousness. Machines just aren't a reliable substitute for the capacity to reason *and* feel simultaneously. Furthermore . . . oops! Sorry. Got carried away again.

Studies on 14 other species in the genus have found the same constituents in them as in *R. rosea*. They can all be used medicinally, they all do pretty much the same things, they all work identically to the usual commercial variety *R. rosea*—see the "Scientific Research" section (page 363) for more. *Rhodiola integrifolia*, by the way, is considered to be a natural hybrid between *R. rhodantha* and *R. rosea*; you can consider it pretty much identical to *R. rosea*.

Synonyms: The rhodiolas look much like sedums and were once included in that genus, so you will see *rosea* sometimes listed as *Sedum rosea* and so on.

Common names: Rhodiola, golden root, roseroot, stonecrop, arctic root. The fresh roots smell a bit like roses, hence the origin of that name. They are golden in color, thus golden root.

Part Used

The root.

Preparation and Dosage

Generally used as capsules or tincture.

TINCTURE

Use the dried root, in an herb:liquid ratio of 1:5, with the liquid being 50 percent alcohol. Some people use a 1:3 formulation. I am not sure it is necessary.

Tonic dose: 30–40 drops 3–4x daily, usually in water.

In acute conditions: ½–1 teaspoon 3x daily for 20–30 days, then back to the tonic dose. There really isn't an upper dosage limit that I can find.

CAPSULES

The root is most often used in capsule form, 100 mg each. Usual dose is 1 or 2 capsules per day. In acute conditions up to 1,000 mg a day can be taken. The capsules are often standardized to contain 2–3 percent rosavins and 0.8–1 percent salidroside. They are usually taken just before meals.

Side Effects and Contraindications

Some people experience jitteriness from the herb; you should not take it at night until you know if you are one of them.

Herb/Drug Interactions

None noted.

Habitat and Appearance

Rhodiolas are plants that like high altitude and cold; either will do. They are a circumpolar genus of the subarctic and cool, mountainous regions of the northern hemisphere and are common in eastern Russia, parts of China, Tibet (which has many species), the mountains and northern climes of Europe, Canada, the mountainous and colder regions of the United States. The United States, Europe, and Tibet appear to have the largest populations, with Tibet having the most species.

The rhodiolas are typical succulents with fleshy, moisture-filled, grayish-green leaves. The plants grow to about 12 inches and they will have, depending on the species, a cluster of yellow, pink, red, or orange flowers at the top of the stalk. *R. rosea*'s flowers are yellow.

The root system is fairly large if the plants grow in a nutrient-rich environment. The farther north they grow, and the poorer the soils, the smaller the root.

There are three species of rhodiola in North America: *Rhodiola rosea,* which grows in the mountains of North Carolina, and in Pennsylvania through New England into Canada and all the way to the Arctic Circle; *R. rhodantha,* which grows in the Rocky Mountain states from New Mexico and Arizona up to the Canadian border; and *R. integrifolia,* which has the widest distribution in North America. It ranges from the Rocky Mountain spine (New Mexico, etc., westward) up into Canada and into the Arctic. There are populations as well in Minnesota and New York State. Most of the eastern rhodiolas are considered endangered.

If you are in the western United States and wanting to wild-harvest your own roots, look for *R. integrifolia*; it is just as useful as *R. rosea* medicinally *and* it is not endangered as many of the eastern United States *R. rosea* populations are.

Cultivation and Collection

Due to the popularizing of the plant as an antiaging and chronic fatigue medicinal, wild populations of rosea are becoming endangered; the Russians have put them on their red list of threatened plants. The largest populations of the plant were formerly in the Altai region of southern Siberia. However, over 45 companies have been harvesting the plant for export ("*Real* Russian rhodiola") and those plant populations have been severely reduced.

If you live in a region in which rhodiola grows, you can harvest your own roots; you won't need to harvest much for yourself and your family. Commercial harvesting, except for very limited amounts in abundant areas, is highly discouraged.

If you find the plant in your area, harvest the roots in the fall after seeding or in the spring just as it is coming up. The roots will be bigger and, in my opinion, more potent in the spring. Slice the bigger roots; the interior of the root will change from white to a brown or reddish color as it begins to dry.

Due to the heavy worldwide demand for the plant, there are increasing efforts to make the plant an agricultural staple in regions where it will grow; Bulgaria, Canada, and Finland are early innovators in growing the crop. The yields are low, only about 3 tons per2.5 acres, and they are labor intensive. Since the roots are taken, and only after 5 years, agricultural production of the plant demands a minimum of five fields, planted in rotation so they can be harvested in successive years in order to keep up continual production.

The seeds are tiny; 1,000 of them weigh only 0.2 gram. The germination rates are low, 2 to 36 percent; they are happier with a little stratification. Thirty days at 23° F (−5°C) will increase germination rates to 50 to 75 percent. Soak the seeds in water overnight, mix into moist soil, store for 1 month at a temperature of 36 to 39° F (2 to 4°C). You will then get about a 75 percent germination rate.

In Finland they get 95 to 100 percent germination if they sow the seeds on the surface of a sand/peat mix and keep the trays outside all winter under the snow. In April/May the boxes are brought into a greenhouse at a temperature of 64 to 72° F (18 to 22°C). Germination begins in 3 days to 1 week.

If you keep the seedlings inside for a year before transplanting, yields are significantly higher. They like sandy, loamy soil, neutral or slightly acidic (NPK: 50/50/70). They don't need additional fertilizer after the first year. The easiest method, however, is to divide the roots of an established plant and plant the root cuttings, much like potatoes.

The plant takes a minimum of 3 years to mature but the roots should not be harvested for 5 years. Dig in the fall, slice, let dry out of the sun. Store in plastic bags, inside plastic containers, in the dark.

Plant Chemistry

Most people think that salidroside (a.k.a. rhodioloside) is the most important compound in the root, while others insist it is the rosavin. Others say, yeah those and . . . rosin, rosarin, and tyrosol. Studies have found, as usual, that salidroside is much more effective when combined with rosavin, rosin, and rosarin. So, I'm guessing, just a wild shot here, that it's the whole root that is most active.

There are, of course, a great many other compounds in the root, at least 85 essential oils and another 50 water-soluble nonvolatiles. Many of the usual plant compounds are present.

Traditional Uses

Rhodiola, as far as I can tell, and in spite of assertions that it is a long-standing medicinal in traditional Chinese medicine, was a contribution to the medicinal plant world by the Russians due to their interest in adaptogens. This is pretty much a Russian-introduced category of medicinal herb—a plant that enhances general overall functioning, somewhat like a tonic but one that increases the ability of the organism to respond to outside stressors of whatever sort, diseases included. It enhances an organism's general resistance to multiple adverse influences or conditions.

The Russians have done a lot of great work on the medicinal actions of plants and deeply developed some unique categories of herbs, such as the adaptogenics. I see a lot of comments here and there picking on them, insisting that they are a dour people. But the Russians themselves say they smile and laugh only when there is truly something to smile or laugh about (which is almost never). Contrariwise they comment, "Have you ever wondered why the first thing Americans do when they meet

you is show you their teeth?" And, of course, they did not laugh when they said that (but I did, it's really funny).

Rhodiola, like the stronger preparations of eleuthero (another Russian-developed herb), is considered to be not just adaptogenic but an adaptogenic stimulant—part of the reason it can cause jitteriness and wakefulness in some. I like it and it tastes yummy (yes, that is a technical Russian term).

A few of my obscure herb reference sources reveal that rhodiola was used in traditional Russian folk medicine to increase physical endurance, work productivity, longevity, resistance to altitude sickness, fatigue, depression, anemia, impotence, GI tract ailments, infections, and nervous afflictions. But they seem to be the only people who used it regularly.

AYURVEDA AND TRADITIONAL CHINESE MEDICINE

I just can't find much mention of the herb.

Rhodiolas *have* (supposedly) been used in Chinese medicine, Tibetan medicine, and Ayurveda for a very long time—according to many reports. But my library, extensive, doesn't list the genus in any of my source books for those systems of healing. I did find some indigenous uses in Tibet, however. The plant is a part of traditional Tibetan medicine for promoting blood circulation and relieving cough. In central Asia the tea has been used for a long time as the most effective local treatment for colds and flu. Mongolian physicians use it for tuberculosis and cancer.

WESTERN BOTANIC PRACTICE

The plant never was a huge medicinal in the West even though there are traces of its use as far back as the seventeenth century in the Scandinavian countries. *Rhodiola rosea* and *R. integrifolia* were used by the indigenous tribes of Alaska as food, and the root was eaten for sores in the mouth, tuberculosis, stomachache, and GI tract troubles. The Eclectics recognized a couple of the sedums but none of the rhodiolas before their name change.

Properties of Rhodiola

Actions

Adaptogen
Adrenal protectant
Anticancer
Antidepressant
Antifatigue
Antioxidant (strong)
Antistressor
Cardiotonic (potent)
Endocrine tonic
Ergogenic
Hippocampal protectant and tonic
Hypoxia antagonist (potent)
Immune tonic
Mental stimulant
Mitochondrial tonic and protectant
Muscular stimulant
Nervous system tonic
Neural protectant
Rhodiola is also possibly a synergist; the plant is strong inhibitor of CYP3A4 and P-glycoprotein.

Active Against

Again, this herb is not primarily an antimicrobial but it does have some antiviral actions. It is active against influenza viruses due to its neuraminidase inhibitory activity. It has been found active against H1N1 and H9N2 viral strains. It is also active against the hepatitis C and Coxsackie B3 viruses. One of its constituents, kaempferol, is specific against Japanese encephalitis virus and enterovirus 71. It has some antibacterial activity as well, against *Staphylococcus aureus* (strong), *Bacillus subtilis* and *Mycobacterium tuberculosis* (moderate), *E. coli* (weak).

Use to Treat

Chronic long-term fatigue, recurrent infections, recovery from long-term illness and infections, nervous exhaustion, chronic fatigue syndrome, chronic disease conditions with depression, low immune function, brain fog, and to accelerate recovery from debilitating conditions.

Note: The plant is specific for the kinds of damage that occur during encephalitis infections. It is highly neuroprotective and strongly anti-inflammatory in the brain and CNS. It should be used in all encephalitis infections.

Other Uses

The leaves of most species can be eaten, chopped finely and added to salads, or cooked as a pot herb. The plants are very high in vitamin C, with 33 mg per gram of fresh plant.

Finding It

You can buy it pretty much everywhere. If you live in the right climate you can probably find it wild or grow it yourself.

Scientific Research

There is a lot of research on this plant right now, and more studies are occurring daily. There have been, unlike the case for many other newish medicinal plants, a lot of human clinical trials with this herb. I am primarily going to look at the neuroprotective/neuroregenerative, immune, and antistress/antifatigue actions of the plant—they are strongly interrelated. The potent antioxidant actions of the plant are deeply interrelated with those as well.

NEUROPROTECTIVE/NEUROREGENERATIVE

In vitro: Compounds in both *Rhodiola sacra* and *R. sachalinensis* protect neurons against beta-amyloid-induced, stauroporine-induced, and H_2O_2-induced death. Salidroside, a common compound in many rhodiolas, protects cultured neurons from injury from hypoxia and hypoglycemia; protects neuronal PC12 cells and SH-SY5Y neuroblastoma cells against cytotoxicity from beta-amyloid and against hypoglycemia and serum limitation; and protects neurons. It does so by inducing the antioxidant enzymes thioredoxin, heme oxygenase-1, and peroxiredoxin-1, downregulating the pro-apoptotic gene Bax, and upregulating the antiapoptotic genes Bcl-2 and Bcl-X(L). It also restores H_2O_2-induced loss of mitochondrial membrane potential and restores intracellular calcium levels.

In vivo: *Rhodiola rosea* enhances the level of 5-hydroxytryptamine in the hippocampus, promotes the proliferation and differentiation of neural stem cells in the hippocampus, and protects hippocampal neurons from injury. *R. rosea* protects against cognitive deficits, neuronal injury, and oxidative stress induced by intracerebroventricular injection of streptozotocin. Salidroside protects rat hippocampal neurons against H_2O_2-induced apoptosis. A combination of rhodiola and astragalus protects rats against simulated plateau hypoxia (24,000 feet). It inhibits the accumulation of lactic acid in brain tissue and serum.

Human clinical trial: A double-blind, placebo-controlled, randomized study with 40 women, ages 20 to 68, who were highly stressed, found that a *Rhodiola rosea* extract increased attention, speed, and accuracy during stressful cognitive tasks. Similarly, *Rhodiola rosea* was used with 120 adults with both physical and cognitive deficiencies (exhaustion, decreased motivation, daytime sleepiness, decreased libido, sleep disturbances, concentration deficiencies, forgetfulness, decreased memory, susceptibility to stress, irritability); after 12 weeks, 80 percent of patients showed improvements. In another study, a combination formula (Xinnaoxin capsule) of *Rhodiola rosea, Lycium chinense* berry, and fresh *Hippophae rhamnoides* fruit juice was given to 30 patients with chronic cerebral circulatory insufficiency; after 4 weeks the condition was significantly improved. A double-blind, crossover 3-week study on stress-induced fatigue on the mental performance of healthy physicians during night

duty found that *Rhodiola rosea* extract decreased mental fatigue and increased cognitive functions such as associative thinking, short-term memory, calculation and concentration, and speed of audiovisual perception.

ANTIFATIGUE/ANTISTRESS

In vitro: Salidroside stimulated glucose uptake by rat muscle cells. *Rhodiola rosea* extract stimulated the synthesis or resynthesis of ATP and stimulated reparative processes in mitochondria.

In vivo: *Rhodiola rosea* extracts increased the life span of *Drosophila melanogaster*, lowered mitochondrial superoxide levels, and increased protection against the superoxide generator paraquat. Four weeks' supplementation with *R. rosea* extract significantly increased swimming time in exhausted mice—it significantly increased liver glycogen levels, SREBP-1 (sterol regulatory element binding protein 1), FAS (fatty acid synthase), heat shock protein 70 expression, the Bcl-2:Bax ratio, and oxygen content in the blood. Salidroside protected the hypothalamic/pituitary/gonad axis of male rats under intense stress—testosterone levels remained normal rather than dropping, secretory granules of the pituitary gland increased, and mitochondrial cells were strongly protected. *R. rosea* extract completely reversed the effects of chronic mild stress in female rats—that is, decreased sucrose intake, decreased movement, weight loss, and dysregulation of menstrual cycle. Rhodiola suppressed increased enzyme activity in rats subjected to noise stress—glutamic pyruvic transaminase, alkaline phosphatase, and creatine kinase levels all returned to normal, and glycogen, lactic acid, and cholesterol levels in the liver also returned to normal. *R. rosea* reduced stress and CRF-induced anorexia in rats. And so on.

Human clinical trial: Twenty-four men who had lived at high altitude for a year were tested to see the effects of rhodiola on blood oxygen saturation and sleep disorders; rhodiola was found to increase blood oxygen saturation significantly and increase both sleeping time and quality. In a double-blind, placebo-controlled study of the effects of *R. rosea* on fatigue in students caused by stress, physical fitness, mental fatigue, and neuro-motoric indices all increased (other studies found similar outcomes). *R. rosea* intake in a group of healthy volunteers reduced inflammatory C-reactive protein and creatine kinase in blood and protected muscle tissue during exercise. *Rhodiola rosea* in a placebo-controlled, double-blind, randomized study was found to increase physical capacity, muscle strength, speed of limb movement, reaction time and attention—in other words it improved exercise endurance performance. A similarly structured study found that 1 week of rhodiola supplementation decreased fatigue and stress levels but more interestingly decreased photon emissions on the dorsal side of the hand. In another study *Rhodiola rosea* increased the efficiency of the cardiovascular and respiratory systems and prevented fatigue during an hour of continuous physical exercise. A phase three clinical trial found that rhodiola exerts an antifatigue effect that increases mental performance and concentration and decreases cortisol response in burnout patients with fatigue syndrome; other studies have found similar outcomes including the amelioration of depression and anxiety.

IMMUNE ACTIONS

In vitro: *Rhodiola imbricata* protects macrophages against tert-butyl hydroperoxide injury and upregulates the immune response. Additionally it potently stimulates the innate immune pathway and initiates strong immunostimulatory actions, increasing Toll-like receptor 4, granzyme B, and Th1 cytokines. *R. sachalinensis* extract enhances the expression of iNOS in macrophages. *R. quadrifida* stimulates granulocyte activity and increases lymphocyte response to mitogens. *R. algida* stimulates human peripheral blood lymphocytes and upregulates IL-2 in Th1 cells and IL-4, IL-6, and IL-10 in Th2 cells.

In vivo: *Rhodiola kirilowii* enhances cellular immunity, stimulating the activity of lymphocytes and increasing phagocytosis in response to microbial organisms. *R. imbricata* enhances specific immunoglobulin levels in response to tetanus toxoid and ovalbumin in rats—the plant has adjuvant/immunopotentiating activity in both humoral and cell-mediated immune response.

Human clinical trial: *Rhodiola rosea* (in combination with schisandra, eleuthero, and leuzea) significantly increased both cell-mediated and humoral immune response in ovarian cancer patients. Rhodiola significantly reduced problems and infection after the treatment of acute lung injury caused by massive trauma/infection and thoracic-cardio operations. A combination formula of rhodiola, eleuthero, and schisandra significantly enhanced positive outcomes in the treatment of acute nonspecific pneumonia. *R. rosea* increased the parameters of leukocyte integrins and T-cell immunity in bladder cancer patients.

OTHER ACTIONS

Rhodiola, various species, has been found effective in the treatment of breast cancer. It inhibits the tumorigenic properties of invasive mammary epithelial cells, inhibits superficial bladder cancer, suppresses T241 fibrosarcoma tumor cell proliferation, and reduces angiogenesis in various tumor lines. *R. imbricata* is highly protective in mice against whole-body lethal radiation.

The plant has also been found highly antioxidant in numerous studies, to be liver protective, and to be highly protective of the cardiovascular system.

The plant is adaptogenic; that is, it increases the function of the organism to meet whatever adverse influences are affecting it, whether stress or illness. Most of the attention has been paid to its ability to increase endurance and mental acuity but its effects on the immune system, though less studied than eleuthero's, are similar.

EPILOGUE

WHAT THE FUTURE HOLDS

Once your life is saved by a plant
Things are never the same again.

We live in interesting times. Although most of us, in the West, were trained to see the world around us as stable, unchanging year after year, that is an anomaly in the long history of this planet. The Earth goes through long periods of stability, then, rather abruptly, things change. The ecological parameters of climate alter and I am not just talking about "climate change" here. Wind patterns shift, currents in the ocean change, animal migration patterns, rainfall, snowfall, soil composition, insect density, mouse populations, and so on—all of them shift. They shift for reasons that few reductionist scientists understand—or want to. There are patterns inside the living physical world that few of us look for or notice, invisible patterns, and upon them our survival depends.

We are just one part of that incredibly large, complex, deeply interwoven ecological scenario, one organism among trillions in a matrix that has lasted billions of years. We aren't, and never have been, in charge. So, things change, as they are wont to do, and they are in the process of changing drastically. None of us will escape the consequences of it.

One of those consequences happens to be the emergence of new disease organisms, their unique movements through the ecological fabric of the world, and their infection of new species, most especially us. (The Covid-19 pandemic, which started 8 years after I wrote this passage for the first edition of this book, is only the beginning of what we face.)

The medical paradigm that most of us in the West know emerged out of a certain historical context operating against the background of a stable ecosystem. It has been shaped by that unique situation *and* by the interests, and hubris, of powerful corporations, educational organizations, government bureaucracy, nongovernmental activist groups, and self-interested trade unions (i.e., medical doctors)—all of whom still assume that the planet is a stable background against which they can

operate. It has most certainly not been shaped by the needs of those who become ill or a genuine understanding of disease organisms and the ecological matrix in which all life forms on this planet are embedded. For anyone who looks, the fraying fabric of that stability is clear to see, and so also the crumbling of the Western medical paradigm.

The Western medical paradigm is failing. It is failing because it is inherently dysfunctional and most especially because it does not accurately understand the nature of disease, most especially the nature of the organisms it has considered responsible for most of those diseases. In the coming decades, within 10 years if some bacterial and viral researchers are to be believed, we will see not only the emergence of microbial diseases more potent than any our species has heretofore experienced but the failure of most antimicrobial pharmaceuticals that medical science uses—primarily due to resistance problems. This means that the kind of complacency that has been in place for most of us throughout most of our lives will have to change. We will no longer be able to go to a physician to cure microbial disease; we will be forced back on our own resources. This is a scary prospect for the emotionally dependent, which all of us have been, at one time or another, when it comes to illness.

And so, we enter difficult times. But as old systems fail, out of the shards, and out of the human capacity that our species has always possessed, we will, of necessity, create something new, something that really does work better and that does reflect the world in which we live more accurately. Ironically, that will include a return to plant medicines as our primary healing agents for infectious diseases.

Some of my ancestors, powerful political physicians, actively worked to destroy the Western tradition of herbal medicines, feeling that they were the outmoded and tragic remnants of a superstitious past. They felt that science would offer the answers, *all the answers*—that through science we could defeat all disease organisms on this planet. It is fascinating to me that in the midst of the failure of that utopian and very psychological projection the plants are returning once again to help us in our lives and with our diseases. They have been here 700 million years, some of them, others a mere 170 million. And they have learned a thing or two in that time. We, here a few hundred thousand years (or perhaps a million or two if you take into account earlier expressions of

Homo spp.), have a great many things yet to learn, among them humility. It is no accident, I suspect, that the Cherokee peoples have repeated a legend for generations to their children, a legend that tells of the time plants were asked by the animals and insects (whom the humans had harmed by their lack of awareness) to turn on humans and give them diseases (just as the animals and insects were doing). The plants thought it over and said, "No, we will not, for they are our children. And for every disease you create for them, we will make a cure. And when they come to us in their need, we will heal them."

We face difficult times, but interesting ones as well. A new paradigm of healing is emerging, one partly based in the older healing systems of the human species (including technological medicine) but one that also contains elements never known before. In *your* own genius resides aspects of that new paradigm. I invite you to bring it, in whatever form it manifests, into the world. We are all going to need each other's help, you know, and we might as well start now.

In veriditas veritas
Silver City, New Mexico

APPENDIX

A BRIEF LOOK AT HERBAL MEDICINE MAKING

[Our bodies] are not distinct from the bodies of plants and animals, with which we are involved in the cycles of feeding and the intricate companionships of ecological systems and of the spirit. They are not distinct from the earth, the sun and moon, and the other heavenly bodies. It is therefore absurd to approach the subject of health piecemeal with a departmentalized band of specialists. A medical doctor uninterested in nutrition, in agriculture, in the wholesomeness of mind and spirit is as absurd as a farmer who is uninterested in health. Our fragmentation of this subject cannot be our cure, because it is our disease.

—Wendell Berry, *The Unsettling of America*

Tremendous empowerment comes from learning to recognize the medicinal plants that surround us, even more in learning how to make them into medicines for healing. And though it takes time, as your knowledge increases, as you learn how to tend to your illnesses and those of your family, the sense of helplessness that so many of us have experienced when we become ill, often ingrained since birth, begins to dissipate.

We have been trained to place our health in the hands of outside specialists who, very often, know neither ourselves nor our families, not the fabric of our lives nor the communities in which we live. They have no understanding of, and often no interest in, the complexity in which we live and from which our illnesses emerge. But for most of us, those specialists are the *only* place we know to go when we are ill, uncertain, and afraid, to seek help—for ourselves or our loved ones.

The world, however, is a great deal more complex than that frame allows and there are many more options to healing than that system acknowledges. All of us live, all the time, in the midst of a living pharmacy that covers the surface of this planet. And that living pharmacy is

there for you, or anyone, to use—anytime you wish. Once you *know* that, once you have been healed by the plants in that living pharmacy, often of something that physicians said could not be healed, things are never the same again. You begin to break the cycle of dependence on which the health care system depends.

Taking back control over personal health and healing is one of the greatest forms of personal empowerment that I know. It does take time and effort, this kind of learning, but the learning goes quickly. Harder, perhaps, is learning to trust the plants with your life. It is a truly frightening moment, that moment of decision, when trust is extended in that way, for, before it occurs, there is no way to experientially *know* what the outcome will be. Most people on this planet, though, people who do not live in the Western, industrialized nations, make that decision every day of their lives. It is a trust they extend every moment of every day. Trusting the healing capacities of the plants is not a new experience to the human species.

The next step is learning how to turn the plants you are learning about into medicines for yourself and your family. It isn't that hard—people all over the globe have been doing it for a hundred thousand years. At least.

This is a brief look at herbal medicine making. It is condensed from a much larger exploration in my book *Herbal Antibiotics,* second edition.

The Different Kinds of Herbal Medicines

Herbal medicines, in general, fall into two groups: 1) those for internal use, and 2) those for external use.

The main forms of herbal medicines for internal use are:

- Water extracts (infusions and decoctions)
- Alcohol extracts (tinctures)
- Percolations (water or alcohol)
- Fluid extracts
- Syrups/oxymels/electuaries
- Glycerites
- Fermentations
- Vinegars
- Fresh juices (stabilized or not)
- Powders (plain or encapsulated)
- Food
- Suppositories/boluses
- Douches
- Essential oils
- Steams
- Smokes

The main forms of herbal medicines for external use are:

- Oil infusions
- Salves
- Evaporative concentrates
- Washes
- Liniments
- Lotions
- Compresses/poultices
- Essential oils
- Smudges

Most of these you can make yourself. In this condensed version, I will primarily look at alcohol and water extractions.

A Comment on Solvents

Unless you are using the plant itself in some form—as powder, food, juice, or so on—what you will be doing when you make your medicines is extracting the chemical constituents of the plant in some kind of liquid solvent. (When you take the whole herb internally, the stomach acids, bile salts, and so on *are* the solvent media. They leach out the active constituents of the plants for you.)

Every solvent has its own properties and people use different ones for many different reasons, some of which I will go into here. Generally, a solvent is referred to as a *menstruum.* The term comes from *menstruus*, a Latin word meaning "month." It was felt, in the old days, that the moon and its cycle of 28 days had an influence on liquids, just as it does on the tides. So, herbs were placed in liquids—on particular days by the fanatical—and left in there for one cycle of the moon. Hence *menstruum.* Though derided as superstition by scientists there is some legitimacy to this kind of thinking. Plants really are stronger when harvested on certain days, the moon does affect the underground aquifers of the Earth, just as it does the oceans (causing the ground to breathe out moisture-laden air), leeches really are useful (surgeons use them regularly now), maggots really do clean gangrenous wounds better than anything else, and . . . oops, sorry, got carried away again.

Anyway, the solvent is called the *menstruum*. Herbs are placed in the menstruum, and once there they begin to *macerate*. Maceration is the soaking of something—usually a plant of some sort—in a solvent until the cell walls begin to break down so the compounds in the herb will leach into the solvent, where they are held in suspension. When you later separate the liquid (containing the medicinal compounds) from the solids, the solids that are left are called the *marc*. The liquid is called whatever kind of medicine you are making: tincture, infusion, or so on.

Water is considered to be *the* universal solvent; it works for most things to some extent. For most of human history it has been the primary solvent people have used. Alcohol is the next most effective solvent. Combining them will give you the most comprehensive solvent medium that exists.

Just as with the plants you harvest, use the best-quality solvents you can get. Your water, especially, should be well, spring, or rain water—if you can get it. If you use tap water, have a filter on the water line if you are at all able to do so. Or else buy a good-quality water. The better the water, the better the medicine. (Tap water is, as well, filled with minute quantities of pharmaceuticals—you really don't want to ingest them. They are highly bioactive.)

Another thing to understand is that the more finely powdered your herb, the more surface area is exposed to the solvent. This allows more of the chemical constituents to leach into the solvent.

When you are making extracts, part of what you learn, and develop in your practice, is knowledge of just what kinds of solvents are right for which herbs and in what combinations. The goal is to get as many of the medicinal compounds as possible into the extractive medium. Each herb is different and needs different combinations of water and alcohol—that is, a different formula for preparation. Some do better in pure alcohol, some in pure water. Some need oils to extract the active constituents (*Artemisia annua* is an example of this; artemisinin is more easily soluble in fats than in either alcohol or water). Some need boiling, some prefer cold liquids.

Pharmacists, prior to World War II (before pharmaceuticals began to dominate medicine) were extensively trained in very sophisticated forms of herbal medicine making—many of which are beyond the scope of this book (and of most pharmacists these days). This is why pharmacists are still called "chemists" in England and the drugstores there the "chemist's shops." Distressingly, that kind of training no longer occurs; it is now a lost art. I doubt there is a medicinal pharmacist in practice anywhere in the world who can prepare a tincture of *Colchicum officinale* and determine, exactly, the amount of colchicine in it—as all pharmacists could do in 1920.

In becoming an herbal medicine maker, you are learning how to be a practical dispensing pharmacist. Part of what that means is discovering how to best prepare the herbs and with which solvents. A brief description follows; my book *Herbal Antibiotics* includes an herbal formulary that will give you the ratio of alcohol and water for several hundred plant tinctures.

Water Extractions

The two most common forms of water extractions are infusions and decoctions.

Infusions

Teas are, at heart, weak infusions. When making medicine, however, you are usually working with what would formally be called an infusion. Infusions are stronger than teas since the herbs sit, or *infuse*, in the water for a much longer period.

An infusion is made by immersing an herb in either cold or hot (not boiling) water for an extended time. Again, the water you use should be the purest you can find, *not* tap water. Water from rain, a healthy well, or a spring is best.

The weakness of infusions, cold or hot, is that they do not keep well; they tend to spoil very quickly. Refrigeration will only slow the process a little. Infusions, unless you stabilize them with something like alcohol, need to be used shortly after you make them. Their strength is that nearly everyone has access to enough water to make them without resorting to the expense of buying alcohol.

HOT INFUSIONS

The following guidelines are for hot infusions and will work with most herbs. Although these guidelines use short timelines for hot infusions, I often make my infusions at night just before bed and let them infuse overnight. I usually make enough for 1 day, then drink the infusion throughout the next day.

Most *hot* infusions are consumed, confusingly, not hot but warm or at room temperature; the infusion periods are too long for the water to stay hot. *Hot infusion,* in this sense, is a description of the extraction process, not of its temperature when used.

Some infusions, however, are best consumed while still hot; often these are diaphoretics that stimulate sweating. An infusion of yarrow, if being used to stimulate sweating to help break a fever, is best consumed hot (steeped 15 minutes, covered). If being used for GI tract distress or to stimulate menstruation it is best prepared as a hot infusion (covered), then consumed hours later at room temperature.

To prepare a hot infusion, bring water to a boil, then pour it over the herb in the following manner:

For leaves: 1 ounce per quart of water, let steep 4 hours, tightly covered. Tougher leaves require longer steeping. The more powdered the leaves (if dried), the stronger the infusion. If you are using fresh leaves, cut them finely with scissors or chop them as finely as possible with a sharp knife.

For flowers: 1 ounce per quart of water, let steep 2 hours, tightly covered. More fragile flowers require less time. Most flowers can be infused whole.

For seeds: 1 ounce per pint of water, let steep 30 minutes, tightly covered. More fragrant seeds such as fennel need less time (15 minutes), rose hips longer (3 to 4 hours). Most seeds possess very strong seed coats to protect them from the world until they sprout. You will need to break the seed coat in order for the solvent to work; the seeds should be powdered as finely as possible.

For barks and roots: 1 ounce per pint of water, let steep 8 hours, tightly covered. Some barks, such as slippery elm, need less time (1 to 2 hours). Most barks and roots are infused after being dried; powder them as finely as possible. If you are using fresh roots, mince them as finely as possible.

If you keep the containers tightly covered, the volatile components in the herb will remain in the liquid rather than evaporating into the air. The heat will vaporize the volatiles and they will rise up in the steam, then collect on the underside of the lid. As the mixture cools, the volatiles will condense and drip from the lid back into the infusion. This ensures that the essential oils, which are very volatile, will still be present. You can easily identify an herb that has a high volatiles content; it will have a strong essential oil or perfumey smell to it. These must always be covered when making a hot infusion.

When you are ready to use the infusion, pour off the water and squeeze out the marc as much as possible. The liquid in the saturated herbs is often much stronger than the infused liquid, so keep it if you can.

COLD INFUSIONS

Cold infusions are preferable for some herbs. The bitter components of herbs tend to be less soluble in cold water. Yarrow, for instance, is much less bitter when prepared in cold water. Usually cold infusions need to steep for much longer periods of time, though each herb is different. The necessity for a cold infusion rarely arises; nevertheless, it may. If so, place the herb in room-temperature water, cover, and let steep overnight.

Measuring Herbal Medicines

It seems nearly everyone uses a different way to describe how much to take; some say milliliters (ml), some say drops, some say dropperful, some say teaspoon or tablespoon, so here is a conversion table for you. It may help.

A drop: A drop is not always a drop (see why there's confusion?). A drop of water and a drop of alcohol are about the same, but a drop of glycerine is bigger—about five times bigger than a drop of water—because it is so viscous. Nevertheless, pretty much everyone treats a drop as a drop. Now, is that clear or what?

Dropperful: A 1-ounce glass tincture bottle has a standard glass dropper that fits in it, and when it's full of tincture that is what I call a dropperful. It generally holds around 30 drops, so I consider a dropperful to be 30 drops, or 1.5 ml. Normally, a glass dropper will fill only halfway with one squeeze, so it takes two to get a full dropper.

A milliliter is, for water or alcohol, 20 drops, or two-thirds of a dropperful.

A teaspoon is 5 ml or 100 drops or three and one-third dropperfuls.

A tablespoon is ½ ounce, 15 ml, 3 teaspoons, 300 drops.

An ounce is in the neighborhood of 600 drops.

INFUSION EQUIPMENT

There are many kinds of infusion pitchers and mugs available; they are pretty common. Most of them have some form of basket in which to place the herbs (and a lid to cover them). The basket is suspended at the top of the mug or pitcher so that the herbs and the liquid do not mix together. It does make it a bit easier. (Avoid plastic if you can; use stainless steel, glass, or pottery infusers.) You can also buy (or make) small cloth bags to hold the herbs, which you then suspend in whatever container you are using. A tea ball will also work but I don't find them as effective; they don't usually hold enough herb.

The best infusers work by holding the herb in the upper part of the pot, so that only the upper portion of the liquid is in contact with the herb. As the water at the top of the infuser becomes saturated with the herbal constituents, it gets heavier and sinks to the bottom. This creates a circulating current in the water that brings the unsaturated water to the top of the jar where it can then infuse as well. This will make the strongest infusion. You can also just put the herb in a jar with hot water and cover it; it will work fine but it won't be quite as strong.

A Hot Infusion for Parasites

INGREDIENTS:

- **2 ounces fresh ginger root, chopped finely**
- **2 ounces dried sida leaf**
- **2 ounces dried wormwood leaf (*Artemisia absinthium*)**
- **2 quarts water**

To make:

Place herbs in container, pour near-boiling water on top, cover tightly, and let sit overnight. Strain and press the marc to remove as much liquid as possible. Drink 1 cup four times per day. This recipe will make enough to last 2 days. Continue for 8 days (making the infusion again as necessary). This is a good infusion for treating intestinal worms (you can just use the wormwood and ginger if you wish). It will be very bitter, though the ginger will help that a bit.

Decoctions

Decoctions are much stronger than infusions. Basically, they are boiled infusions. There are two forms of decoctions: 1) simple decoctions, and 2) concentrated decoctions. A simple decoction is any water extract that is boiled for a short length of time. Concentrated decoctions are boiled until the water is reduced to some extent. Normally, herbs that are highly resinous or filled with volatile oils are not decocted. Only herbs whose constituents are not damaged by heat are boiled.

It is important to begin with cold water, not warm or hot, then add the herbs and bring it to a boil. The extraction will be more efficient if you begin with cold water because different constituents extract better at different temperatures.

Some herbs, such as isatis, are stronger if they are boiled for a few minutes simply because the higher heat is a better extractant. Herbs high in polysaccharides, such as reishi, are also often helped by boiling; polysaccharides tend to extract more efficiently when decocted. In essence, anytime an herb is boiled, no matter how short a time, it is considered to be a decoction. If you are just boiling the herb to better extract the constituents, you are making a simple decoction.

A Simple Decoction

INGREDIENTS:

1 ounce herb **1 pint cold water**

To make:

Combine the herb and water. Bring to boil. Boil for at least 15 minutes (some herbs will need longer). Let cool enough that you can handle it. Strain the decoction to remove the herb. Press the drained herb to remove all the liquid.

Add enough water to the liquid to bring it back to 1 pint. Take as directed.

In a concentrated decoction, which is more common than simple decoctions, the herb is boiled in water long enough that the amount of water you began with is reduced to some extent, often by half, sometimes more. This acts to concentrate the constituents in less liquid,

making the medicine stronger. Concentrated decoctions are not often drunk as a tea (reishi is an exception). However, they are sometimes used in smaller doses similarly to a tincture. Once the decoction is made it is allowed to cool, the liquid strained, then dispensed a tablespoon at a time—usually three or four times a day depending on the herb and the disease. The usual dosage range for concentrated decoctions, depending on the herb, is 1 to 4 fluid ounces a day.

The most common form of medicine made from concentrated decoctions is a cough syrup. They are also used to make fomentations—that is, very condensed water extracts that are soaked into a cloth and applied to the surface of the body (to treat pain and inflammation in a joint, for example). Decoctions are also used as enemas—should the need arise, which everyone hopes it won't. This gets a very strong concentrate into the bowel where it will, usually, rather easily move across the membranes of the colon into the bloodstream.

When you are making your concentrated decoctions, use porcelain, glass, or stainless steel pots if you can; iron and aluminum will often contaminate the mix. When the decoction is cool, prepare it as needed for whatever you are going to use it for. Concentrated decoctions will last longer than infusions, especially if kept cold. Syrups will often last a year in a refrigerator just fine.

A Concentrated Decoction for Sore Throat and Upper Respiratory Infection

INGREDIENTS:

- 1 ounce dried elderberries
- Pinch of cayenne
- 3 cups cold water
- Wildflower honey
- Juice of 1 lemon

To make:

Combine the elderberries and the cayenne with the water. Bring to a boil, then reduce the heat and simmer, uncovered, until the liquid is reduced by half. Let cool enough that you can work with it. Strain the liquid and press elderberries to remove as much liquid as possible. Add wildflower honey to taste. Add the lemon juice. Store in the refrigerator. Take 1 tablespoon or more as often as needed at the onset of sore throat or upper respiratory infections.

Alcohol Extractions

Because alcohol extractions, i.e., tinctures, keep so well over time and because they are so easily dispensed, many herbalists prefer them over infusions. They are made by immersing a fresh plant in full-strength alcohol or a dried plant in an alcohol and water mixture.

I am a fan of using pure grain alcohol for tinctures. What that means in practice, however, is using an alcohol that is 190 proof, or 95 percent alcohol. (There is such a thing as 100 percent or 200-proof alcohol, but the only people who generally use it are scientists or large commercial enterprises; you will probably never see it.) Most people buy their 190-proof alcohol at their local liquor store; the most common brand in the United States is called Everclear.

Some states—some countries—will not allow their citizens to buy 190-proof alcohol (for their own good, of course). If you live in such a place, you will have to cross state (or country) lines and buy your alcohol from a more enlightened place or else make do with what they allow you buy. In such places, most people use a 40 to 50 percent alcohol-content vodka; that is, 80 to 100 proof. Get the highest proof you can—you will see why this is important as we go on.

In the United States, the amount you pay for liquor, regardless of what you are buying, is directly proportional to its alcohol content. The actual cost of a gallon of 190-proof alcohol is about US$1.00. The rest of the cost is federal and state taxes—which are then taxed again as sales tax when you buy the thing. So you may be tempted to buy a lower-proof vodka because it is cheaper. That is a bad idea. Your tinctures will be weak.

Fresh Plant Tinctures

Fresh plant tinctures, again, are made by putting the fresh herb in pure grain alcohol. These tinctures are nearly always made in a one-to-two ratio, which is written 1:2. (There are a few exceptions.) This ratio means you are using 1 part herb (dry weight measurement) to 2 parts liquid (liquid measurement). The amount of herb in such ratios is always indicated by the first number, the amount of liquid by the second number.

So, for example, if you have 3 ounces (dry weight measure) of fresh echinacea flower heads, you would place them in a jar with 6 ounces (liquid measure) of 190-proof alcohol. I generally use well-sealed Mason jars, stored out of the sun and shaken daily. At the end of 2 weeks the herb is decanted and squeezed through a cloth until as dry as possible (an herb or wine press is good for this), and the resulting liquid is then stored in labeled amber bottles.

Fresh plants naturally contain a certain percentage of water and alcohol is a very good extractor of water. (One of the main symptoms of a hangover comes from the alcohol extracting the water from your body—you get the same kind of headache from too much alcohol as you do from dehydration.) Alcohol will pull not only the medicinal constituents out of the plant but the plant's water as well.

The water in the fresh plant dilutes the alcohol; how much depends on the kind of plant it is. Peppermint has a lot of water in it, 50 percent or more by weight. So what you get when you tincture fresh peppermint leaves is a tincture that is about 50 percent alcohol and 50 percent water. Myrrh gum has virtually no water in it, so you end up with a tincture that is 95 percent alcohol and 5 percent water—and all that water was already in the alcohol, assuming you began with 95 percent alcohol.

Fresh leafy plants may be chopped or left whole before being placed into the alcohol or pureed with the alcohol in a blender. Fresh roots should be ground with the alcohol in a blender into a pulpy mush. (I generally think it better to make root tinctures from dry roots but there are a few exceptions; coral root is one.)

The Origin of "Proof"

As an aside: In the eighteenth century the English navy paid sailors partly in rum. The watering of drinks has always been a problem. So to test their rum before accepting it as pay, the sailors would soak gunpowder with it. If the gunpowder would still burn, the rum was "proved." Hence 100 proof. The rum had to be a minimum of 57.5 percent alcohol for the gunpowder to burn, but that has since been watered down to a simple rule of thumb, 50 percent alcohol = 100 proof.

Dried Plant Tinctures

Plants, as they dry, lose their natural moisture content. When making a tincture of a dried plant, the amount of water you add to the menstruum is the amount that was present in the plant when it was fresh. This enables the extraction of the water-soluble constituents to occur.

Dried plants are usually tinctured at a one-to-five ratio, which is written 1:5. (There are, as always, exceptions.) That means 1 part dried herb to 5 parts liquid. Fresh *Echinacea angustifolia* root, for example, contains 30 percent water by weight. If you have 10 ounces of powdered root (dry weight), you would then add to it 50 ounces of liquid (liquid measurement). This gives you your 1:5 ratio. The tricky part for many people comes in figuring out how much of that liquid should be water and how much should be alcohol. In this instance you want your liquid to be 30 percent water (fresh echinacea root's water content), that is, 30 percent of 50 ounces, which would be 15 ounces of water. The rest of the liquid will be alcohol, that is, 35 ounces.

In a formulary or materia medica, the tincture instructions for this particular plant would look something like this:

> *Echinacea angustifolia*, fresh root tincture 1:2; dried root tincture 1:5, 70% alcohol. 30–60 drops as needed. Acute conditions: 30 drops minimum each hour.

It is just *assumed* that you already know that all fresh plant tinctures at 1:2 will be using 95 percent alcohol. (Note: Everyone I know just assumes that the 95 percent alcohol they are using is 100 percent; no one I know takes that 5 percent into account in figuring this stuff out. Life is too short.)

Again, don't use tap water if you can avoid it. Powder the herbs you are tincturing as finely as possible—many people in the United States use a Vitamix for this. It is a pretty indestructible mixer/grinder, especially if you get a commercial-grade unit. (The demo video shows them grinding 2x4s into sawdust.)

Unless the herbs become tremendously hard when dried (as red root does), it is best to store herbs as whole as possible until they are needed.

This reduces the cell surface area that is exposed to air. Oxygen degrades plant matter fairly quickly.

Dried plant tinctures, like fresh, are left to macerate for 2 weeks, out of the light, before decanting.

Combination Tincture Formulas

In spite of our aversion in the United States toward the metric system, all scientific glassware in the United States is metric. Most herbalists use a *graduated cylinder* to measure the amount of tincture they are pouring out (available from any scientific glassware company). Most herbal bottles, of course, are in ounces, while the measuring cylinders are in milliliters. Roughly, 30 milliliters is equal to 1 ounce.

As an example, if you were going to make a combination tincture formula for the early onset of colds and flu, a good mix would be 10 ml each of echinacea, red root, and licorice tinctures mixed together. This would give you 1 fluid ounce total.

You can mix something like this in the graduated cylinder, as long as your hand is steady, then pour the mixture into a 1-ounce amber bottle with a dropper lid. Dosage would be one dropperful at least each hour during the onset of upper respiratory infections. This will usually prevent the onset of colds and flu if your immune system is relatively healthy.

Pressing Herbal Tinctures

When your tinctures are done and you pour off the liquid, the marc will still have some, often a great deal of, liquid in it. The marc needs to be pressed to remove the remaining tincture. Most people do this by hand. The best thing to use is a good-quality cloth with a close weave to it—I use the same surgical cloths hospitals do; they hold up really well. An herb press facilitates this immensely, though a cider press, depending on the style, will work very well, too. You will get a lot more out of a press than doing it by hand, but they do tend to be expensive.

With fresh plants you can generally get out about as much liquid as you put in; with dried material, especially roots, you get out as much as you can. Sometimes this isn't much.

How Long Will Tinctures Last?

Tinctures should be kept out of the sun—a dark, cool room is good. Keeping them in dark or amber glass jars is even better—though if they are in the dark you can leave them in clear jars as many of us do with our larger quantities of tincture. Tinctures will, in general, last many years. However, you should know about precipitation, a very neglected area of herbal medicine.

The constituents that you have extracted from the herbs are held in suspension in a liquid medium. Over time, some of these constituents will precipitate out and settle on the bottom of the tincture bottle. Some herbs such as *Echinacea angustifolia* root are heavy precipitators, while others, like elder flower, are such light precipitators that you will almost never see a precipitate in the bottle. Unfortunately, there has been little study on this, nor has a chart of herbal precipitation rates ever been prepared (as far as I know; intent searching has never turned one up). Technically, we need one that shows both rate of precipitation and the amount of precipitation for each plant.

Some herbalists will add 1 to 2 ounces of glycerine to every 16 ounces of tincture (10 to 15 percent of the total liquid) to help slow down or eliminate precipitation. It does help retard the precipitation of tannins; I am not sure how well it works for other constituents or over time but you might try it and see how it works if precipitation becomes a concern for you.

You will find that some herbs will produce an ever larger precipitate on the bottom of your storage bottles as time goes by. It is not possible to get that precipitate back into solution. Most herbalists simply shake the bottle prior to dispensing and suggest the user do the same before ingesting it. I do it this way and it seems to work fine, medicinally speaking.

There is, as yet, no data on whether the efficacy of a tincture is affected by precipitation. Certainly the ones that do not precipitate are good for decades if kept in a dark, cool location in well-sealed bottles.

Treatment of Children

Children's bodies are much smaller than adults', and if you are using herbal medicines with them, you need to adjust the dosages. You can determine the dosages for children through one of three approaches.

Clark's rule: Divide the weight in pounds by 150 to give an approximate fraction of an adult's dose. For a 75-pound child, the dose would be 75 divided by 150 or ½ the adult dose. (This is the rule I find most useful.)

Cowling's rule: The age of a child at his or her next birthday divided by 24. For a child coming 8 years of age, the dose would be 8 divided by 24 or ⅓ the adult dose.

Young's rule: The child's age divided by (12 + age of child). For a 3-year-old it would be 3 divided by (12 + 3; that is, 15) for a dose of ⅕ the adult dose.

Childhood Ear Infections

Most childhood ear infections can be treated successfully with herbs. Tinctures, glycerites, honeys, teas, and herbal steams are all effective approaches.

Children are most susceptible to ear infections from antibiotic-resistant strains of *Haemophilus influenzae, Staphylococcus aureus, Streptococcus pneumoniae,* and *Branhamella catarrhalis*. The following kinds of remedies have been found highly effective for treating them, individually or together. These kinds of ear infections often accompany flus and colds; this will help if they do.

Children's Ear Oil

INGREDIENTS:

- 5 cloves garlic
- 4 ounces olive oil
- 20 drops eucalyptus essential oil

To make:

Chop garlic finely, place in small baking dish with olive oil, cook over low heat overnight, and strain, pressing garlic cloves well. Add essential oil to garlic oil and mix well. Place in amber bottle for storage. To use: Hold glass eyedropper under hot water for 1 minute, dry well (quickly), and suction up ear oil from bottle. Place 2 drops in each ear every half hour or as often as needed for 2 to 7 days.

Brigitte Mars's Herbal Tea for Ear Infections

INGREDIENTS:

- 1 ounce elder flower (*Sambucus* spp.)
- 1 ounce licorice root
- 1 ounce Mormon tea (*Ephedra viridis*)
- 1 ounce peppermint leaf
- 1 ounce rose hips
- 1 quart water
- Wildflower honey (optional)

To make:

Roughly crush all herbs. Bring water to a near boil, then pour over the herbs and allow to steep until cooled enough to drink. Consume as hot as is comfortable for drinking. Sweeten with honey if desired. As much as is wanted can be consumed. The Mormon tea is a decongestant, the rose hips are slightly astringent and anti-inflammatory and high in vitamin C, the elder flowers are slightly sedative and reduce fevers, the licorice root is anti-inflammatory and tastes good and is antiviral and antibacterial, and the peppermint helps reduce fevers and decongests and is calming. Catnip can be added to help lower fever.

A Comment on Alcohol

There has been a tremendous resurgence of puritanitis in the United States and a few other parts of the globe (notably the UK) the past 20 years or so. One object of attention of this spasming of the puritan reflex has been the evils of alcohol. Many on the Right and on the Left seem to think it is some sort of inherently evil substance that is going to destroy Western civilization or at least make God really, really mad.

Alcohol existed long before human beings emerged out of the ecological matrix of this planet. It is a highly natural substance, both inside and outside of our bodies. *All* living beings partake of it, including trees, bees, and elephants. (Not kidding.) All of them enjoy it. It facilitates the functioning of the body, enhances organ function in many respects, and reduces the incidence of many diseases. It is not an evil substance.

One of the continual queries about tinctures concerns the alcohol content. Many people are afraid to take tinctures because of the evil alcohol in them.

Ear Infection Tincture Combination

INGREDIENTS:

- 1 ounce *Echinacea angustifolia* tincture
- 1 ounce ginger tincture
- 1 ounce licorice tincture
- 1 ounce red root tincture

To make:

Mix together the tinctures. Give one dropperful (30 drops) of the combination tincture each hour per 150 pounds of body weight until symptoms cease. Best administered in juice. Dosage should be altered for the child's weight. Eucalyptus and sage tinctures can also be used. You can also prepare this as a glycerite or a medicinal honey.

To Lower a Fever in a Child

The best herb for lowering seriously high fevers is coral root (*Corallorhiza maculata*), as either a tea or tincture. Tea: 1 teaspoon of the root steeped in 8 ounces water for 30 minutes and then drunk. Tincture: Up to 30 drops for a child of 60 pounds. Brigitte Mars's herbal tea for ear

To be really specific: The amount of alcohol in tinctures is incredibly tiny. Less than you will get from eating a few pieces of bread (yes, bread does have alcohol in it, enough to produce a breathalyzer reading of 0.05 just by itself). If you are taking 20 drops of a 60 percent alcohol tincture *every* hour for an acute condition, you will get about 1/17 of an ounce of alcohol over the course of a day (less than 2 ml). If you are taking a general dose (20 drops three times daily), you will be getting about 1/30 of an ounce over a day. Again, this is less than you will get from eating two slices of bread.

If this truly is a problem for you, you can make infusions or use glycerites—though the glycerites really aren't as effective and the water extractions won't extract some of the more important alcohol-soluble constituents. Some people heat their tinctures to remove the alcohol; it doesn't work very well and I suspect the heat alters the quality of the tincture. I don't recommend it.

infections (page 386), with the addition of catnip, is also exceptionally effective in lowering fevers. Yarrow and peppermint teas are excellent too. Finally, bathing with cool water will also work very well.

Treating Diarrhea in Children

The use of a tea and tincture combination is usually effective. See the recipes that follow.

Rosemary Gladstar's Tea for Diarrhea

INGREDIENTS:

- 3 parts blackberry root
- 2 parts slippery elm bark

To make:

Mix the herbs together (for example 3 ounces blackberry root and 2 ounces slippery elm bark). Simmer 1 teaspoon of the herb mixture in 1 cup water for 20 minutes. Strain and cool. Take 2 to 4 tablespoons every hour or as often as needed.

Tincture Combination for Diarrhea

INGREDIENTS:

- 1 ounce acacia tincture
- 1 ounce berberine plant tincture
- 1 ounce cryptolepis tincture
- 1 ounce evergreen needle tincture

To make:

Combine the tinctures, and shake well. Give 1 dropperful (30 drops) for every 150 pounds of body weight every 1 to 2 hours in water or orange juice until symptoms cease.

A Final Note

You, more than anyone else ever will, know how you are feeling in your body. Pay close attention to how you respond to any medicines you take. If you don't feel right when you take an herbal medicine, stop taking it.

NOTES

Chapter 1

1. I don't provide an in-depth look at the scientific literature on chikungunya fever in this book, but some herbs have been found effective against it (in vitro): *Trigonostemon cherrieri, Flacourtia ramontchi, Anacolosa pervilleana.*
2. Stuart Levy, *The Antibiotic Paradox* (New York: Plenum Press, 1992), 3.
3. Levy, *The Antibiotic Paradox*, 3.
4. Frank Ryan, *Virus X: Tracking the New Killer Plagues* (Boston: Little, Brown, and Company, 1997), 9.
5. Lynn Margulis and Dorion Sagan, *What Is Life?* New York: Simon and Schuster, 1995), 88.
6. Richard Lewontin, *The Triple Helix* (Cambridge, Mass.: Harvard University Press, 2000), 102–3.
7. Lynn Margulis, *Symbiotic Planet* (New York: Basic Books, 1998), 75.
8. Ryan, *Virus X*, 10.
9. Lewontin, *The Triple Helix*, 125–26.
10. Ryan, *Virus X*, 51.
11. Ryan, 52.

Chapter 2

1. In contrast, read the description of the Native American use of lomatium while treating the flu epidemic on page 310.

Chapter 6

1. If you look at the bibliography for this book you will notice that I use a large number of scientific journal papers as sources for information on the plants. I have a couple of comments about that:

   In general, I tend to give highest credence to studies performed in Asia (primarily Japan), South America (of which there are too few) and Africa (the best). For antibacterials, African researchers are doing the best work. For antivirals, researchers in China, Korea, Japan, and India are doing the best work.

   Unfortunately, U.S. researchers are too often biased against plant medicines, too influenced by pharmaceutical money, and too biased in favor of technological medicine to be completely reliable. The unreliability of Western science in this respect is a growing problem, one becoming commonly recognized throughout the world. In fact, our scientific tradition in the West risks becoming an unreliable joke throughout the world in the coming decades.

   Marcia Angell, M.D., has commented on this in a number of articles. She comments:

   > It is simply no longer possible to believe much of the clinical research that is published, or to rely on the judgment of trusted physicians or authoritative medical guidelines. I take no pleasure in this conclusion, which I reached slowly and reluctantly over my two decades as an editor of *The New England Journal of Medicine*. [M. Angell, "Drug companies and doctors: A story of corruption," *New York Review of Books*, January 15, 2009]

   Angell observes, earlier in that same article:

   > In view of this control and the conflicts of interest that permeate the enterprise, it is not surprising that industry-sponsored trials published in medical journals consistently favor sponsor's drugs—largely because negative results are not published, positive results are repeated in slightly different forms, and a positive spin is put on even negative results. A review of seventy-four clinical trials of antidepressants, for example, found that thirty-seven of thirty-eight positive studies were published. But of the thirty-six negative studies, thirty-three were either not published or published in a form that conveyed a positive outcome.

John Ioannidis, in a rather remarkable article published in *PLoS Medicine*, noted:

> There is increasing concern that in modern research, false findings may be the majority of, even the vast majority of, published research claims. However, this should not be surprising. It can be proven that most claimed research findings are false. [J. P. A. Ioannidis, "Why most published research findings are false," *PLoS Medicine* 2, no. 8 (2005): e124]

And then he goes on to do just that.

The point of all this is to say that modern scientific research, while useful, is not the last word, nor are Western studies the most reliable. If we are to create a modern healing tradition in the West, in the true sense of that word "healing," then a very different paradigm than the one in use at the moment needs to be developed. So, while I do focus in some depth on journal papers and what are normally considered to be "scientific" studies, I take them with a large grain of salt.

It takes some time to really learn to read journal papers and I don't mean from this developing an understanding of the terminology. While all the journal papers tend to follow the same structural outline and most use the same type of authorial voice, there are huge differences in the papers. With time and experience it is possible to tell which of the papers' authors know what they are doing and which do not, which are truly deep-thinking and which are barely average researchers, which of them are doing it for the money and which are genuinely interested in understanding what they are studying, which allow their humanity to guide their work and which do not. These factors alter the outcomes of the work considerably, though there has been little study of their influences. However, most people *think* that the use of journal papers confers legitimacy—and to be fair, some of them really are very good.

2. Curiously enough, many of the strongest antibacterial and antiviral plants are invasives. The dynamics of this are complex, not nearly so simple as one might think. Yes, invasives are tremendously potent simply by virtue of their capacity to take over ecosystems into which they are introduced. But this ignores the homeodynamis factors and deeply interwoven feedback systems that exist in the Earth ecosystem. Plants move throughout ecosystems in response to multiple complex factors, not simply because a seed hitchhiked on someone's shoe. Their impacts as they move are extremely complex and often highly sophisticated. And the question must always be asked, "What are they *doing* here?"

To give a very simple view outside contemporary perspectives: Amur honeysuckle (*Lonicera maackii*) is a shrub native to Japan, Korea, China, and Russia. It is an escaped cultivar in the United States and is invasive nearly everywhere it gets established. It is labeled invasive/banned in Connecticut, prohibited in Massachusettes, a noxious weed in Vermont, and invasive in Wisconsin and Tennessee. According to contemporary orientations regarding invasive species, the plant is considered to be a serious threat to ecosystem diversity and health and it is to be eradicated with extreme prejudice. However, the plant strongly affects the numbers of eggs laid by the mosquito *Aedes triseriatus*, a primary vector of La Crosse encephalitis virus (named after La Crosse, Wisconsin), reducing egg numbers considerably. The more honeysuckle there is, the fewer mosquitoes, the less the incidence of the viral disease in those areas. The plant also contains compounds that are specific for reducing inflammation, especially in the brain, during infection. They protect neurons and microglial cells from damage. This particular mosquito is also a vector for dengue fever (and other viruses), which is an emerging virus in the southern United States in such states as Texas and Georgia. Amur honeysuckle is also invasive in both those states. After 30 years of this work, I have continually seen that invasives show up in the regions where they are needed for the exact diseases that are emerging there. I can no longer discount it just because the mechanism for that process can't be seen with reductionist eyes.

3. S. H. Song and Z. Z. Wang, "Analysis of essential oils from different organs of Scutellaria baicalensis," *Zhong Yao Cai* 33, no. 8 (2010): 1265–70.

4. American Botanical Council, *The ABC Clinical Guide to Elder Berry* (Austin, Tex.: American Botanical Council, 2004), 8.

5. L. Johnson, "Elderberry for Cold and Flu Relief," *Healthy Living* (blog), CBN .com, October 13, 2011, https://www1.cbn.com/healthyliving/archive/2011/10/13/elderberry-for-cold-and-flu-relief.
6. E. Parziale, "Elderberry," part of the Backyard Herbalist site hosted at Tripod.com, https://earthnotes.tripod.com/elderberry.htm, 1999.
7. F. Vandenbussche et al., "Analysis of the in planta antiviral activity of elderberry ribosome-inactivating proteins," *European Journal of Biochemistry* 271 (2004): 1508–15.
8. Thanks, Adam.
9. S. Harada, "The broad anti-viral agent glycyrrhizin directly modulates the fluidity of plasma membrane and HIV-1 envelope," *Biochemical Journal* 392 (2005): 191–99.
10. M. Moore, *Medicinal Plants of the Pacific Northwest* (Santa Fe, N.M.: Red Crane Books, 1993), 167.
11. Moore, *Medicinal Plants of the Pacific Northwest*, 167–68.
12. P. S. Beauchamp et al., "Essential oil composition of six *Lomatium* species attractive to Indra swallowtail butterfly (*Papilio indra*): Principal component analysis against essential oil composition of *Lomatium dissectum* var. *multifidum*," *Journal of Essential Oil Research* 21 (2009): 535–42.
13. E. Krebs, *Bulletin of the Nevada State Board of Health,* no. 1 (Carson City, Nev., January 1920).

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The problem with digital books is that you can always find what you are looking for but you need to go into a bookstore to find what you weren't looking for.

—Paul Krugman

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Journal Papers and Other Publications

Comment: Many of the research papers that are published in the West, and a good number of those from the East and South, are easily found on the very useful internet database PubMed. That has made this kind of research much easier; it is fostering the wide dispersal of the scientific study of plant medicines. At the same time, there is a powerful movement among many of the world's researchers to begin publishing their studies only in open-source internet journals, which means that you can access the whole journal article, not just the abstract. A substantial number of the journals that had been prominent

in the past are now so exclusive, so expensive, that many universities are abandoning them. Some journals that originally cost $200 per year for a university subscription are now in the $20,000 range—and at the same time, the professors and researchers are still doing all the work on them without remuneration. Further, many of the formerly prominent journals are now owned by big corporations, sometimes pharmaceutical companies, and they do control what is printed. And finally, many of the non-open-source journals are now writing their abstracts in such a way as to eliminate any reasonable transfer of information. If you want *any* of the useful information from the study, you gotta pay. The normal fee range for a four-page article tends to be anywhere from $34 to $51 for 24-hour access. Some of the publishers even restrict users' ability to print the articles; they are read-only. As the Supreme Court of the United States once said (in its better days), "Well, there's no definition of piggish in the law, but we recognize it when we see it."

The open-source movement is altering things considerably, and it's about time.

As I have noted in this book and elsewhere, many of the Chinese studies have not been translated into English and are not available on PubMed. However, the Chinese National Knowledge Infrastructure (CNKI) database is developing into an Eastern form of PubMed. It is in its infancy but it is going to be a powerhouse eventually, especially when it comes to plant medicines. The Asian cultures are not caught up in the pharmaceutical-dominated prejudice of the Western medical system. They know plant medicines work; they just want to find out how to use them most effectively. So they are beginning to create a unique hybrid composed of traditional healing approaches and Western medicine, which we in the West would do well to emulate in our own fashion. (But you know, horses might make a comeback, so we keep putting our cultural money on the manufacture of buggy whips—the new ones we are making even have computer chips in them, *and* a GPS.)

Though the Chinese journal articles, for the most part, have not been translated into English, the abstracts for them *are* in English. This is opening up a tremendous amount of research that has, formerly, not been accessible. The best way to access this site, if you are interested, is to get on Google Scholar, then type in what you are looking for, like this: scutellaria cnki. Or: cordyceps cnki. This will open that world to you—it's worth it.

My bibliographical references for CNKI listings will look like this:

Wang, G., et al. Anti-tumor activity study of extract from Scutellaria barbata D. Don. *Modern Journal of Integrated Traditional Chinese and Western Medicine*, 2004-09, CNKI.

The first number after the journal title is the year of publication, and the second number is the issue. The paper above, for example, was published in issue 9 of 2004. Google's search engine does have a bit of trouble scanning the CNKI database at this point, so if you are trying to access that exact article you pretty much have to type the title in verbatim on Google Scholar with the appendage CNKI and then look over the entries that appear. It will be there somewhere. (Some of the abstract translations are challenging, so be prepared.)

Google Scholar, PubMed, and CNKI are much like bookstores—you will often find what you were not looking for. A lot of useful discoveries come from that, if you are willing to trust it.

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METRIC CONVERSION CHARTS

WEIGHT

TO CONVERT	TO	MULTIPLY
ounces	grams	ounces by 28.35
pounds	grams	pounds by 453.5
pounds	kilograms	pounds by 0.45

US	METRIC
0.035 ounce	1 gram
¼ ounce	7 grams
½ ounce	14 grams
1 ounce	28 grams
1¼ ounces	35 grams
1½ ounces	40 grams
1¾ ounces	50 grams
2½ ounces	70 grams

US	METRIC
3½ ounces	100 grams
4 ounces	113 grams
5 ounces	140 grams
8 ounces	228 grams
8¾ ounces	250 grams
10 ounces	280 grams
15 ounces	425 grams
16 ounces (1 pound)	454 grams

VOLUME

TO CONVERT	TO	MULTIPLY
teaspoons	milliliters	teaspoons by 4.93
tablespoons	milliliters	tablespoons by 14.79
fluid ounces	milliliters	fluid ounces by 29.57
cups	milliliters	cups by 236.59
cups	liters	cups by 0.24
pints	milliliters	pints by 473.18
pints	liters	pints by 0.473
quarts	milliliters	quarts by 946.36
quarts	liters	quarts by 0.946
gallons	liters	gallons by 3.785

US	METRIC
1 teaspoon	5 milliliters
1 tablespoon	15 milliliters
¼ cup	60 milliliters
½ cup	120 milliliters
1 cup	240 milliliters
1¼ cups	300 milliliters

US	METRIC
1½ cups	355 milliliters
2 cups	480 milliliters
2½ cups	600 milliliters
3 cups	710 milliliters
4 cups (1 quart)	0.95 liter
4 quarts (1 gallon)	3.8 liters

SOURCES OF SUPPLY

A weed is a plant that has mastered every survival skill except for learning how to grow in rows.

—Doug Larson

Many of the herbs I have talked about in this book—and, of course, a great many others—grow wild. Even if you live in a city you can find many of them cohabitating with you or only a short drive away. Since many of these herbs are invasives, most people will be glad for you to take them away.

If you need to buy your herbs, the internet is a good way to seek them. I suggest running a web search for the herbs you are looking for to find the cheapest prices; if you are persistent you can often save half off normal retail.

If you are going to be buying a lot of herbs and you live in the United States it makes sense to buy a resale license from your state. The price is often minimal and it will allow you to buy wholesale; most wholesalers will want a resale certificate before they will sell to you.

And, of course, you can grow them yourself. Once established most of the herbs in this book will provide medicine for you and your family forever.

Here are some of the best sources I know of for the herbs in this book. All of them are in the United States.

1st Chinese Herbs
Olympia, Washington
888-842-2049
https://1stchineseherbs.com
Wonderful people with a very large selection of Chinese herbs, including most of those discussed in this book. Most herbs by the pound.

Desert Tortoise Botanicals
Tucson, Arizona
https://www.desertortoisebotanicals.com
Very high-quality formulations and products.

Earthashram
New Paltz, New York
https://www.etsy.com/shop/Earthashram
A great Etsy herb shop with high-quality herbal extracts.

Elk Mountain Herbs
Laramie, Wyoming
307-742-0404
https://elkmountainherbs.com
Wonderful tinctures from local wild-crafted Western plants.

Green Dragon Botanicals
Brattleboro, Vermont
802-246-1090
https://www.greendragonbotanicals.com
A good source for Japanese knotweed.

Healing Spirits Herb Farm and Education Center
Avoca, New York
607-566-2701
https://www.healingspiritsherbfarm.com
Matthias and Andrea Reisen have been growing wonderful medicinal plants for years. The plants just jump out of the bag and laugh when you open it up.

Montana Farmacy
Eureka, Montana
406-297-3276
https://www.montanafarmacy.com
Very high-quality formulations and products.

Mountain Rose Herbs
Eugene, Oregon
800-879-3337
https://mountainroseherbs.com
A nice selection, sustainably produced.

Pacific Botanicals
Grants Pass, Oregon
541-479-7777
https://www.pacificbotanicals.com
This is perhaps the best wholesaler (they also sell retail) in the U.S. Their herbs are magnificent. Normally, all are sold by the pound.

Reverence Botanicals
Celo, North Carolina
https://reverencebotanicals.com
A good source of high-quality herbal extracts. They have an Etsy shop as well.

Sage Woman Herbs, Ltd.
Colorado Springs, Colorado
888-350-3911
https://www.sagewomanherbs.com
They have some otherwise hard to get items, such as isatis tincture (just the root though).

Strictly Medicinal Seeds
Williams, Oregon
541-846-6704
https://strictlymedicinalseeds.com
Richo Cech has spent much of his life learning how to grow common and rare medicinals. He has seeds or young stock for most of the plants in this book as well as great information on how to grow them.

Woodland Essence
Cold Brook, New York
315-845-1515
https://woodlandessence.com
Kate and Don make wonderful tinctures and medicines and can sell you many of the herbal tinctures that I discuss in this book; if they don't have them, they can probably point you in the right direction.

Zack Woods Herb Farm
Hyde Park,Vermont
802-888-7278
https://www.zackwoodsherbs.com
Melanie and Jeff are wonderful people and grow tremendously beautiful medicinal plants. Very, very high-quality herbs. Usually sold by the pound.

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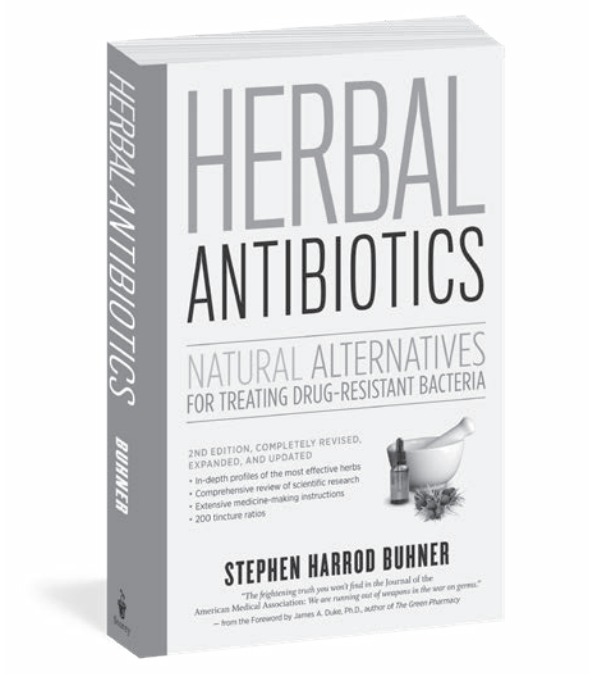
By the Same Author



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—Wise Traditions in Food, Farming, and the Healing Arts, *the quarterly magazine of the Weston A. Price Foundation*

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