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Suzanne Somers and A New Way to Age

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The Examiner of Alternative Medicine

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From the Publisher

Coping with the Covid-19 Virus

I spend most of my week living and working within miles of the epicenter of the Covid-19 virus outbreak in the US. This is in Kirkland, WA – yes, the same town where Costco was founded. Also, near the nursing home with the outbreak is the Redmond Microsoft campus. And just across Lake Washington sits Seattle's Amazon campus. While Covid-19 has not been labelled a pandemic (yet), it is now officially endemic in our community. What that means is that in this first week of March schools have been closed and white-collar workers are being ordered to work from home. (I suppose working from home with pay is a benefit). We had a day where people lined up at 8:00 in the morning to rush into Costco to purchase toilet paper – the thinking was there would be no more shipments of it from China. Rush hour traffic disappeared going in and out of Seattle on a weekday. (Of course, some parents are now being really stressed trying to get their kids to study and stay off the screen.) Meanwhile the death rate from the virus continues to eke up on a daily basis (even though it is fewer than those who die daily worldwide from snake bite). There are those

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Letter from the Publisher

➤ *continued from page 2*

clamoring to impose a more community-wide quarantine; there are those who are despairing, worrying about what they will do when they begin to get cold and flu symptoms. People want to get tested, but there are not enough test kits; people don't want to get tested so they can charade that they are not ill and don't have the virus. In Seattle we are preparing to be human guinea pigs for the Covid-19 vaccine testing when it has passed muster with animals. In the meantime, if you have a loved one in that Kirkland nursing home, don't expect to get visitation anytime soon. Local authorities are holding their breath fearing a sudden spike of critically ill patients that would overwhelm regional critical care units. Staving off that fear is going to take more than just staying home and washing one's hands.

In the first week of March the Shanghai Clinical Treatment Expert Group published a "Comprehensive Treatment and Management of Corona Virus 2019: Expert Consensus Statement." (*Chin J Infect Dis*, 2020, http://rs79.vrx.palo-alto.ca.us/opinions/ideas/pharma/ortho/08_Cov/2019-NCoV/timeline/mar/01/protocol/)

The protocol, authored by infectious disease and critical care physicians, in China offers conventional and traditional medicine treatment interventions for non-severe and critically ill patients. For antiviral treatment it is advised to use oral hydroxychloroquine or nebulized interferon gamma. High-dose intravenous ascorbic acid and heparin anti-coagulation is suggested. Fluid and electrolyte replacement are obligatory to prevent organ failure. To reduce lung interstitial inflammation, broad spectrum protease inhibitors are advised. Parenteral nutrition is recommended through nasal feeding. Oxygen by nasal catheter is needed; mechanical ventilation is obligatory with severe respiratory distress and shock. Corticosteroids should be administered cautiously; cultures should be done to determine need for co-infection treatment with antibiotics and/or antifungals. Given the breadth of therapies, monitoring for nosocomial infection and iatrogenic problems is obligatory. Traditional Chinese medicine with herbals is also advised.

When We Do Harm Prescribing Supplements

One of the truisms that we all believe is that unlike pharmaceuticals supplements pose little risk to the patient and may offer great benefit. Given that they are composed primarily of vitamins, minerals, amino acids, digestive enzymes, and botanicals, there is little likelihood of harm from their consumption. Of course, some supplements are adulterated with drugs and chemicals that do have the same risk profile as pharmaceuticals. Equally insidious are herbs

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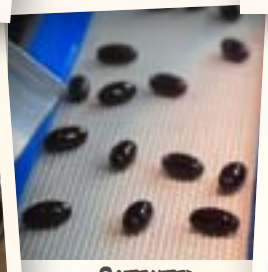
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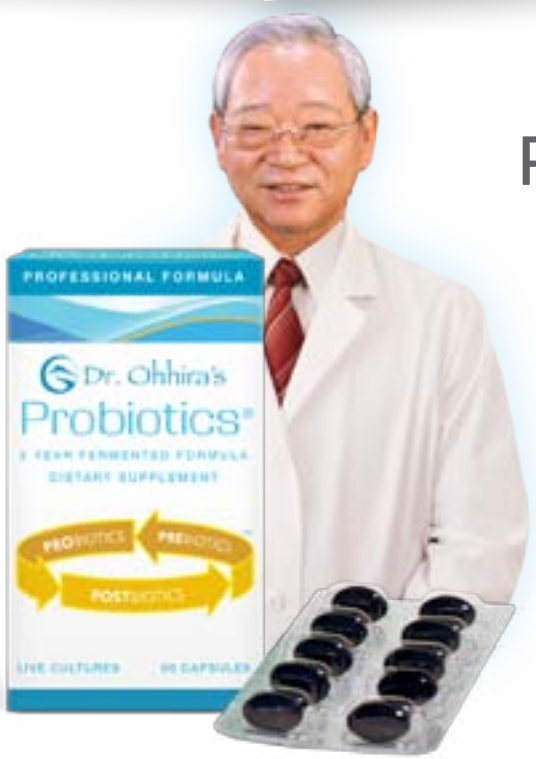

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that have been contaminated with toxic metals and harmful chemicals. A protocol of extensive supplementation, requiring the use of 10, 20, 30 or more individual supplements, increases the risk that there may be adulteration with drugs or chemicals and interaction with prescribed medications. It is very difficult to assess such risk given the proprietary nature of most supplement manufacturing and the rarity of disclosure of quality control testing. Consumers and doctors accept on blind faith that the label accurately specifies the supplement's contents and assume no adulteration. Hence, it is very difficult to determine if a patient's condition has been negatively impacted by the use of supplementation.

On a Fox television series, "The Resident," an episode featured a leading hospital physician promoting a supplement touting a unique formulation that offered major health prevention benefits. As patients began to use the supplement, there appeared a bizarre incidence of organophosphate poisoning among a few of its users. The TV drama focused on the panicked concern by the surgeon promoting the supplement and a possible connection between the supplement use and the spate of pesticide poisoning. Worried about the serious fallout that would ensue if such a relationship could be confirmed, the company executives were being asked to pull the supplement from the marketplace. At the last minute a physician asked to review the hospital cases of organophosphate poisoning and noted how individuals who had been successfully treated relapsed after putting on their street clothes immediately on being discharged. The astute resident observed that clothing purchased from a mail order house by these patients was the culprit as the garments were contaminated with the pesticide. Consequently, the poisoning was the result of direct skin contact not by consuming an adulterated food supplement.

Television drama oversimplifies medical etiologies. However, the possibility that patients may be incurring harm using supplements is an ever-present risk – one that we need to always consider as our patient's health changes.

Can We Really Survive to 125?

Our cover story in May featuring Suzanne Somers focuses on anti-aging – how one can extend one's life not just in chronological years but while manifesting vibrant health physically and mentally. Some in the life extension field have predicted that not only will this be a reasonable goal in the near future but we should anticipate life expectancies of 125 or longer. Of course, once one has begun to incur major illness and degenerative disease, such longevity begins to seem less

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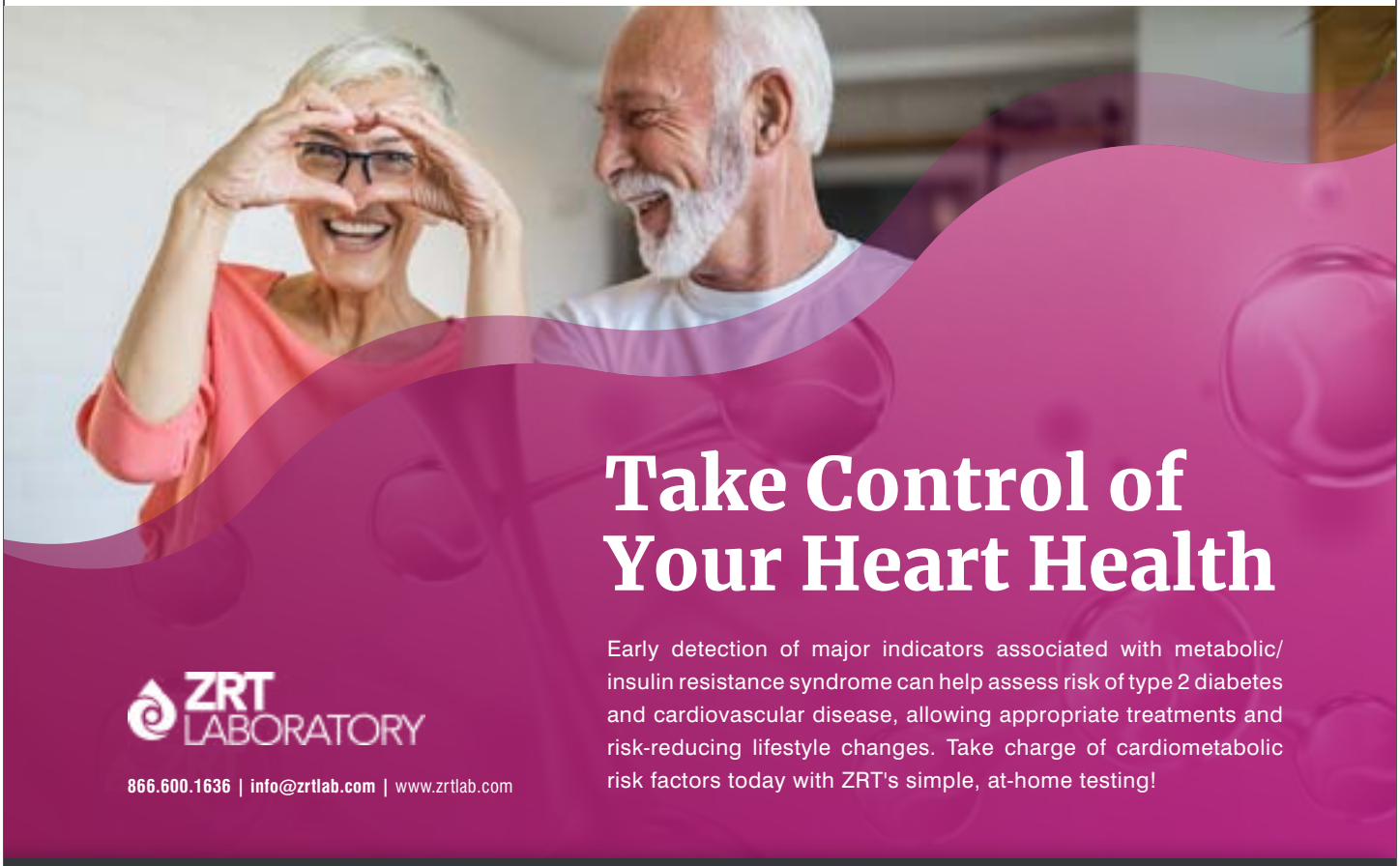

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likely if not unlikely. Nevertheless, Somers would argue having personally survived breast cancer that one can turn one's life around and embrace a life extension plan that will indeed manifest in good health and vitality. Fortunately for Somers, her cancer never progressed to metastatic disease; indeed, she would also opine that we need to incorporate a vigorous health-building plan before such serious degeneration develops. It is courageous to use one's own health as an example, a model, for others to emulate and serve as a guidepost to limitless vim and vigor. Yet, Suzanne is willing to stick her neck out and offer herself as such a paragon – and she has written 27 books over her life, interviewing doctors and researchers who confer their expertise on longevity.

Still how does one put their head around the idea of living to 125 when no one lives to that age? Well, yes, no one has lived to that age but there is a single individual, a French woman, Jeanne Calment who lived from 1875 to 1997, documented in the *Guinness Book of Records* as living to age 122. The Feb. 17/24, 2020 issue of *The New Yorker*, ran an extended article by Lauren Collins on the evidence that Calment lived as long as she claimed; the article, "Living Proof," examines reports that have appeared recently attacking the veracity of Calment's long life. It is a fascinating story well worth reading. Undoubtedly, her hormone levels were definitely higher than most women when she was age 90 – she would be able to conduct herself physically, socially, and mentally like a woman 40 years younger. Of course, Calment never used supplemental hormone therapies – she just behaved like a woman who did. Indeed, Calment did not use any life-extending treatments and did not restrict her diet nor did she avoid alcohol. But she was a "tough cookie." "She maintained a rigid schedule, rising at 6:45 a.m., said her prayers, performed calisthenics, and listened to classical music on her Walkman…She proudly told *Paris Match* that her breasts remained as firm as 'two little apples'…Behind her back, the nurses called her *la commandante*. She only quit smoking at 117, never giving up having a nightly glass of port."

What are we to make of Calment's use of tobacco and alcohol? Her physical exercise? Her toughness and demanding nature? Are these the keys to longevity?

Upcoming June issue to focus on inflammation, kidney, and liver health.

Special feature to examine the role of oral health on inflammation.

Suzanne Somers: *A New Way to Age*

For sure, Somers would not tout tobacco or alcohol. Her new book, *A New Way to Age: The Most Cutting-Edge Advances in Antiaging,* asks us to consider adapting a health plan that is more aggressive than simply eating well, exercising, using some supplements, sleeping adequately, and reducing stress. Such a prudent diet, being active, and having good sleep hygiene will certainly lead us to a good life through our 80s but not longer and will not necessarily fend off terrible degenerative conditions or cancer. The doctors Somers interviews would have us use supplemental bio-identical hormone therapy, even if we are not symptomatic, if our hormone levels are not optimal. Detoxification of toxic metals and chemicals is critical for a longevity plan – not only do we need to eliminate toxins that we consume in our beverages and food but also in our cosmetics and household cleaning agents. An active program to remove these toxins, using the Hubbard protocol with infrared sauna treatment, is necessary. Somers advocates strongly for the use of enhanced external counterpulsation (EECP) as a technique to use in patients who suffer from heart disease.

As expected Somers also advocates for the use of the "latest anti-aging" supports promoted by life-extension advocates. Bill Faloon, founder of Life Extension Foundation, champions the use of NAD+ (nicotinamide adenine dinucleotide) supplementation that can be administered intravenously, topically, and as oral supplementation; NAD+ is reported to repair DNA breaks, a common part of the aging process. Another genetic issue, chromosomal shortening referred to as telomere shortening, is also thought to contribute to the aging process; certain supplementation has been established to reverse the shortening of telomeres. No discussion of anti-aging medicine would be complete without examination of stem cell therapy. The orthopedic use of stem cell therapy in combination with platelet rich plasma (PRP) has nearly become mainstream in clinics around the US as treatment for degenerative arthritis and related conditions. Certainly, it would be reasonable to consider such treatment before agreeing to surgical replacement of joints.

Suzanne Somers provides us with her view of "a new way to age" in this issue. The book also serves as a resource for the doctors working in life extension medicine. A good read for the waiting room.

I didn't get a chance to talk about this issue's theme – cardiovascular health – and the doctors who penned their thoughts on maximizing one's heart status. Please spend some time taking a gander at Fraser Smith, ND's examination of cardiovascular inflammation, Joel Kahn, MD's approach to treating heart failure, and Jeremy Mikolai, ND's use of carnitine in circulatory disorders. Don't forget Jacob Schor, ND's curmudgeonly comments regarding eggs and cholesterol.

Jonathan Collin, MD

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Hospital-Based Intravenous Vitamin C Treatment for Coronavirus and Related Illnesses

by Andrew W. Saul and Atsuo Yanagisawa, MD, PhD

Orthomolecular Medicine News Service

No matter which hospital a coronavirus patient may seek help from, the question is, will they be able to leave walking out the front door, or end up being wheeled out the basement backdoor? Prompt administration of intravenous vitamin C, in high doses, can make the difference.

Abundant clinical evidence confirms vitamin C's effectiveness when used in sufficient quantity.[1] Physicians have demonstrated the powerful antiviral action of vitamin C for decades.[2]

Specific Instructions for Intravenous Vitamin C

The Japanese College of Intravenous Therapy (JCIT) recommends intravenous vitamin C (IVC) 12.5/25g (12,500 - 25,000 mg) for acute viral infections (influenza, herpes zoster, common cold, rubella, mumps, etc.) and virus mimetic infections (idiopathic sudden hearing loss, Bell's palsy). In adults, IVC 12.5 g is given for early stage illness with mild symptoms, and IVC 25 g for moderate to severe symptoms. IVC is usually administered once or twice a day for two-to-five continuous days, along with or without general treatments for viral infections.

IVC 12.5 g cocktail	
Sterile water	125 mL
50% Vitamin C	25 mL (12. 5g)
0.5M Magnesium sulfate	10 mL
Add Vitamin B complex	
Drip for 30-40 min	

IVC 25 g cocktail	
Sterile water	250 mL
50% Vitamin C	50 mL (25g)
0.5M Magnesium sulfate	20 mL
Add Vitamin B complex	
Drip for 40-60 min	

Patients with acute viral infections show a depletion of vitamin C and increasing free radicals and cellular dysfunction. Such patients should be treated with vitamin C, oral or IV, for neutralizing free radicals throughout the body and inside cells, maintaining physiological functions, and enhancing natural healing. If patients progress to sepsis, vitamin C should be added intravenously ***as soon as possible*** along with conventional therapy for sepsis.

> Fred Hui, MD believes that administering vitamin C intravenously is a treatment worth trying. And he'd like to see people admitted to hospital for the pneumonia-like virus treated with the vitamin intravenously while also receiving the usual drugs for SARS. "I appeal to hospitals to try this for people who already have SARS," says Hui. Members of the public would also do well to build up their levels of vitamin C, he says, adding that there is nothing to lose in trying it. "This is one of the most harmless substances there is," Hui states. "There used to be concern about kidney stones, but that was theoretical. It was never borne out in an actual case." Hui says he has found intravenous vitamin C effective in his medical practice with patients who have viral illnesses.[3]

Additional administration details are readily obtained from a free download of the complete Riordan Clinic Intravenous Vitamin C Protocol.[4] Although initially prepared for cancer patients, the protocol has found widespread application for many other diseases, particularly viral illnesses.

> Research and experience has shown that a therapeutic goal of reaching a peak-plasma concentration of ~20 mM (350-400 mg/dL) is most efficacious. (No increased toxicity for posoxidant [sic] IVC plasma vitamin C levels up to 780 mg/dL has been observed.) . . . [T]he administering physician begins with a series of three consecutive IVC infusions at the 15, 25, and 50 gram dosages followed by post IVC plasma vitamin C levels in order to determine the oxidative burden for that patient so that subsequent IVCs can be optimally dosed.
>
> Pages 16-18 of the Riordan protocol present IVC administration instructions.

There are four pages of supporting references.

Winning the Hospital Game

When faced with hospitalization, the most powerful person in the entire hospital system is the patient. However, in most cases, the system works on the assumption that the patient will not claim that power. If on your way in you signed the hospital's legal consent form, you can unsign it. You can revoke your permission. Just because somebody has permission to do one thing doesn't mean that they have the permission to do everything. There's no such thing as a situation that you

cannot reverse. You can change your mind about your own personal healthcare. It concerns your very life. ***The rights of the patient override the rules of any institution.***

If the patient doesn't know that, or if they're not conscious, or if they just don't have the moxie to do it, the next most powerful person is the spouse. The spouse has enormous influence and can do almost as much as the patient. If the patient is incapacitated, the spouse can, and must, do all the more. If there is no spouse present, the next most powerful people in the system are the children of the patient.

When you go to the hospital, bring along a big red pen, and cross out anything that you don't like in the hospital's permission form. And before you sign it, add anything you want. Write out "I want intravenous vitamin C, 25 grams per day, until I state otherwise." And should they say, "We're not going to admit you," you reply, "Please put it in writing that you refuse to admit me." What do you think their lawyers are going to do with ***that***? They have to admit you. It's a game, and you can win it. But you can't win it if you don't know the rules. And basically, they don't tell you the rules.

This is deadly serious. ***Medical mistakes are now the third leading cause of death in the US.*** Yes, medical errors kill over 400,000 Americans every year. That's 1,100 each day, every day.[5]

There are mistakes of commission and mistakes of omission. Failure to provide intravenous vitamin C is, literally, a grave omission. ***Do not allow yourself or your loved ones to be deprived of a simple, easy to prepare and administer IV of vitamin C.***

It Can Be Done

Vitamin IVs can be arranged in virtually any hospital, anywhere in the world. Attorney and cardiologist Thomas E. Levy's very relevant presentation is free access.[6,7]

Both the letter and the intent of new USA legislation now make this easier for you.

> The new federal Right to Try Act provides patients suffering from life-threatening diseases or conditions the right to use investigational drugs... It amends the Food, Drug, and Cosmetic Act to exempt investigational drugs provided to patients who have exhausted approved treatment options and are unable to participate in a clinical trial involving the drug. Advocates of right to try laws have sought to accelerate access to new drugs for terminally ill patients who are running out of options. Arguably, the law does not represent a radical change in this and several other states, however, because in 2016, California had already joined the majority of other states in adopting a law enabling physicians to help terminally ill patients pursue investigational therapies, without fear of Medical Board or state civil or criminal liability. . . The new Right to Try law should give physicians, as well as drug manufacturers, some added comfort about FDA enforcement in these cases.[8]

"Given the rapid rate of success of intravenous vitamin C in viral diseases, I strongly believe it would be my first recommendation in the management of corona virus infections."

Victor A. Marcial-Vega, MD – Puerto Rico

"It is of great importance for all doctors to be informed about intravenous vitamin C. When a patient is already in hospital severely ill, this would be the best solution to help save her or his life."

Karin Munsterhjelm, MD – Finland

"If a family member of mine died due to coronavirus infection, after a doctor refused to use intravenous vitamin C, I would challenge his or her treatment in a court of law. I would win."

Kenneth Walker, MD, surgeon

Therefore, in regards to intravenous vitamin C, do not accept stories that "the hospital can't" or "the doctor can't" or that "the state won't allow it." If you hear any of this malarkey, please send the *Orthomolecular Medicine News Service* the text of the policy or the law that says so. In the meantime, take the reins and get vitamin C in the veins.

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Rebecca Harder is the author of "Gastra Girl: Saving America One Colon at a Time," and owner of an immaculate and highly esteemed clinic in Portland, OR. She offers this well-researched comprehensive resource guide of holistic health information on topics such as environmental toxicity, vaccines, EMF, autism, hyperbaric oxygen, ozone therapy, colon hydrotherapy, far infrared saunas and much more.

Rebecca had come across the Relax Sauna at professional conferences many times before she decided to finally try it. She had been committed to wooden infrared saunas for 10 years at her respected clinic. Immediately after trying the Relax Sauna, she experienced instant dramatic positive healing results. She was so impressed with it that she dedicates an entire 8 pages to the Relax Sauna in the chapter "Why infrared Sauna is an absolute necessity for Everyone." She enthusiastically recommends the Relax Sauna to her clients and lets them know that it is the best way to rid the body of toxins and feel good.

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Green Heart Louisville

by Jacob Schor, ND

Here is some good news. If you consult the website ClinicalTrials.org under the designation NCT03670524 you will find a fascinating trial described

The study known as Health, Environment and Action in Louisville, or by its acronym HEAL Louisville is most often referred to as Green Heart Louisville. The study is sponsored by the University of Louisville, National Institute of Environmental Health Sciences (NIEHS), and The Nature Conservancy. The official description goes like this:

> The purpose of this study is to examine how the environment and neighborhood characteristics affects the health of the area residents. The study will help determine how changing neighborhood characteristics, such as green space, affect heart health, risk factors for other diseases, sense of well-being or neighborhood cohesion.

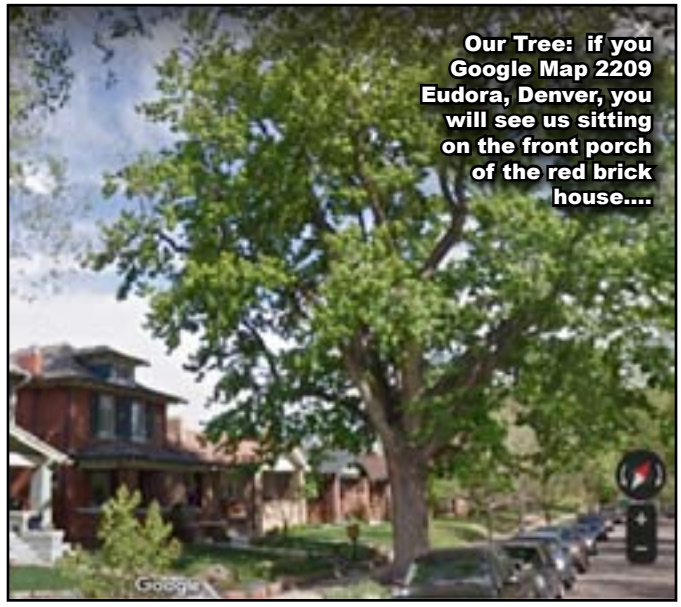
Put into more straightforward words, the researchers are going to 'green' several neighborhoods by planting trees in Louisville and see what it does. Not a few trees, but the goal is a lot of trees, 8,000 of them, and not necessarily small trees either. News stories talk about 50-foot tall trees. Researchers plan on tracking a long list of health parameters and psychological measures in 700 people to see what effect this intervention will have. The Nature Conservancy is teaming up with the University of Louisville to make all of this happen.

The primary hypothesis underlying the study is that exposure to neighborhood green space diminishes cardiovascular disease risk by decreasing the levels of ambient air pollution. Heart disease costs money, and planting trees may save money.

There is a list of secondary outcomes the researchers are hoping to see that includes a decrease in hospital admissions, lowering measures of psychosocial stress, decreased risk of mortality, buffered noise, decrease in crime, increased physical activity in residents and increased property values. We have good reason to hope for these kinds of changes based on results from already published research. These earlier studies have shown statistical associations between green space and all of these plus other positive population attributes.

Association does not prove causation, as we all know, and there may be other plausible explanations for why we see green space appear so beneficial. The easier may simply be economics. Maintaining a green neighborhood costs money. Increased income is always associated with better health. This is not just because rich people see more doctors or eat better food. There is a psychological benefit to having money and status that improves health. People who win big in life live longer. And trees cost money. We have a 100-year-old maple in front of our Denver home, and paying the arborists who care for it every year or so is not inexpensive.

This Louisville Green Heart study appears to be the first prospective trial of this sort, certainly on this scale, that tests our current belief that green space will improve community health.



There won't be a control group in Louisville. You cannot plant a placebo tree.

If the hypothesis proves valid the results of this study will argue that taking care of our urban environments and maintaining an adequate green canopy pays for itself. This could be exciting.

And yet part of me feels pinched by this equation. It feels so, what is that word that has gotten popular? Transactional. We do this and we want this outcome in trade. Somehow this deal doesn't capture just why trees are so important.

Deep down I suspect that all life is sentient to some extent. Certainly, trees appear to be. I also think that in some way that we are aware of this sentience, that it creates a background of thought that enriches our experience of consciousness. Kind of like the soundtrack to a movie or the laugh-track to a sitcom. We may not be focused on what trees are thinking, but they provide a backdrop to life. We can live without it, but we are poorer for the experience. Being surrounded by nature enriches our life in ways that we need not and should not put dollar signs on but should nevertheless desire.

Leaving these ruminations aside though, this Green Heart Study remains the most upbeat news I have heard in a long time. Listen to the story links below and I bet you'll be hunting for your work gloves and shovel and wanting to volunteer to help plant trees in Kentucky.

Listen to the full story of the Green Heart Study: https://www.nature.org/en-us/about-us/where-we-work/united-states/kentucky/stories-in-kentucky/green-heart-project/

Watch a short feature from PBS: https://www.youtube.com/watch?v=e0QLQe13gww

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FCT and the Corona Virus Pandemic: Who Is Responsible for the Needless Bloodletting?

by Savely Yurkovsky, MD©

As the world suffers from the deaths, sickness, and tremendous economic loss that stem from the coronavirus in China, the actual tragedy originated in the United States in 1910. It is the year when the infamous Flexner Report, influenced by the American Medical Association and paving the way to a monopoly of pharmaceutical medicine, did away with most of the alternative medicine and particularly, homeopathy, considered an overdiluted water, void of substance. This report established a biomedical model (scientific for "drug medicine") as premier science, which also became a guide for WHO to establish "scientific" standards for the education of medical doctors worldwide. No wonder that China too, which due to its overcrowdedness of people and livestock has been the source of several recent deadly viral epidemics, considers homeopathy as impermissible quackery. But the paradox of this is—whether in China, in America, or anywhere else in the world—homeopathy is the only medical system that can halt any epidemic, especially one that cannot be contained by vaccines due to their absence, shortage, ineffectiveness, or side effects.

Under the circumstances, homeopathy can use several effective approaches. Among these are *epidemic simillimum* or a remedy that produces similar symptoms in healthy volunteers, *isode*, a remedy prepared from a microbial culture or sample from the contaminated environment or infected bodily fluids, *autoisode*, a remedy prepared from a patient's own infected bodily fluids, and a *simillimum*, a remedy that is based on an individual assessment of a patient. Over centuries, homeopathy used any of these methods to successfully abolish or contain even far deadlier infections than the coronavirus, such as smallpox, malaria, typhus, meningococcal, leptospirosis and others. Besides these methods, a combination of viral isodes to elicit a non-specific, heightened immune response during annual flu seasons may also successfully be used, as experience has shown. In addition to the homeopathic methods described here, my book presents in detail how people themselves can prepare homeopathic isodes and autoisodes in minutes.[1] (What a $ nightmare for scientific medicine!) I also present important measures for the public to protect their immune system from the draining impacts of bad diet and electromagnetic radiation. As ample experience with antibiotics and other conventional and alternative treatments has confirmed, magic wands do not exist in medicine.

Chest X-ray Report for Man with Severe Pneumonia

Final

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763-389-1313

Adm:
Pt Class: Outpatient
Dsch:
Phone: 763-389-0216 Sex: Male

IMAGING REPORT

Exam: XR Chest 2 Views

Date/Time Completed: 03/06/2020 1052
Authorizing Provider: Yurkovsky, Savely
Ordering Provider: Yurkovsky, Savely
Attending Provider:
CC Provider:

CHEST TWO VIEWS 3/6/2020 10:52 AM

HISTORY: Follow-up evaluation of pneumonia.

COMPARISON: 7/31/2019

IMPRESSION: No evidence for acute focal alveolar infiltrate or consolidation that would represent pneumonia. No pleural fluid or pneumothorax. Mild hyperinflation which can be seen with COPD in the proper clinical setting. Normal heart size. Normal pulmonary vascularity. Bilateral calcified granulomas. Minimal degenerative changes in the spine.

Here are just a few out of countless examples from my practice in treating infectious diseases.

A Dying Young Man

An alarmed mother, a registered nurse, contacted me for emergency treatment for her 20-year-old son, hospitalized in the intensive care unit and placed on a respirator due to ARDS (Adult Respiratory Distress Syndrome). Its estimated mortality rate is 30-40%. It usually develops rapidly due to some respiratory infection, and in his case started with an ordinary cold. Despite receiving many drugs in the ICU, his lungs remained drowned in fluid. Following my assessment, high potencies of viral and bacterial digital isodes (US patent pending, #16/544,644), prepared from lower potencies of homeopathic isodes, were shipped overnight. Within 48 hours, his mom reported a dramatic turnaround that culminated in his walking out of the hospital a few days later.

A Man with the Face of an Open Watermelon

Following an insect bite, a man's face became red, itchy, blotchy and doubled in size. An emergency room visit and prescribed drugs offered no relief. Following a digital autoisode made from his blood, both the shape and health of his face were promptly restored.

Dental Cleaning or Contamination?

For months, following a dental cleaning, this woman's gums and teeth became painful, particularly when chewing. Bioresonance testing indicated an unknown bacterial infection in gums and teeth. To avoid the side effects of antibiotics, an autoisode prepared from her saliva ended the ordeal.

Severe Pneumonia Resolved Without a Single Antibiotic

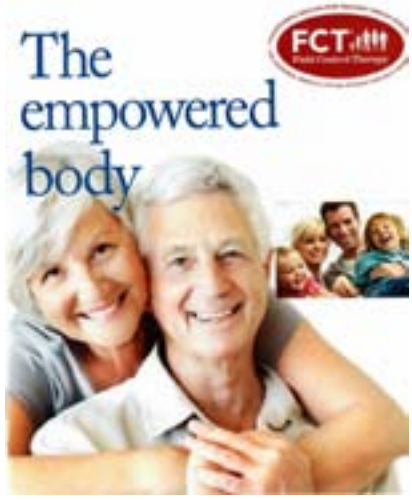
A middle-aged man developed classical signs of pneumonia with a fever of 104° and severe coughing with yellow-green mucus. The chest x-ray confirmed it; but with him having a history of sustaining severe side effects of antibiotics on popular Lyme treatments and getting nowhere, he refused antibiotic treatment and opted for FCT. Even with him continuing to smoke through the pneumonia ordeal, in spite of my objections, and working and living in a heavily contaminated mold, dust, and chemically polluted environment, which had to be addressed with the corresponding isodes, the copy of his chest x-ray report (page 14) speaks for itself.

There was another story that this patient also shared, concerning his friend who developed exactly the same symptoms and pneumonia. The friend was hospitalized for weeks only to be treated with intravenous antibiotics and then orally. Yet he never recovered, even months later, and only made "day and night" progress after my patient, taking the initiative, gave him my remedies.

Concluding the issue of doing away with "unscientific homeopathy" because the remedies are "overdiluted water placebos," materials scientists, who have the ultimate say in determining the nature of all substances, call this "ignorance in and distortion of science."[2] Other reputable scientists from around the world, including Nobel laureates in medicine and physics, stated the same. Besides hundreds of thousands of needless deaths from SARS, H1N1, Ebola, coronavirus and just ordinary flu viruses, tens of thousands

continued on page 18 ➤

An FCT Practitioner, A Biological Dentist, Ann Pearcy DDS:
"FCT is a revelation and a revolution in healing."



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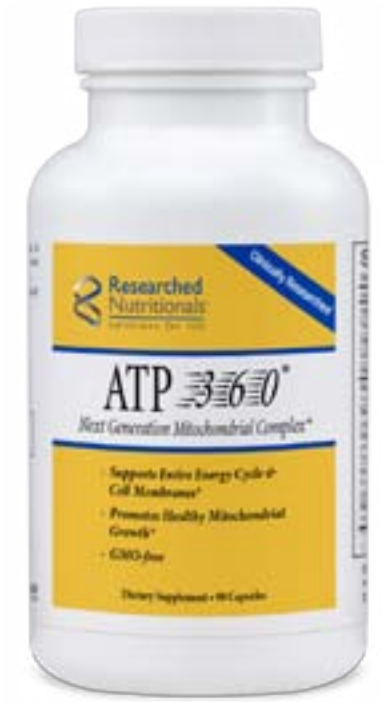

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In this Issue

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FCT and the Corona Virus Pandemic

➤*continued from page 15*

of needless deaths are added annually in the US alone due to the exploding antibiotic-resistant bacterial epidemics. All of these are on the watch of "scientific medicine." Tragically, more epidemics and deaths will come as it is likely that billions of pigs and chickens (not wearing face masks) in China and other overcrowded environments or drastic climate changes will continue to lead to new aggressive microbes that will strike us. In light of these catastrophic unresolved problems, the new trigger that the FDA is about to pull against homeopathy and all of us could outlaw homeopathic isodes. Looks like another Flexner Report massacre!

Even if it is carried out in good faith, "to protect the public against contaminated solutions," how in the world can these solutions be "overdiluted placebos with nothing left in them," in the first place and yet be contaminated by the homeopathic manufacturers at the same time?! Tragically, this is not funny as it is very serious.

Savely Yurkovsky MD, Board-certified in internal medicine and Board-eligible in cardiovascular medicine, undertook a particular interest in mercury toxicity as both its victim, and a clinician managing a busy private practice. Shortly after moving to the US from the former Soviet Union, he received several silver amalgam fillings, which he recognized later as the cause of his mounting health problems. These problems persisted despite removal of the fillings that prompted him to explore various mercury detoxifying approaches: oral, intravenous, homeopathic. After observing their corresponding partial benefits, limitations and aggravations, on himself and his patients, he resorted to bioresonance testing and causative homeopathy, based on relevant knowledge from physics and toxicology to optimize benefits and safety of the detoxification. The guidance of his physics' consultant, Stanford University's materials' science Professor William A. Tiller, PhD was instrumental in enhancing diagnostic ability of bioresonance testing to address the known limitations of lab tests to detect the presence of toxicants in the internal organs. This testing also was used to draw a better comparative capacity between various mercury detoxifying treatments as well as to evolve a safer therapeutic strategy leading to minimize the reintoxication or dumping effect which are common to these treatments. It also optimized an unlimited therapeutic potential of homeopathy that has a unique capacity to therapeutically connect with any organ and tissue, via specific signals, as no other treatment can.

His book, *Biological, Chemical, and Nuclear Warfare – Protecting Yourself and Your Loved Ones: The Power of Digital Medicine*, has been endorsed by Professor Emeritus William A. Tiller, PhD of Stanford University and IT Physics Professor George Pugh PhD. He presented this system at the Combating Bioterrorism Conference in 2005, sponsored by the Office of Homeland Security.

Dr. Yurkovsky founded a teaching organization, "*SYY Integrated Health Systems, Ltd.*," in 1999 which is dedicated to training health practitioners in this biophysical system under the concept of FCT – Field Control Therapy®. He has lectured extensively in the US and Europe.

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Injection Therapies – Helping Chronically Afflicted Patients Overcome Hard-to-Treat Conditions

by Marc Harris, ND

In 1990, JG came into my office in a wheelchair. His history was from a traumatic accident 10 years earlier. He had been on fentanyl for four years until he could no longer renew the prescription. He had 83 surgeries in the year following the accident.

His chief concern was suicidal pain. He reported trying every aspect of care he could find (prescriptions, TENS, acupuncture, chiropractic, massage, Rolfing etc.) He was unable to get any relief. After reading a study that came out in 1989,[1] I suspected his pain was coming from sensory nerves. (See Figure 1.) Sensory nerves are the cutaneous nerves that provide feelings of heat, cold, vibration, pruritis, and pain.

Medical students and surgeons are typically not educated on cutaneous nerves, and they cannot be seen in anatomy class. After seeing the pictures in the Kruger[1] study and researching the nerves, I suspected the pain was coming from the scar tissue transection of the cutaneous nerves.

A previous arcane degree became fortuitous. I started dextrotherapy, where dextrose, with a couple of possible additions, are injected to the areas of cutaneous nerve pain. (See Figure 2.) The cutaneous nerves can give radiating pain and regional pain patterns I consider mistakenly diagnosed as tendon or trigger points. This can be said confidently as they respond to the injections with long-term (i.e. permanent) relief. Additional studies have looked at dextrose for pain relief.[2,3,4]

Some (many) pain syndromes have been misdiagnosed and are really trapped cutaneous nerves. They typically have the diagnostic labels such as ileo tibial band syndrome, plantar fasciitis, many (if not most) migraines, tension headaches, back pain, baker's cyst, complex regional pain syndrome, fibromyalgia and many other pain syndromes.

KF is a 10-year-old who came into my office with wrist pain. It had been ongoing for five months. Upon examination there was no displacement or palpatory pain of carpal or metacarpal bones. Active, passive and resisted motion did not produce pain. Test for carpal tunnel syndrome was negative. Upon palpation of the supine forearm, the pain was reproduced. Two injections were done, and she reported "warmth" moving into her wrist and was pain free. A second treatment was necessary. She has been pain free for 12 months at the time of this writing.

Let us briefly pause to say that not all pain originates from the cutaneous nerves. Degenerative joint disease and similar pathologies do not originate from the cutaneous nerves. These conditions respond very well to progenitor cell injections (those cells that prompt the body to activate and reproduce stem cells).

Progenitor cell therapy (it goes by different names) is effective for joint pathology and systemic pathology. It is often effective for those conditions we consider incurable: Diabetes (Type I),[5-7] Parkinson's,[8,9] and Alzheimer's disease,[10-13] as well as many other neurologic,[14-16] cardiovascular,[17-19] and other conditions.

LR is an 86-year-old man who came in with right knee pain. X-ray showed bone on bone. He was on coumadin and told he was not a candidate for knee replacement due to previous clotting issues. For LR we combined prolozone, platelet-rich plasma (PRP), and progenitor cells into his right knee. This was a single therapy. He had been pain free for two years and wanted an x-ray to compare it to his previous imaging. The second X-ray showed full cartilage spacing.

As we consider additional injection therapies, we next look to Frank Shallenberger, MD, who combined prolotherapy and ozone therapy to create prolozone. Prolozone is used in joints and via different methods used systemically.[20-23]

As our list of injection therapies grows, we have to next ask what the proper therapy for each individual is. This gets the most unsatisfying of answers in that it varies from person to person. For most of the incurable conditions mentioned so far, progenitor therapy is the best of all the methods described herein. Then we also see that PRP helps to increase progenitor/stem cells.[24]

The uses of prolozone and ozone may be unlimited.[25-29] Prolozone/ozone is used in dentistry for sterilization and osteoblastic activity. If ozone increases osteoblastic activity in the teeth, would it do so for bones and joints? Thirty years of use in my practice would seem to indicate it would and it does.

Ozone/Prolozone is also used in veterinary medicine in such widespread conditions as infertility (in cows) disc herniations (in dogs) and endometriosis (in cows). Remember this is veterinary medicine. While the studies have not been done, we can wonder about systemic ozone use for human endometriosis and infertility. It is also used in cows for mastitis (ozonated water). Mastitis generally is of bacterial origin: Studies show the capacity of ozone to neutralize infections agents[30-32] and toxins as it is also used in water sterilization.[33] We now have insights (systemic ozone) into some of most difficult to treat infections (*Borrelia*, *Babesia* and *Bartonella*[34]).

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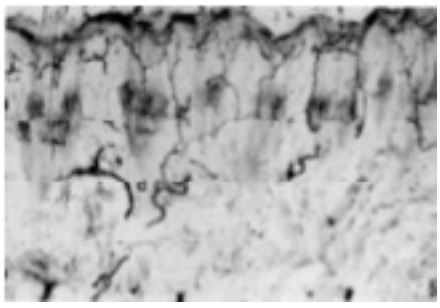

***Figure 1. CGRP stained cutaneous nerves of epidermis.**

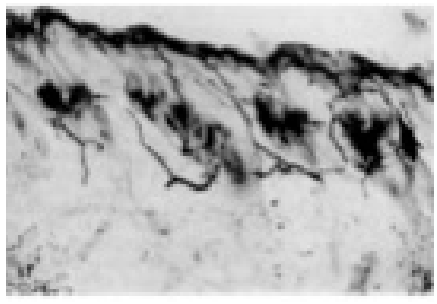

***Figure 2. Branched patterns of stained cutaneous nerves of epidermis.**

➤

Ozone/Prolozone seems to be beneficial for most conditions and has a wide variety of infiltration methods.

Returning to patient LR, (the 86-year-old with bone on bone osteoarthritis)…. After his initial treatment, we had a follow-up visit two months later. He told me his knee was doing great and, by the way, his diabetes was resolved. I said I didn't know you had diabetes. He said, "Oh yes, I have been a type I diabetic for 60 years."

In describing progenitor therapy above, it was mentioned the body's own stem cells can be increased by PRP.[7] In this study patients in for hip replacement had bone marrow removed to produce stem cells. The stem cells were inoculated with PRP (platelet-rich plasma) or FCS (fetal calf serum). The authors expressed concerns over the possibility of the fetal calf serum as a vector for prions. Their concerns were allayed when the experiment concluded, as the PRP-inoculated stem cells produce twice as many stem cells as the FCS cells. There was no need to have the theoretical prion fear as the PRP was far superior. (80 x 10^6 stem cells for the PRP treated group, compared to 40 x 10^6 stem cells for the FCS treated group; see Table 1). While this was an *in vitro* study, additional studies have been done. The pathways of PRP seem to include growth factors and stem cell stimulation.[35-37]

We will round up our discussion of the most effective injection therapies with neural therapy. Neural therapy was devised by two German medical doctor-brothers, Walter and Ferdinand Huneke, in 1925. This is often the most effective injection therapy for scar tissue, ligaments, and tendons.

AE was having migraine headaches that came on after appendix surgery. Neural therapy injections were injected into the scar tissue, and his migraines have resolved for the last 13 years and counting. We see similar results in rotator cuff injuries ligament laxity and conditions of a dysregulated autonomic nervous system.[38-43]

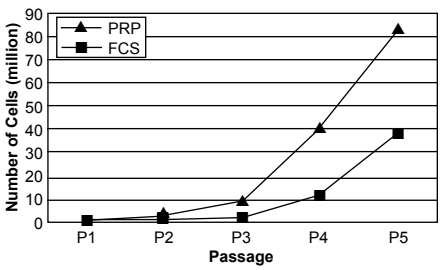

Table 1. stem cell counts of 80 x 10^6 with PRP vs 40 x 10^6 with FCS**

Now, having looked at five types of injection therapies we begin to see the potential cures of conditions that plague a high percentage of chronic patients: pain, degenerative joints, disc herniations, chronic infections, rotator cuff injuries, and the list of pathologies and cases presented here are just an introduction to the myriad healing possibilities of injection therapies.

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Non-Invasive Neural Therapy with Microcurrent Point Stimulation

by Kelly Armstrong, OTR/L, PhD

Introduction

Science now recognizes that the primary problem experienced post-surgery is chronic pain. Called chronic post surgical pain (CPSP) or persistent postoperative pain (PPP), it is defined as pain lasting more than three to six months post surgery. Up to one-third of patients undergoing common surgical procedures report persistent or intermittent pain of varying severity at one year postoperatively.[1]

For most physicians and patients, the postoperative management of scars or scar-related pain continues to lag behind decades-old research. There is a wide variety of ineffective commercial products, with few viable clinical options available currently for physicians to offer patients relief.[2]

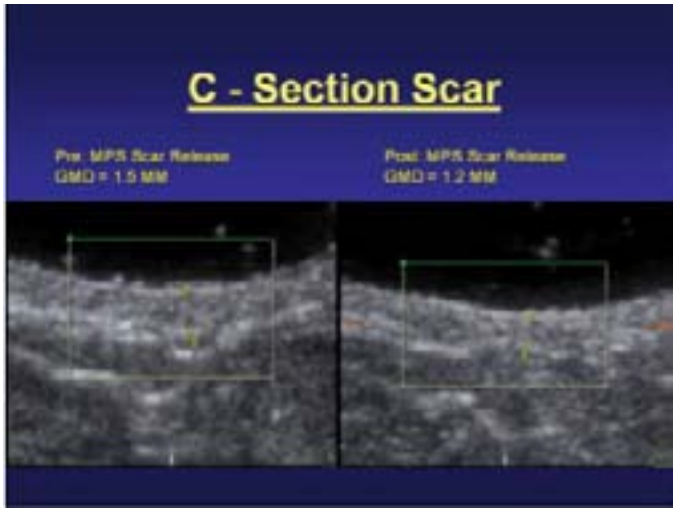

Figure 1. 3D 17 MHz volumetric linear probe of Grey Mean Depth Pre-post MPS-SRT Therapy

One option that has proven effective is neural therapy (NT). NT is an invasive therapy that recognizes surgical scars as systemic agonists for stress and chronic pain(s). Neural therapy is based on the premise that the injection of negatively poled local anesthetics, usually procaine or lidocaine, into positively poled scar tissue restores the function of damaged nerve cells and regulates the autonomic nervous system (ANS).[3]

The outcomes from neural therapy are reported to be positive with few negative side-effects. However, NT is a learned art that requires specialized training and direct physician intervention or oversight to apply. Is there another way to deactivate scars and reduce their negative influence on human health without being invasive?

Scar Reduction Therapy: Non-Invasive Neural Therapy

Scar release therapy (SRT) is an innovative breakthrough in reducing scar size, patient pain levels, and improving functional outcomes. SRT uses the latest advances in science to address both chronic pain and dermal scarring after any trauma or surgery. SRT applies concentrated microcurrent point stimulation (MPS), a patented solution that is based on the scientific concept of increasing skin's inter-cellular metabolism, protein synthesis, and healing function to re-awaken the skin's ability to self generate.

Applying concentrated microcurrent (MPS) bi-laterally to scars non-invasively "injects" electrically repolarising current deep into fibrous scar tissue. Newly "repolarized" scar is noticeably softer, with increased pliability and diminished depth and physical appearance. The results of SRT are scientifically measurable.

Table 1. Pre-and Post-Treatment Descriptives and Results for MPS-SRT Stimulation Applied to Scars as measured by 3D 17 MHz volumetric linear probe: Grey Mean Depth (MG) (N=17)

	N	Mean	Minimum	Maximum	Std. Deviation	Percentage% Improvement	Confidence Interval (>=95% CI)
Pre-MPS-SRT Stimulation	17	1.8764mm	1mm	4.2mm	0.8584		
Post-MPS-SRT Stimulation	17	1.4294mm	0.8mm	3.2mm	0.6410	-23.8244%	p<0.001

SRT Reduces Scar Size

In clinical trials performed at the Bard Diagnostic Center in New York City, a scar patient sample (n=17) was provided SRT. The results were measured with 3D 17 MHz volumetric linear probe: Grey Mean Depth (MG). The findings revealed there was statistical evidence for significant reduction of 0.447mm or 23.8244% reduction in post-treatment scar depth, when compared to pre-treatment mean scar depth. (See Table 1 and Figure 1)

The changes recorded in this clinical trial validate the potential application of MPS to fibrous tissue as a viable option for both clinicians and patients for the post-operative management of scar size. It was reported "the procedure provided significant outcomes after a single application and has the unique ability to be personally adapted to the individual's wound characteristics." Although this trial is demonstrative that SRT physically reduces scar depth, can it extrapolate into improved patient pain relief?

SRT for Pain Management

To determine the efficacy of SRT on CPSP pain, a study was undertaken on a sample of patients with history of non-specific chronic pain. SRT was applied to (N=51) patients, and VAS pain scales were recorded post application and 48-hour follow-up after microcurrent point stimulation was applied to scars.

The immediate post-application VAS response of MPS applied to a N=51 patient scar sample with chronic pain reflected a statistically significant reduction of 3.706 points or 59% reduction in mean pain levels when compared to initial pain levels [95% CI (3.033, 4.379; p=0.000159%]. When VAS pain levels were measured at the 48-hour follow-up, there was an additional statistically significant reduction of 0.902 points or 34% reduction in mean pain levels post treatment [95% CI (0.406, 1.398; p=0.001], for a total statistically significant reduction of 4.608 points or 73% reduction in mean pain levels [95% CI (3.940, 5.275); p=0.0001].

The data from this study[4] illustrates the application of MPS to physical scars had a marked improvement in pain outcomes, and the outcomes were even more impressive given the patient sample for pain duration (mean 7.61 years) and the intensity (mean 6.33/10) that improved after a single MPS scar release application. (See Table 2)

Why Microcurrent for Scar Release?

Microcurrent therapies involve applying weak direct currents (80 µA 1 mA) and are now being increasingly recognized as an adjunct for pain relief and autonomic nervous system regulation.[5,6] It is theorized that AC and DC electro-therapies have different modulating affects on the autonomic nervous system and pain outcomes.[7,8]

DC microcurrent therapies below 500 mca (≥ 0.5 mA) are reported to activate ATP, protein synthesis, and increased metabolism,[9] while higher currents inhibit these vital processes, suggesting low amplitude DC microcurrent is more

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Table 2. Pre-and Post-Treatment Patient and VAS Pain Descriptives and Results for MPS-SRT Applied to Scars (N=51)

N=51	Minimum	Maximum	Mean	Std. Deviation
Duration of Pain	90 days	36 years	7.61 years	1.339 years
Age (years)	21	72	47.34	12.812
Pain Before Treatment (0-10)	4	10	6.33	1.912
Pain after Treatment (0-10)	0	10	2.63	1.913
Follow up Pain - 2 Days after Treatment(0-10)	0	10	1.73	1.930

beneficial to cellular regeneration than high amplitude AC stimulation.[10-12]

It is also suggested in the literature that DC microcurrent mimics human bio-cellular communications, enhancing autonomic nervous system regulation[13] and the production of beta-endorphins,[14,15] resulting in a body-wide therapeutic benefits.

Whether the plausible explanations for accelerated fibrous tissue reduction with MPS applied to scars is systemically or cellular based is subject to further clinical research; it is clear MPS-SRT has positive pain and scar reduction benefits and could provide supportive role as a clinical adjunct to traditional neural therapy.

Advantages of SRT Over Traditional Neural Therapy

- Non-invasive
- Faster, safer to apply
- SRT can release deep internal adhesions or hard to get to scars (tonsillectomy)
- Multiple scars may be treated in single session
- Scars may be "connected" or release simultaneously
- May be applied by ancillary staff without complications

Chronic pain can limit quality of life, restrict work and social engagement, and is often blamed for the development of drug dependency of various forms. Scars and trauma are universal and impact the population of all professions and walks of life. Clinical feedback suggests that integrating scar release therapy is an important adjunct into any clinical setting for reducing muscle tone, fascial restrictions and sympathetic stress prior to any other therapeutic interventions. This simple non-invasive technique can quickly improve your ability to optimally heal your patients. "WorkSmarter, not Harder" is the motto in MPS scar release therapy.

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Kelly is author of *Functional Acupuncture for Women's Health* and co-author of *Functional Acupuncture for Pain Management*. Kelly Armstrong in an international speaker, lecturing extensively on the topics of scar release therapy, cranial release, and concussions, pain management, pediatric health, and women's health for over seven years. She is qualified continuing education (CE) provider for occupational and physical therapy nationally and has instructed over 250 pain seminars to date. Blog: www.QueenofScars.com

Literature Review & Commentary

by Alan R. Gaby, MD
drgaby@earthlink.net

Does Eating Nuts Help Prevent Cardiovascular Disease?

A meta-analysis was conducted on 19 prospective studies that examined the association between nut consumption and risk of cardiovascular disease (CVD). An inverse association was found between total nut consumption (comparing highest vs. lowest categories) and CVD incidence (risk ratio [RR] = 0.85; 95% confidence interval [CI], 0.80-0.91), CVD mortality (RR = 0.77; 95% CI, 0.72-0.82), coronary heart disease (CHD) incidence (RR = 0.82; 95% CI, 0.69-0.96), CHD mortality (RR = 0.76; 95% CI, 0.67-0.86), stroke mortality (RR = 0.83; 95% CI, 0.75-0.93), and atrial fibrillation (RR = 0.85; 95% CI, 0.73-0.99). No association was found with stroke incidence and heart failure.

Comment: In this meta-analysis of observational studies, increasing nut consumption was associated with a reduced incidence of, and reduced mortality from, various cardiovascular disease outcomes. Nuts are a good source of cardioprotective substances such as magnesium, essential fatty acids, and fiber. In randomized controlled trials, consumption of various types of nuts lowered serum cholesterol levels. The polyunsaturated fatty acids in nuts are relatively unstable and can become oxidized to form potentially toxic lipid peroxides. Nuts should therefore be stored in an airtight container in the refrigerator or freezer. Nuts are best consumed raw and unsalted.

Becerra-Tomas N, et al. Nut consumption and incidence of cardiovascular diseases and cardiovascular disease mortality: a meta-analysis of prospective cohort studies. *Nutr Rev*. 2019;77:691-709.

Does Eating Sugar Contribute to Heart Disease and Cancer?

The association between consumption of sugar-sweetened beverages and risk of total and cause-specific mortality was examined in 37,716 men from the Health Professional's Follow-up study (from 1986 to 2014) and 80,647 women from the Nurses' Health study (from 1980 to 2014) who were free from chronic diseases at baseline. During 3,415,564 person-years of follow-up, after adjustment for potential confounding variables (including age, smoking, alcohol intake, physical activity, family history of cardiovascular disease, history of hypertension and hypercholesterolemia, body mass index, total energy intake, and intake of whole grains, fruit, vegetables, red meat, and processed meat), consumption of sugar-sweetened beverages was associated with a significantly higher risk of total mortality, cardiovascular disease mortality, and cancer mortality. Comparing the highest and lowest categories of intake, the increase in risk was 21% for total mortality, 31% for cardiovascular disease mortality, and 16% for cancer mortality.

Comment: In this prospective cohort study, higher intake of sugar-sweetened beverages was associated with a higher risk of total mortality and death due to cardiovascular disease and cancer. Some of the biochemical effects of sugar intake that could promote the development of cardiovascular disease include increasing serum levels of triglycerides, uric acid, insulin, and cortisol; increasing platelet aggregation; and increasing blood pressure. Excessive sugar intake may also impair various aspects of immune function, which could conceivably increase the risk of developing cancer.

Malik VS, et al. Long-term consumption of sugar-sweetened and artificially sweetened beverages and risk of mortality in US adults. *Circulation*. 2019;139:2113-2125.

Vitamin D and Cardiovascular Disease

A meta-analysis was conducted on 21 randomized clinical trials (including a total of 83,291 patients; mean age, 66 years) that examined the effect of long-term vitamin D supplementation (at least 1 year) on cardiovascular disease (CVD) events and all-cause mortality. Compared with placebo, the risk ratio in the vitamin D group was 1.00 (p = 0.85) for major cardiovascular events, 1.00 (p = 0.92) for myocardial infarction, 1.06 (p = 0.16) for stroke, 0.98 (p = 0.68) for CVD mortality, and 0.97 (p = 0.23) for all-cause mortality.

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Comment: In observational studies, lower serum 25-hydroxyvitamin D levels were associated with a higher risk of CVD events. However, observational studies cannot prove causation. One potentially important confounding factor in these studies is that 25-hydroxyvitamin D levels decline in response to inflammation, and chronic inflammation plays a role in the pathogenesis of CVD. Therefore, the inverse association between vitamin D status and CVD risk that was reported in observational studies might have nothing to do with vitamin D, but might simply indicate that people with inflammation have a higher risk of CVD than people without inflammation. That possibility is supported by this meta-analysis of randomized controlled trials, which found that vitamin D supplementation did not decrease the incidence of major cardiovascular events, myocardial infarction, stroke, CVD mortality, or all-cause mortality.

Barbarawi M, et al. Vitamin D supplementation and cardiovascular disease risks in more than 83 000 individuals in 21 randomized clinical trials: a meta-analysis. *JAMA Cardiol*. 2019 Jun 19 [Epub ahead of print].

Magnesium Prevents Coronary Artery Calcification in Patients with Chronic Kidney Disease

One hundred twenty-five Japanese adults (mean age, 70 years) with stage 3 or 4 chronic kidney disease who had risk factors for coronary artery calcification (CAC) (diabetes, history of cardiovascular disease, high LDL-cholesterol level, or smoking) were randomly assigned to receive, in open-label fashion, in a 2 x 2 factorial design, 198 mg per day of magnesium (as magnesium oxide), 6 g per day of AST-120 (a carbon adsorbent), both treatments, or neither treatment (control) for two years. The magnesium dosage was adjusted every one-to-three months to achieve a serum magnesium level of 2.5-3.0 mg/dl. The primary outcome was the percentage change in CAC score. The mean dose of magnesium at the end of the study was 304 mg per day. Only 21.7% of the magnesium-treated patients achieved the target serum magnesium level. The median increase in CAC score was significantly smaller with magnesium than with control (11.3% vs. 39.5%; $p = 0.001$). The proportion of patients with a 15%-or-greater increase in CAC score per year was also significantly lower in the magnesium group than in the control group (24% vs. 62%; $p < 0.001$). The dropout rate was higher in the magnesium group than in the control group (27% vs. 17%), primarily because of diarrhea.

Comment: In this study, magnesium supplementation slowed the progression of coronary artery calcification in patients with stage 3 or 4 chronic kidney disease. Two lines of circumstantial evidence suggest that increasing magnesium intake might also prevent arterial calcification in people without chronic kidney disease. First, the typical Western diet contains low or suboptimal amounts of magnesium. Second, in previous animal studies, magnesium deficiency promoted the development of arterial calcification.

Sakaguchi Y, et al. A randomized trial of magnesium oxide and oral carbon adsorbent for coronary artery calcification in predialysis CKD. *J Am Soc Nephrol*. 2019;30:1073-1085.

Biotin May Interfere with Lab Test for Heart Attacks

The United States Food and Drug Administration recently issued a statement warning that laboratory tests for cardiac troponins (which are considered the gold standard for diagnosing acute myocardial infarction) may produce falsely low results in patients taking large doses of biotin, potentially leading to a missed diagnosis. In a new study, cardiac troponins were measured in 572 patients admitted to an acute cardiac care unit in the Netherlands. The measurement was repeated after biotin was removed from the serum samples by adding an excess of streptavidin-coated magnetic microparticles. Although an estimated 30% of people in Europe take dietary supplements (many of which contain biotin), there was no evidence of interference with any of the cardiac troponins tests.

Comment: These findings suggest that the amount of biotin present in a typical multivitamin or B-complex vitamin (typically 300 µg or less) does not interfere with lab tests for cardiac troponins. However, much larger doses of biotin are being used in some cases, such as 2 mg per day or more to treat diabetes, and up to 300 mg per day to treat multiple sclerosis. The present study does not rule out the possibility that these larger doses would interfere with tests for cardiac troponins.

Previous studies have found that high-dose biotin can also interfere with laboratory tests for testosterone, estradiol, progesterone, dehydroepiandrosterone (DHEA) sulfate, prostate-specific antigen, parathyroid hormone, luteinizing hormone, follicle-stimulating hormone, and vitamin B_{12}, as well as various thyroid function tests. Patients receiving high-dose biotin should be advised to discontinue the supplement for at least 72 hours before blood tests are done.

Vroemen WH, et al. Biotin interference in high-sensitivity cardiac troponin T testing: a real-world evaluation in acute cardiac care. *Cardiovasc Res*. 2019;115:1950-1951.

Can Vitamin D Slow the Progression of Alzheimer's Disease?

Two hundred ten Chinese patients (aged 65 years or older; mean age, 67.8 years) with Alzheimer's disease were randomly assigned to receive, in double-blind fashion, 800 IU per day of vitamin D or placebo for 12 months. The mean serum 25-hydroxyvitamin D level at baseline was 19.1 ng/ml, which is consistent with mild vitamin D deficiency. Compared with placebo, vitamin D significantly improved blood levels of biomarkers related to the pathogenesis of Alzheimer's disease (i.e., amyloid beta-related biomarkers) and significantly improved full scale IQ and several other measures of cognitive function. If this study is legitimate, the results suggest that a modest daily dose of vitamin D can slow the progression of Alzheimer's disease.

Comment: During the past year, I have pointed out several times in the *Townsend Letter* that a large and growing number of nutrition studies have left me wondering whether the research is fraudulent. While most of these eyebrow-raising papers are coming from Iran, many are coming from China. The present study from China raises a number of concerns.

First, while one of the inclusion criteria was age 65 years or older, the mean age of participants at baseline was 68.02 ± 5.90 years in the vitamin D group and 67.53 ± 5.15 years in the placebo group. Assuming a normal (Gaussian) distribution of

ages among the study participants, the age of approximately 16% of the participants would be at least 1 standard deviation below the mean for their group. Thus, 16 or 17 patients in the vitamin D group would be younger than 62.1 years, and 16 or 17 patients in the placebo group would be younger than 62.4 years. It therefore seems impossible that all of the study participants could have been at least 65 years old.

Second, in order to participate in the study, patients had to have a "clear mind" and to sign an "informed consent" form. However, scores on the Mini-Mental State Examination at baseline indicated that the participants on average had moderate-to-severe Alzheimer's disease. It is difficult to imagine how people with moderate-to-severe dementia could have a clear mind or could give consent that was truly informed.

Third, of the 210 patients who enrolled in the trial, only 1 failed to complete the full 12 months. Such a low dropout rate would be highly unusual for any clinical trial, and particularly for a trial involving patients with moderate-to-severe Alzheimer's disease.

Fourth, the mean serum concentration of 1,25-dihydroxyvitamin D at baseline was reported as 30 ng/ml, whereas the usual laboratory reference range for 1,25-dihydroxyvitamin D is 18-78 pg/ml. One nanogram (ng) is equal to 1,000 picograms (pg), so the baseline value reported in this study was 384 times higher than the upper limit of normal for 1,25-dihydroxyvitamin D.

Jia J, et al. Effects of vitamin D supplementation on cognitive function and blood Abeta-related biomarkers in older adults with Alzheimer's disease: a randomised, double-blind, placebo-controlled trial. *J Neurol Neurosurg Psychiatry*. 2019;90:1347-1352.

Thiamine and Heart Failure

Sixty-nine patients (mean age, 64 years) with heart failure and a low left ventricular ejection fraction (LVEF) were randomly assigned to receive, in double-blind fashion, 200 mg per day of thiamine or placebo for six months. Compared with placebo, thiamine had no significant effect on a measure of quality of life or on exercise capacity (distance walked in 6 minutes). The mean absolute change in LVEF compared with baseline was -1.6% in the thiamine group and +0.2% in the placebo group ($p < 0.05$ for the difference in the change between groups, suggesting that there was an adverse effect of thiamine on LVEF).

Comment: Thiamine (as thiamine pyrophosphate) is a cofactor for several enzymes that play a role in myocardial energy production. Severe thiamine deficiency is a well-recognized cause of heart failure (beri beri heart disease). Patients with heart failure may be at increased risk for thiamine deficiency, because of a diuretic-induced increase in urinary thiamine excretion, malabsorption, and advanced age. Laboratory evidence of thiamine deficiency has been found in 12-98% of patients with heart failure in various studies, depending on the assay method used. In case reports, thiamine supplementation improved cardiac function in patients with heart failure and thiamine deficiency. However, no clear benefit has been observed in randomized controlled trials, and in the present study there appeared to be an adverse effect on LVEF.

A possible explanation for the negative results in the present study is that the patients in the thiamine group were sicker at baseline than those in the placebo group (i.e., lower mean LVEF

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Brassicasterol 600 mcg		*
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and more patients with advanced heart failure). In addition, the thiamine group had a higher proportion of cigarette smokers (23% vs. 3%). These factors may have biased the study toward worse outcomes in the thiamine group.

Another factor to consider in this study and previous studies is that many patients with heart failure are deficient in magnesium, a nutrient that plays a key role in myocardial function. Magnesium is also required for the conversion of thiamine to its biologically active form, and some thiamine-dependent enzymes also require magnesium. In patients who are deficient in both thiamine and magnesium, symptoms of thiamine deficiency may not respond to thiamine supplementation unless magnesium deficiency is also corrected. Moreover, administration of large doses of thiamine can exacerbate magnesium deficiency.[1] Supplementation with large doses of thiamine (200 mg per day would be considered a large dose) could also conceivably promote imbalances with respect to other B vitamins, some of which also play a role in myocardial energy production.

Despite the negative results in the studies described above, it is premature to rule out the possibility that thiamine supplementation, when given in combination with magnesium and other B vitamins, could be beneficial for some patients with heart failure.

Keith M, et al. Thiamin supplementation does not improve left ventricular ejection fraction in ambulatory heart failure patients: a randomized controlled trial. *Am J Clin Nutr*. 2019;110:1287-1295.

Intravenous Iron: Proceed with Caution

A 36-year-old woman developed light brown skin staining after receiving intravenous iron. The staining, which was primarily a cosmetic problem, was still present two months later. Between the years 2000 and 2016, there were 51 reports in the French pharmacovigilance database of skin pigmentation (mostly brown) occurring after intravenous administration of iron. The pigmentation persisted for more than one month in 37% of cases and for more than six months in 18% of cases.

Comment: Intravenous iron is preferable to oral iron in certain clinical situations because it causes fewer gastrointestinal side effects and results in more rapid and more effective iron repletion. Intravenous iron is not risk-free: anaphylactic reactions have occurred on rare occasions, and hypophosphatemia as a result of increased urinary excretion of phosphorus occurs relatively frequently. The present report documents another potential adverse effect – staining of the skin. The risk of skin staining can presumably be reduced by practicing good injection technique and by inspecting the infusion site frequently for possible extravasation.

Crowley CM, et al. Skin staining following intravenous iron infusion. *BMJ Case Rep*. 2019;12:e229113.

References

1. Wester PO. Magnesium. *Am J Clin Nutr*. 1987;45:1305-12.

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Shorts

briefed by Jule Klotter
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Diabetes and Environmental Toxins

Back in August 2016, Joseph Pizzorno, ND, editor-in-chief of *Integrative Medicine: A Clinician's Journal*, wrote an editorial about the role of environmental chemicals in the 7-to-10-fold increase in type 2 diabetes over the past 50 years. While he agrees that obesity, lack of exercise, and sugar consumption are also factors, Pizzorno says the increase in sugar consumption, which started about forty years before diabetes began to rise, does not correlate with diabetes incidence. Instead, he says that environmental chemicals found in food, air, and drinking water – sometimes called obesogens or diabetogens – are the main cause. In researching this topic, Pizzorno found that people with the highest body load of persistent organic pollutants (POPs), such as PCBs, DDE, and hexachlorobenzene, have the greatest risk of diabetes – regardless of body mass. Obese people with low POP levels do not have an increased risk of diabetes while thin people with high POP levels do. These pollutants are among those that impair insulin sensitivity and/or decrease insulin production.

In his article, Pizzorno focuses on seven types of diabetogens: arsenic, bisphenol A (BPA), dioxins, organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs), phthalates, and polycyclic aromatic hydrocarbons (PAHs). Arsenic and chemicals in plastics appear to be the most problematic. Arsenic damages pancreatic β cells, decreasing insulin production. In many areas of the US, drinking water has arsenic levels that exceed the EPA limit (10 µg/L). High arsenic levels have also been found in seafood, rice, mushrooms, and poultry. Chemicals in plastics, including BPA and phthalates, are also major contributors to diabetes risk. BPA is used to make plastic water bottles, the inner linings of food cans, implanted medical devices, and other polycarbonate plastic items. BPA causes insulin resistance by blocking insulin receptors, resulting in visceral fat accumulation as well as increased diabetes risk. Phthalates, also used in food packaging, increase plastics' flexibility, transparency, and durability. These chemicals readily contaminate foods they contact, particularly foods that contain fats – such as milk, cheese, butter, and meat. Grease-resistant packaging used by fast-food restaurants and to make pizza boxes and microwave popcorn packaging is another source of these chemicals. In March 2020, *Environmental Health* published a consensus statement from an international group of scientists stating that chemicals in packaging, kitchen utensils, tableware, and food processing equipment need to be better regulated because these chemicals do leach into food and drinks and only a few have been investigated for health effects.

Dr. Pizzorno says that conventional laboratory tests, particularly GGT (aka GGTP) for measuring γ-glutamyl transferase, can indicate whether chemical load is a factor: "This enzyme recycles glutathione for detoxification of POPs and is induced in proportion to exposure." He refers to a four-year study in which increased GGT correlated with increased risk of diabetes (Lee et al. *Diabetologia.* 2003;46(3):359-364). Measuring arsenic content in toenail clippings is the best way to assess body load of this metal: "Blood and urine levels typically only indicate acute exposure."

Avoidance tops the list of Dr. Pizzorno's strategies for reducing toxic load. Increasing glutathione production (to facilitate phase 2 detoxification and help protect from oxidation) and increasing dietary fiber that binds to the chemicals so that they can be excreted are also essential. Dr. Michelle Perro presents a case history in this month's "Pediatric Pearls" column that illustrates the use of these strategies for reversing type 2 diabetes in a teenager.

In a 2019 podcast with Ari Whitten, Dr. Pizzorno says that diabetes is not the only disorder tied to environmental chemicals. Fluorinated hydrocarbons in Teflon-coated pans, GORE-TEX clothing, and Scotchgard carpet and furnishings increase uric acid levels, which leads to gout. PCBs bind to cartilage, which can lead to the autoimmune disorder rheumatoid arthritis: "… what was previously normal tissue in the cartilage, when you bind a chemical [or metal] to it, it is now an abnormal tissue and the body develops an immune reaction to that. We call it autoimmune disease."

Dr. Pizzorno published *The Toxin Solution,* which outlines the health effects of environmental toxins and how to reduce body load for the lay reader, in 2017. "There are about a hundred toxins now in our environment at high enough levels to induce disease in humans," he told Whitten, "and that is why I wrote

continued on page 32 ➤

Multi-Peptide ELISA is the most comprehensive method for the detection of Lyme disease and other tick-borne diseases.

(US Patent 7,390,626 B2)

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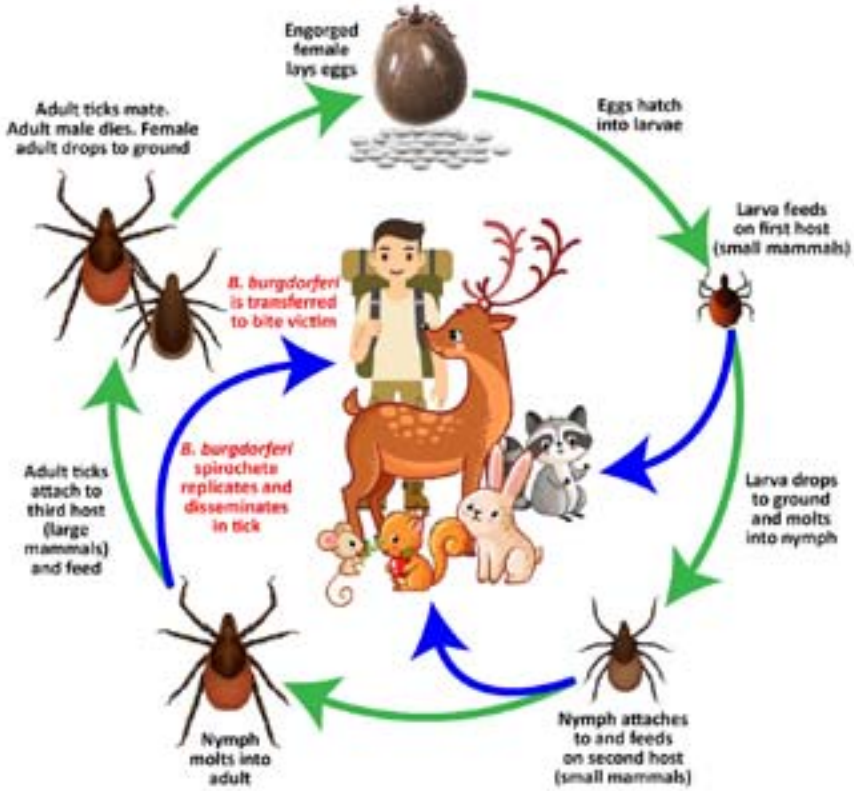

Untreated Lyme disease can turn into chronic Lyme disease, which in turn can lead to Lyme arthritis and neuroborreliosis.

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Shorts

➤ *continued from page 30*

that book." He also co-authored a textbook with Walter J. Crinnion called *Clinical Environmental Medicine.*

Gross L. Scientists say lax regulation of chemicals in food packaging endangers human health. March 2, 2020. https://thefern.org.
Pizzorno J. Is the Diabetes Epidemic Primarily Due to Toxins? *Integrative Med.* August 2016;15(4):8-17.
The Unknown Link Between Toxins and Disease and How to Detox Your Body with Dr. Joe Pizzorno (transcript). The Energy Blueprint podcast. September 14, 2019.

Exercise for Patients with CHD and Diabetes

Exercise/physical activity is a recommended therapy for people with diabetes and coronary heart disease (CHD); but, is moderate or vigorous activity more beneficial? High intensity exercise (anaerobic) has a stronger beneficial effect on HbA1c, the biomarker that indicates average blood sugar level over the previous two to three months. Consequently, some have thought more vigorous exercise would be better for diabetes patients. A 2019 German study challenges that belief. In their small pilot study, Bernhard Schwaab et al tested the effect of moderate exercise (aerobic) vs intense exercise (anaerobic) on patients with coronary heart disease who had been newly diagnosed with type 2 diabetes mellitus (T2DM), using the standard 75 g oral glucose tolerance test (OGTT). None of the participants were using any type of hyperglycemia or diabetes dietary intervention or medication, but all were on some type of medication for heart disease.

Out of 16 consecutive patients who met the inclusion criteria, only 10 completed the study; one refused to take part, and five could not complete the cardiopulmonary exercise testing (CPX) on a cycle ergometer because of angina (n=2), dyspnea (n=2), or muscle fatigue (n=1). The remaining participants took part in anaerobic exercise (CPX-1) at 7 am after an overnight fast, within 3-5 days after their diagnostic OGTT (OGTT-0); the goal was to reach a respiratory exchange ratio of 1.20, the point at which metabolic acidosis occurs "at the end of maximum incremental cycle ergometer exercise in sedentary men." After their heart rate, blood pressure and gases returned to resting values, the participants had another OGTT. The next morning at 7 am, aerobic exercise – which sought to maintain a respiratory exchange ratio between 0.90-0.95 – was conducted and a third OGTT ensued when heart rate, blood pressure and gases had returned to resting values.

The researchers found no significant differences in plasma glucose (PG) between the three OGTTs at the 1-hour measurement. At the 2-hour point, however, they found significant differences between the two types of exercise. Anaerobic exercise produced mixed plasma glucose levels among the participants with five showing a further increase in plasma glucose, two showing a decrease, and three having the same levels as at the 1-hour point. "Mean values at 2-h-PG did not differ between OGGT-0 (at rest) and OGTT-1 (anaerobic)." All patients showed a decrease in plasma glucose at the 2-hour point after aerobic exercise: "…the mean value was significantly

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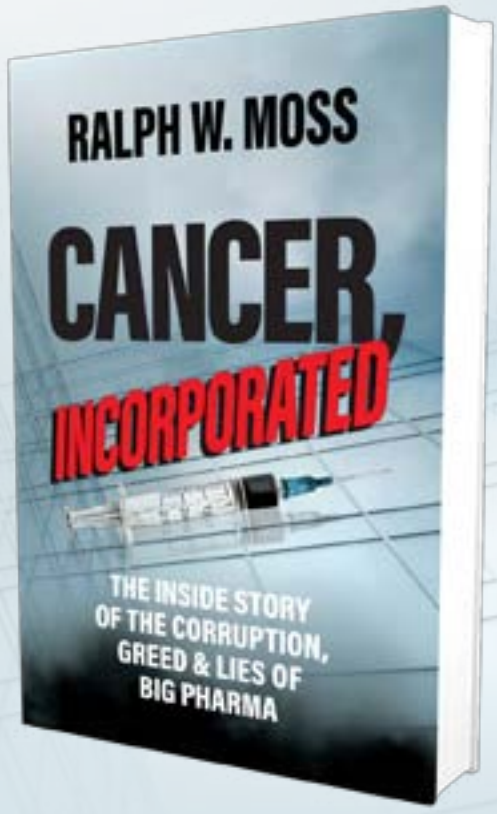

lower as compared to 2-h-PB in the screening OGTT-0 at rest (9.4 ±2.3 vs. 12.6 ± 2.2 mmol/l; p<0.05)." The researchers noted that patients with T2DM and coronary heart disease have highly individualized glycemic responses to exercise.

"A very low (to at most moderate) exercise intensity might be appropriate in this deconditioned cohort with T2DM and CHD, as shown in this study," conclude the authors. This study has several limitations: its small size, its short length, and the use of fasting measurements only. Non-fasting results may differ. The authors say, "Long-term response to different exercise intensities should be investigated without disregarding personal preferences for a specific type of exercise in order to increase the adherence to sustainable lifestyle changes in patients with T2DM."

In a commentary on this study, Stephan Jacob and Andrew J. Krentz relay that the European Society of Cardiology and researchers at Belgium's University of Hasselt have developed a pilot evidence-based interactive decision-support tool (www.escardio.org) for physicians who want to make exercise prescriptions for patients with cardiovascular risk factors or disease. In lieu of that, they suggest that patients exercise in the 'comfort zone' (in which he/she can walk and talk) "to avoid potentially detrimental anaerobic metabolic stress."

Jacob S, Krentz AJ. Exercise prescription in patients with type 2 diabetes and coronary heart disease: could less be more? *Cardiovascular Endocrinology & Metabolism.* 2020;9(1).

Schwaab B, et al. Effects of aerobic and anaerobic exercise on glucose tolerance in pateints with coronary heart disease and type 2 diabetes mellitus. *Cardiovascular Endocrinology & Metabolism.* 2020; 9(1).

Heart Rate Variability and Solar and Geomagnetic Energy

A 2019 study, conducted by researchers from Saudi Arabia, Lithuania, and the US (HeartMath and NASA Ames Research Center), found evidence that solar activity and geomagnetic activity affect heart rate variability. Every 10.5 to 11.0 years, the magnetic north and south poles on the sun change places (aka the solar cycle). As activity increases, the sun displays more sunspots and coronal flares and emits more ultraviolet and solar radio flux radiations. It has long been recognized that social unrest as well as human innovation and creativity increase as solar activity increases. But solar activity also affects individual physiology, particularly the nervous and cardiovascular systems. Based on scientific literature, the authors write, "It appears that sharp or sudden variations in geomagnetic and solar activity as well as geomagnetic storms can act as stressors, which alter regulatory processes such as melatonin/serotonin balance, blood pressure, breathing, reproductive, immune, neurological, and cardiac system processes."

Their study involved 16 women with no known physical or mental health disorders who were employed full-time at the Prince Sultan Cardiac Center in Hofuf, Saudi Arabia: 8 nurses, 6 housekeeping staff, 4 from the research department. The women's heart rate variability (HRV) was recorded for 72 consecutive hours each week for five months using Bodyguard HRV recorders (Firstbeat Technologies Ltd, Finland). HRV is the measure of beat-to-beat changes in heart rate and indicates autonomic nervous system function.

The women had individualized responses to the geomagnetic and solar activity changes; but when taken as a group, the authors found a correlation between daily autonomic nervous system responses and the solar energy. Specifically, they found "increases in solar radio flux, cosmic rays and Schumann resonance power are all associated with increased HRV and parasympathetic activity [the rest and digest response]." The authors say, "The findings support the hypothesis that energetic environmental phenomena affect psychophysical processes that can affect people in different ways depending on their sensitivity, health status and capacity for self-regulation."

We have no control over the solar cycle, of course. How and why environmental energy has a physiological effect is still unclear. Figuring out why some people are strongly affected by environmental energy and others are not may lead to new ways to promote health. Perhaps more and larger studies with different populations, new designs, and conducted in other locations will give more clues. Unfortunately, we still seem to be at the stage of downplaying the physiological effects of the electromagnetic energies (natural and man-made) in our environment. What you can't see, touch, hear, or taste can't affect you, right?

Alabdulgader A, et al. Long-Term Study of Heart Rate Variability Responses to Changes in the Solar and Geomagnetic Environment. *Scientific Reports.* 2018;8:2663.

Zinc and Hypertension Drugs

Many drugs used to control high blood pressure produce zinc deficiency, according to a 2018 study from Poland. The researchers recruited 98 patients (61 females; 37 males) who had not received treatment for primary arterial hypertension. For this randomized, prospective study, each patient was prescribed one antihypertension drug for three months. Thirty-six received a diuretic; 18 received a calcium antagonist; another 18 took a β-blocker; 14 took an angiotensin-converting enzyme inhibitor (ACE-I); and, 12 took an angiotensin II receptor antagonist. Participants were instructed to maintain their normal diets and activity levels throughout the study and to avoid taking any dietary supplements. Before treatment and after three months of therapy, the researchers took samples to measure iron (Fe), zinc (Zn), and copper (Cu) levels in serum, erythrocytes, hair, and urine.

While all the drugs produced a significant reduction in blood pressure levels, some also caused a decrease in zinc levels. Diuretics caused a significant drop in serum and erythrocyte zinc concentrations, and they increased the excretion of zinc in urine. Ca-antagonists produced a significant decrease in erythrocyte zinc concentration, and angiotensin-converting enzyme inhibitors caused a significant decrease in serum zinc concentration. Zinc is a cofactor for over 300 enzymes in the body. It is essential for DNA replication, energy production, glutathione activity, immune response, blood clotting, and more. Although this is a small study with more women than men, it might be prudent to check for zinc deficiency in patients who are taking a diuretic, Ca-antagonist, or angiotensin-converting enzyme inhibitor – and supplement accordingly.

Chasapis CT, et al. Zinc and Human Health: An Update. *Archives of Toxicology.* November 2011;86(4):521-34.

Suliburska J, et al. Diuretics, Ca-Antagonists, and Angiotensin-Converting Enzyme Inhibitors Affect Zinc Status in Hypertensive Patients on Monotherapy: A Randomized Trial. *Nutrients.* 2018;10:1284.

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On the cover

A New Way to Age

by Suzanne Somers

A new way to age is about successful aging. Clearly, aging is about worn out parts... this disrepair begins very early in life from poor diet and lifestyle habits. It all adds up. There is no *free lunch*.

At 73, I realize all my choices, throughout my life, good and bad, are now rearing their heads. Add to that – we are under the greatest environmental assault in the history of humanity. It's a daily bombardment of chemicals everywhere; our air is polluted; our water is contaminated. Did you ever think that water would be so precious and expensive? Growing up, water came out of the tap. That's what we drank, what we bathed in, what we watered our gardens with. In my home I found it necessary to install a water purifier, one that removes fluoride and that also has an option for alkaline water or purified water. This is called preventative – as in making choices to prevent disease down the road.

Conversely, poor choices of processed food and not tending to good lifestyle habits such as proper sleep, exercise, and clean food lead to an uphill battle. But it's never too late. Starting today you can reverse that damage and prepare your body to take advantage of this dramatic life extension we are all now afforded (like it or not). We will be kept alive, but in most cases with no quality of life. To me that is not living. I look forward to living an extra 20 or more years, but I want to be alive while I'm alive. That's what this book, *A New Way to Age,* is about.

Living longer is a beautiful thing. At my age I'm realizing that there's a whole new chapter I never thought was coming; it's peaceful, with satisfaction and contentment. The wisdom is coming rapidly, a lot of "ah ha" moments, and that makes everything make sense. So, I sleep easily, peacefully and happily.

What you think is very important, as our thoughts determine our outcome. We are in charge of our programming to be happy, sad or angry by the thoughts we think. Forgiveness is a gift to yourself. It frees you from being the victim. To not forgive allows 'it' or 'them' to win and you'll be the loser, the victim.

In my new book *A New Way to Age*, you will come to understand the importance of educating yourself to listen to the language of your body. It's always talking to you...the dry skin, stomach problems, hormone imbalance, constipation, insomnia, lack of libido, memory loss. That language is the body talking (yelling) begging us to listen and act forcibly. The sooner you "hear," the more quickly you can begin the process of reversing deterioration to essentially stop the clock.

My journey into health, which has led to my writing 27 books, started with my body language. It was talking all the time, but it took a long time for me to 'hear'. Depending on the severity of your individual decline, your lab results will determine exact deficiencies. Then it's about putting back what you are missing. It makes perfect sense when you're no longer making a full complement of balanced hormones, and a urine test will determine your deficiencies.

The new approach to aging is to put back all in which you are declining, be it hormones, minerals, nutritional

deficiencies. My goal with this book was to create a bible, a book where the uninitiated could be introduced to the new kinds of doctors who are doing such great work. And those who are already living life this new way will get fantastic new information about reverse aging, cellular health, the importance of jaw alignment to protect your heart (no one that I am aware of has made this connection except Dr. Leonard Feld whom I feature in this book).

All of these doctors have courageously stepped out of their standard-of-care box to treat people in a non-allopathic way. Because they are MDs, they are free to go to pharmaceuticals when it's absolutely necessary. When you need pharmaceuticals, they are a godsend for pain or infection or if natural remedies can't do the job. These doctors have other methods in their arsenal for building you up through supplementation, again determined by your deficiencies. I call it "filling the tank." In doing so, you get to be *you* again. It's glorious.

I'm so aware that there's a whole other life chapter I didn't know existed, but it's right now. I'm 73 and I've never felt better. I like the way I look. I have a strong libido. I have no bone loss because of my hormone replacement. My hair is silky and not stringy. I'm not terribly wrinkled. I have energy and a love of life that I've never had before. Every day of my life I live in gratitude. Again, thoughts create. What you think is what you experience. I meditate daily, in gratitude. It keeps me upbeat and happy.

Food is our fuel. If you had a Mazarati, one of the finest cars in the world, you would never put inferior fuel in it, nor would you ignore strange sounds and ticks. You'd take it right to a mechanic. The human body is more incredible than a Mazarati, yet we put inferior fuel into it regularly, and we frequently ignore the sounds or ticks in our body.

I believe *A New Way to Age* is my best book so far. We humans choose not to hear the language of the body – those aches, those symptoms, particularly menopausal symptoms, where you can't sleep, lose your libido, your hair loses its luster, or your skin starts to wrinkle, you can't lose weight, etc. A *New Way to Age* explains how to replace all you have lost by replacing hormones and using lab testing to determine your nutrient and mineral deficiencies and to determine your personal toxic burden.

There's much, much more but this book makes it easy for the reader to apply to himself/herself, and the book tells where to go to get the proper testing and finding the right doctor today. All doctors are not created equally. There are those who came out of medical school and are stuck in that thinking of allopathics – here's the symptom and here's the drug to fix it. Then there are the doctors I interview, who are bonafide MDs, educated at fine medical institutions, but who realize we've hit a wall with allopathic medicine.

When I start a book, I make myself a blank slate to see what emerges. I'm not a doctor, but I'm passionate about health and this book surprised me with talk about what I call the "collective consciousness" meaning that doctors are now consciously aware of the necessity for cellular health. It makes sense to me, being that each human being

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is approximately 40 trillion cells. This is true regeneration, true age reversal through breakthrough science such as removal of senescent cells. As we age, our cells gather debris just like the pipes in your home get clogged and then you have to call the Roto-Rooter man to come and clean them out. Removing cellular debris is possible by taking senolytics (a supplement I write about). You take it only once a week. Essentially, this supplement cleans out your cells, making them operate as they did in your youth, thus making you feel younger, giving you more energy, and restoring your youthful levels of NAD. NAD is a new supplement that repairs DNA breaks. Now you have one supplement cleaning out your cells, and the other one repairing them! This is true reverse aging. If your health is precarious you might prefer NAD by IV treatment to aid in repairing cellular damage. Right there, with those two supplements you're making a huge difference in your energy and health to turn back the clock.

With this book what I've noticed is acceptance. On this book tour not one interviewer or person attacked me. I was

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treated with respect and the book shot to #1 in its category the first day. What I realized mostly is that people are finally ready to hear this message. They see me and want what I have. I'm very open about my age, and my looks are clearly natural. What I do is keep my insides young – my cells, my hormones, my organs, my glands – by the organic food I eat (and grow myself) plus supplementation. It's up to each one of us to take good and tender care of ourselves. No one will care as much about you as you.

Along the way my devotees and followers have always stayed with me, as well as the doctors I feature. And now, best of all, doctors are switching over to a more integrative path. The patients are demanding it! As a result, doctors are swarming to switch over to integrative and alternative medicine in droves. It's very satisfying. The ocean liner is turning around big time. My readers are informed, making it easier for the doctor to not have to start with each patient in kindergarten. My readers are educated, so they can ask intelligent questions. When I first started out, I could only find 30 doctors in America who were informed in alternatives. Now there are thousands of these doctors all over the world. Hopefully my books have contributed to the movement.

I live the advice I offer, and my constituency knows this. It's also important to know that you can do everything right and still be affected by something out of our control. The environment and the world in which we live is tragically polluted and filled with EMFs and electromagnetic radiation from our cell phones. Plus, we are bombarded with electromagnetic radiation from our computers, night and day. Clearly, we are all going to be affected in some way. Sadly, it's an experiment on us in this modern era. But if it were to happen to you, if you were to be affected by the environment or cancer, the information in this book will give you the tools to make your body stronger to fight and win.

My plan for the future is to never stop! I love what I do. I make sure to enjoy each day and to be grateful for the love in my life, my incredible husband, my health, my wonderful food, my beautiful family. I am "present." I try not to miss anything. There's a beautiful quote by Lao Tsu:

> If you are depressed, you are living in the past.
> If you are anxious, you are living in the future.
> If you are at peace, you are living in the present.

I work hard to always be present, and I am present.

I urge people to go to SuzanneSomers.com for information, and products such as my incredible Suzanne Organics Skincare, Hair Care, & Cosmetics. These products are clean and toxic free. I urge readers to take a look. We've worked so hard on making them the best and purist products available. I also urge readers to go to my IGTV and Facebook Live shows, which we have two or three times a week from my home. You may find me at Big Al's Bar where my husband Alan and I will share a tequila. I can feel our viewers at home pouring one with us and we have a fun little chat. Sometimes I do my shows in bed! Yesterday I did a show from my outdoor tub in the wilderness to have fun using my organic bath and body products. I make it fun, and kind of sexy. We have so much fun and sometimes it gets a little naughty! All my shows are archived on my Facebook account so if you want to see how I am aging, these shows will give you a firsthand account of A NEW WAY TO AGE! ◆

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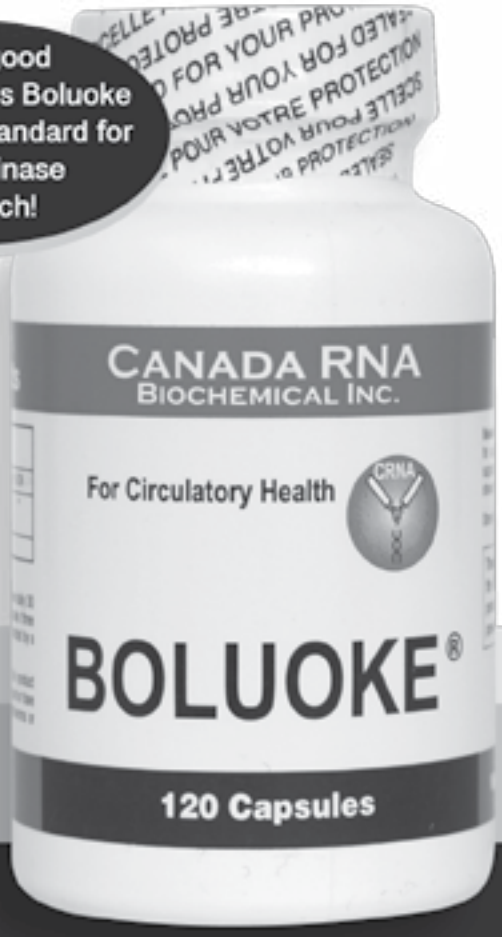

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Molecular Fire Prevention: Understanding Accelerators of Inflammation in Atherosclerosis and How to Avoid Them

by Fraser Smith, MATD, ND

Cardiovascular disease is the number one cause of mortality in the United States and worldwide. The primary target for medical therapy continues to be levels of LDL cholesterol. While the absolute reduction in risk by targeting LDL can be debated, there is compelling evidence that LDL elevations generally increase risk.

But this is not the whole story. There is a powerful, underlying phenomena that accelerates risk; and like many other chronic conditions, it is inflammation. Let's examine the nature of atherosclerosis as a process and how inflammation really accelerates it. Then, in the second half of this paper, we will look at some key drivers of inflammation. That will include strategies to detect and avoid them and nutritional and botanical considerations to mitigate their effects.

A Fire That Grows

Atherosclerosis is defined as the deposition of lipids into a plaque in the lining of medium-sized arteries. This plaque has an inner core of oxidized LDL as well as unfortunate white blood cells that attempted to scavenge it and died in the process. There is a covering on this cellular debris of fibrous tissue. Although this keeps the plaque from shearing off and blocking blood flow, this "scar tissue" that covers the plaque of oxidized LDL (cholesterol) and dead cells is never quite the normal inner lining (endothelium) of the artery.[1] It remains vulnerable to sucking in more oxidized LDL.

The precise causes of the beginning of this vicious cycle are not fully known. Oxidative stress in the vicinity of the arterial endothelium, in younger years, can cause it to dysfunction. High circulating LDL makes LDL invasion of the artery more likely. Oxidation of LDL makes it a more dangerous particle.

Inflammation is the central phenomenon. In the case of atherosclerosis, a positive feedback loop emerges. Typically, in the regulation of the body's systems, we see lots of negative feedback loops, but not positive. A negative feedback loop can be like thyroid hormone: Levels of thyroid hormone drop, the pituitary releases factors to get the thyroid to make more, as levels of thyroid hormone rise, the pituitary makes less thyroid stimulating hormone. This is how a control system works.

In atherosclerosis, a cycle of events feeds forward and reinforces itself. Oxidative stress damages tissue and damages the LDL particle.[1] The LDL particle migrates to the arterial intima (inner lining). The immune system attacks. More inflammation means more damage. The damaged tissue is more susceptible to LDL penetration. Higher inflammation means more damage to LDL, and so on.

It is possible that inflammation is not the ultimate root cause. Perhaps a conflagration of factors simply set in motion a vicious cycle. New lines of research examine the phenomenon of "efferocytosis," which means to remove dead matter. In the human body, cells die constantly. The immune system, mostly through scavenger cells, removes these dead cells. In the lining of an artery undergoing atherosclerotic changes, the toxic matter (oxidized LDL) and the dead scavenger cells that went in to ingest the toxins and never came out, somehow remain embedded in the tissue. This sets up recurring and unwanted attention from immune cells, who ramp up the inflammation and yet cannot completely resolve the problem. An analogy would be a splinter or foreign body that causes an abscess where the body simply cannot expel it.

While future treatments for atherosclerosis may zero in more and more on immune phenomena and the failure of macrophages to carry out effective efferocytosis, there is no doubt that increased inflammation is no mere symptom. It helps accelerate the disease process. When inflammation is high, that indicates a higher risk of atherosclerosis progression.

The naturopathic medicine approach to inflammation in this case involves the following:

A. Address the determinants of health, which includes removing toxic exposure or disturbances.
B. Make dietary changes to modulate the inflammatory response and support normal function.
C. Have the patient engage or use practices that support overall health, such as stress reduction, acupuncture, cardiovascular exercise.
D. Use specific natural medicines to reduce inflammation and to improve the integrity of the arteries.

With a view to "A," let's examine some, but certainly not all, of the factors that can drive inflammation.

The Fuels That Drive Inflammation

Arsenic: A variety of pro-inflammatory toxins can either damage the lining of the artery or accelerate inflammation. A prime example is arsenic, which is known to pathologists as a toxin to the cardiovascular system.[2] It can increase the risk of death from heart attacks and accelerate atherosclerosis. Arsenic causes a burst of oxidative stress in the body, damage to DNA and mitochondria, and oxidation of LDL.[2,3] Groundwater in many parts of the United States exceeds the EPA allowed level of arsenic; and since well water is not regulated, some sources are quite high in arsenic.[4]

Processed Vegetable Oil: For years, health authorities urged people to substitute "bad" animal-sourced fats with vegetable oil. That would be good advice if the vegetable oil in question were pure, undamaged, and chock full of its natural antioxidants and sterols. Unfortunately, the vegetable oil that crowds the shelves and makes up most of the cooking oil in America's very busy restaurant industry is pro-atherogenic.[5]

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Case Study

A 59-year-old male presents with occasional tightness of the chest and several episodes of a mild ache in the chest that came on from exertion. The pain is localized to his left chest and subsides after a few minutes once he "catches his breath." He has no other major health problems except some difficulty sleeping due to occasional leg pains in the night. His vital signs are as follows: pulse 74 bpm; respiration rate; 17/min; temperature 98.8°F; blood pressure (left arm sitting) 150/66.

He smoked one pack a day from the age of 18 to 32 and then quit because his fiancée was very intolerant of smoking. He is a salesman for a roofing company. When business is slow, like in the dead of winter (he lives in central Illinois), he drives Uber and even does some hours at an Amazon fulfillment center, which is adjacent to the nearby interstate.

He and his wife of 26 years have two children in college, and he has helped them out; but he is under enormous stress financially. The household credit card debt is so high he has considered declaring bankruptcy, but he is concerned about his ability to co-sign on the children's student loans in that case.

The family home is in small town, and everyone has a private well. He is aware that the high mineral content of the water can also have some toxins, and he maintains a water softener machine with his pump; but he is so hard-pressed financially that he has not bothered replacing the filters in the filtration system.

His diet is fairly high in meat, potatoes, and not too high in plant foods. He tends to eat a fair amount of takeout food due to his essentially working two jobs and not being home very often. A typically day's diet might include coffee, a bagel with peanut butter, hash browns, or donut, French fries and a burger, spaghetti and meatballs and sometimes a salad.

Although not as troubling as the pain in his chest, he does have gum disease and cannot afford to get the extensive deep cleaning and possible periodontal surgery that his dentist wants him to have. He has several pockets of infection deep in his gums and some temperature sensitivity to foods and drinks.

Testing is ordered to check out his general level of inflammation. He has an elevated C-reactive protein, and a urinary test reveals very high F2-isoprostanes. These are byproducts of arachidonic acid that are formed by high oxidative stress in the arterial surface. High F2 isoprostanes are associated with possible vascular damage and much higher risk of metabolic syndrome. He also receives an ADMA/SDMA test to see if his nitric oxide levels in the arteries are low, which leads to vessel tightening. His total cholesterol is 205 mg/ dL and his LDL is 155 mg/ dL.

Referral to a cardiologist is made, and he receives a stress test which shows signs of reduced heart performance and ischemia due to exercise. He cannot afford an angiogram due to his poor health insurance, but he does find an imaging center that will do a coronary artery calcium scan for 70 dollars. He scores high, about 800, which shows calcification in his heart arteries.

He is immediately put on a Mediterranean diet although in his case he is doing a bit less seafood and is allowed some lean chicken meat as well. He finds that he likes the extra virgin olive oil from Costco and begins to use it generously. He eliminates almost all seed oils, like soy and canola, from his diet and this means eliminating restaurant food, which actually is helpful to his budget. He brings raw trail mix combos in individual bags from Trader Joe's to his work so he can get energy in between meals. Although his diet is still a bit pasta heavy, it has about six servings of fruits and vegetables a day, which is a vast improvement. His dietary magnesium intake has probably tripled.

He begins to eat two squares of 85% cocoa extra dark chocolate per day and drinks green tea twice per day. He is told to take 3 grams of vitamin C per day. Also, he diverts some of his saved money from restaurants to a deep dental cleaning and irrigation (but not surgery) and begins to rinse with a hydrogen peroxide solution every day.

The Home Depot offers a special on home water testing kits, and he has his well water tested. It turns out to be high in arsenic (30 ppb), which surprises him. He replaces his old and useless filter for the household water and resolves to keep it up to date.

He is retested for C-reactive protein and F2-isoprostanes after three months (he asked to wait on other tests for the sake of his pocketbook) and C reactive protein has dropped from 'high' to "moderate' risk for cardiovascular disease. His F2 isoprostanes greatly improved and are normal. His LDL is down to 120 mg dL.

He is not having any chest pain, but he will get a stress test in a few months and hopes to see his lipids improve. He certainly feels healthier and more energized than he can remember feeling.

Molecular Fire Prevention

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This issue is somewhat fraught, as there is positive evidence that polyunsaturated fatty acid (PUFA) substitution for saturated fats can help prevent heart disease. The problem is the heating and chemical solvents during extraction, the bleaching to provide a neutral taste, and the fact that they are often overheated in both commercial frying and home use.

When PUFAs are oxidized, they form a number of free radical propagators, such as lipid hydroperoxides, that can increase inflammation in the artery.[6-8]

Plastic Byproducts: Linked to cancer, plastic conditioning agents, such as bisphenol A are also linked to atherosclerosis. More of this evidence is, for now, in animal research models, like most toxicology information; but it appears that the damage to arteries is real. Interestingly, in test animals, the BPA-exposed rabbits developed more insulin resistance, obesity, and fatty liver.[9,10] This shared traits with "metabolic syndrome" as we see it in humans. Moreover, plasticizers have been found to be "obesogens" that can promote obesity in humans.[11] They do this by increasing resistance to insulin but also by causing fat cells to proliferate.

Stress: The most general and pervasive phenomenon, stress, can energize us in the right amounts and act to exhaust and prematurely age us in larger exposures. This is especially true in situations where we cannot escape stressful situations that we cannot accept. A combination of metabolic syndrome, obesity, and stress can be particularly atherogenic.[12]

Air Pollution: Although the connection between particulate matter, ozone, and other forms of air pollution to lung disease is clear enough, this source of toxins can also accelerate atherosclerosis. The calcification of arteries that is associated with progressing atherosclerosis was found to be correlated with air pollution exposure in over 6700 adults in six US metropolitan areas.[13]

Periodontal Disease: Chronic gum disease that progresses to deeper infection and inflammation can raise inflammation across the body. This is because there are pockets of bacteria that can be held in check but not completely removed. This serves as a constant provocation of the immune system, which causes white blood cells to release higher levels of inflammation activating signals. This inflammation can definitely increase cardiac risk.[14] It has also been found that chronic periodontal disease activates certain inflammasomes, proteins that are part of our basic innate immune response that can mount non-specific but powerful responses to invading bacteria. Inflammasomes can, in turn, activate enzymes that cause increases in signals that fire up immune cell activity.[15,16]

Dousing the Flames

It is clear from just examining a few common examples of pro-inflammatory influences that a concerted effort to identify and reduce (or eliminate) pro-inflammatory influences is needed for many, many people. This may seem daunting or even hopeless, but intelligent changes can pay off. For example, an efficient filter for drinking water as well as some regular testing can help reduce heavy metals. Purchasing healthy "cold pressed" plant oils and not overheating olive oil when cooking can reduce vegetable oil toxins. Additionally, reducing restaurant visits can cut off much of these unhealthy oils at their source.

The work of reducing our risk exposures can seem to incite more and more effort, longer and longer "to-do" lists, and a lot of expense, an approach that can simply increase stress. On the contrary, simplifying our life with slower eating, natural eating, and a growing awareness of what is healthy and natural and what is synthetic and potentially harmful can take root. Not all societies are as driven and as out of touch with the rules of healthy living as ours. So, while it takes an effort to make changes, over time a lot of health-promoting practices that properly address the determinants of health can become the new normal.

There are divergent approaches to healthy eating and individual variances in genetics and biochemistry. It is a safe bet for practically everyone to emphasize plant foods in their diet.[17] While advice from conventional health sources (such as the American Diabetic Association as one example) are putting the concept of a plate loaded with plant foods out there, the actual practice in our society has some catching up to do. Processed foods, restaurant foods, and a loss of cooking skills in some homes has taken its toll. Still, the resurgence in interest in cooking, the growth of interest in natural foods, organic and non-GMO foods, and locally sourced foods gives reason for hope.

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It should be noted here that there are specific tests that can help identify cardiovascular inflammation.[18] A common one is C-reactive protein, which many internists will be happy to order. But there is much, much more on the menu and integrative physicians are often quick to take advantage of this insight and would do well to do so.

Nature's Fire Brigade

It should be clear that merely throwing supplements at atherosclerosis risk, in particular the unseen but relentless inflammation in the arterial inner lining is no solution. Addressing the determinants of health can never really be bypassed. Nevertheless, supplements, plant extracts and nutrients can do a lot of good. A few examples are the following:

- Curcuminoids from turmeric can interrupt inflammation at multiple points.[19]
- Grape seed extract and other sources of procyanidolic oligomers (including many berries) can strengthen the lining of the arteries and reduce oxidative stress.[10]
- Culinary herbs and spices, such as pepper, ginger, garlic, onion, cinnamon and chili, are packed with polyphenolic molecules and other useful compounds that can slow disease progression.[20]
- Magnesium deficiency, a double digit and widespread phenomenon, can undermine the cardiovascular system. Magnesium can strengthen the arterial structure, make vessels more elastic and flexible, and promote the synthesis of protective antioxidant enzymes in the cell.[21]

Conclusion

Patients need a very complete inventory of the disturbances to the determinants of their health in order to lower their atherosclerosis risk. Changes to lifestyle, diet, and the use of the very best of nutrients and herbal medicines can change the physiology and inflammation of their arteries and their body as a whole. These changes cost very little and can pay dividends across time.

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Heart Failure – A Metabolic Approach

by Joel Kahn, MD, and Daniel Chong, ND

Heart failure (HF) is a syndrome of cardiac dysfunction in which the heart can no longer pump enough blood around the body to support its organs and systems. Identified risk factors for HF include conditions that damage the heart, such as hypertension, coronary heart disease, heart attack, and diabetes; also lifestyle factors such as smoking, obesity, lack of exercise, and a high-fat and/or high-sodium diet.[1] While HF may occasionally come on suddenly for certain reasons, the majority of cases are typically chronic and worsen over time without care. For the purposes of this article, HF will refer to chronic HF specifically. If untreated or poorly treated HF continues to worsen, it will eventually become congestive heart failure (CHF), in which fluid may back up into the lungs, liver, gastrointestinal tract, arms, and/or legs. HF afflicts over five million persons in the United States, at a cost of over $30 billion yearly.[2] It also accounts for over 1 million hospital admissions yearly and is the cause of one out of every nine deaths.[2] With over 800,000 hospital admissions for HF occurring each year, despite only a minority ending up truly requiring acute care, hospital initiatives have been launched to decrease readmissions for HF within the first 30 days of discharge.[3]

The Awesome Foursome

An orthomolecular approach to improving energy production in the heart was proposed at least a few decades ago.[4,5] However, Dr Stephen Sinatra may be given credit for popularizing a metabolic therapy approach to HF in the 1990s when he began using supplements in his cardiology practice to restore cardiac energy production. Based on earlier research findings, Dr Sinatra recommended coenzyme Q10 (CoQ10), L-carnitine, and magnesium, and identified beneficial responses.[6] His aforementioned colleague, Dr James Roberts, suggested adding D-ribose powder to the combination, and the "Awesome Foursome" was created, as detailed in their book.[7] These agents will be featured here as primary therapeutic interventions that seek to address the unique needs of this crucial organ.

Coenzyme Q10

An antioxidant and critical component of cardiac energy production, CoQ10 is concentrated in healthy heart muscle and can become deficient in the myocardiocytes of patients suffering from HF.[8] The most definitive evidence to date can be found in the Q-SYMBIO study, which was a placebo-controlled, double-blind, randomized trial of patients with chronic HF.[9] In this study, 420 HF patients were randomized to CoQ10 (100 mg TID) or placebo and followed for two years. At 16 weeks, there were no differences between the two groups in heart function, six-minute walk test, or circulating biomarkers (brain natriuretic peptide, or BNP). However, the long-term, primary endpoint of a composite of major adverse cardiovascular events was reached by 26% of the placebo, vs only 15% of the CoQ10 group.[9] In addition, cardiovascular mortality, all-cause mortality, and incidence of hospital stays for chronic HF were significantly lower in the CoQ10 treatment arm.

D-Ribose

D-Ribose shares the structure of the backbone of ATP and is a 5-carbon sugar, affording it some unique properties that show promise in the treatment of HF. Animal research has shown that heart cells subjected to ischemia (acute or chronic) experience a reduction in ATP production capacity.[10] What's more, while recovery from the effects of ischemia is typically sluggish due to certain rate-limiting enzymatic steps in the pentose phosphate pathway (PPP), supplemental D-ribose appears able to enter the PPP and bypass the rate-limiting steps, leading to improved efficiency of ATP production.[10,11]

Importantly, human clinical trials have shown related effects. A 1992 study of stable but severe coronary artery disease patients found that supplementing these patients with D-ribose enabled them to increase their "ischemic threshold" and exercise longer with fewer symptoms.[12] Additional studies have demonstrated similar effects of D-ribose on the enhancement of depleted cellular energy stores following ischemia.[13] The same authors also reviewed preclinical and clinical trials examining D-ribose that demonstrated its ability to improve diastolic heart function, quality of life, and ventilatory parameters.[14]

Magnesium

Magnesium is a crucial cofactor in many pathways involved with cardiac bioenergetics, yet it is understudied (and underutilized) in conventional cardiology.[15] Compounding this problem, recent research has shown that nearly half of all Americans do not consume recommended amounts of magnesium from the foods they are eating.[15] The likely deficiency created by such poor intake is then amplified by various pharmaceutical agents frequently prescribed to these same people, such as certain types of antacids, antibiotics, and birth control pills. HF patients – the very people who may need magnesium the most – are also frequently prescribed medications for their condition that further worsen the likelihood of magnesium deficiency, namely diuretics and the cardiac glycoside, digoxin.[16] It is interesting to note in one study that higher serum magnesium levels in patients hospitalized for HF were actually associated with increased all-cause mortality.[17] However, after adjusting for baseline differences, this association was no longer significant, and factors such as advanced age and poor renal function were considered more likely than magnesium to be related to the excess risk.[17]

Given that magnesium has effects on over 300 enzymatic processes and is the second most abundant intracellular cation (next to potassium) in the human body, the mineral certainly plays a significant role in human health beyond its impact on heart function.[18] Key heart-related functions of magnesium, all of which would impact HF, include impacting potassium's role in electrical conductance and modulating heart cell function by contributing to the maintenance of the sodium/potassium and calcium gradients of the cardiac cell membrane. Thus, through various means, magnesium exerts a powerful influence on the electrical and contractile functions of the myocardium.[18] Its physiological contributions to cardiac function, in turn, suggest the important role a deficiency would play in HF, and an equally important role optimization of tissue magnesium levels would play in its treatment. Research in support of this conclusion has thus far been promising, including cardiac arrhythmias – frequently seen in HF patients – responding positively to both long-term oral supplementation[19] and acute IV administration of magnesium.[20]

A recent double-blind, placebo-controlled study also showed magnesium orotate supplementation over the course of one year to improve survival rate in severe HF patients by nearly 25%, while also significantly improving their symptoms and quality of life.[21]

An integrative, metabolic approach can improve heart bioenergetics, left ventricular function, and quality of life.

L-Carnitine

Considering the significant impact dysfunctional mitochondria appear to have in the pathogenesis of HF, as well as research on its use thus far, L-carnitine should be a consideration if other therapeutic interventions do not yield sufficient results. However, new data suggests that reevaluation of this perspective may be appropriate.[22] Researchers at the Cleveland Clinic recently commenced a hunt for new dietary factors and metabolites related to developing clogged arteries, or atherosclerosis, and eventually published data in 2013, identifying a compound known as trimethylamine-N-oxide or TMAO, which can rise in the bloodstream after meals, and can be considered pro-atherogenic, or plaque-causing.[23] The study detailed how, in the omnivores studied, the amino acid L-carnitine, found in meat (especially red meat) and choline, found mainly in egg yolks, could be metabolized by the gut microbiome into the metabolite TMA, which was then converted into TMAO in their livers.

The study also found a strong correlation between the blood levels of TMAO and rates of atherosclerosis and kidney disease. Interestingly, they also published more data that year, this time involving volunteers eating a vegan diet, to see if they would also produce TMAO after consuming L-carnitine, like the omnivores did.[24] To accomplish this, the vegan volunteers agreed to eat steak like the omnivores did (for research purposes only). Unlike the omnivores however, the vegans produced far less TMAO after the steak challenge. From this data, the researchers hypothesized that the microbiome of vegans was different than omnivores and does not produce TMA (and subsequently TMAO) when exposed to L-carnitine, as observed in the omnivores. They reported that "the present studies suggest reduction in carnitine and total choline ingestion with attendant reductions in TMAO levels may contribute to the cardiovascular health benefits observed in vegan/vegetarians."

Interestingly, published data relating TMAO levels and cardiovascular health also appear to be of interest in the treatment of congestive heart failure. In a review of TMAO and outcomes of cardiovascular disease, TMAO levels were significantly associated with a 1.18 to 1.79-fold increased mortality rate in people with chronic heart failure.[25] Also of note, in patients with type 2 diabetes, higher TMAO levels were significantly associated with a higher overall mortality rate of 2.1 to 2.7-fold.

In another study of TMAO metabolites and congestive heart failure, 22 patients admitted for congestive heart failure and 11 age-, sex- and comorbidity-matched, hospitalized control subjects without a history of HF were studied.[26] Plasma concentration of TMAO was found to be increased in heart failure patients compared to those without heart failure.

Again, these findings provide important evidence that, while L-carnitine supplementation is potentially therapeutic for people with HF, it should be used with caution, and if included in a treatment plan, TMAO levels should be closely monitored over time.

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Additional Supplements and Other Alternative Therapies

Taurine is a sulfur-containing amino acid that may augment the role of conventional medications for HF because of its role in inhibiting the harmful effects of catecholamines and angiotensin II in HF.[27] In a randomized study of placebo vs taurine supplementation in HF, at a dose of only 500 mg TID over two weeks, various aspects of exercise performance increased significantly in patients receiving the active supplement.[28]

Berberine is a quaternary ammonium salt found in medicinal plants such as goldenseal, Oregon grape root, and coptis. In a study of 156 patients with CHF, in which all patients were given conventional medications for the disease, those who were also given 1.2-2 g of berberine per day experienced improvements in ejection fraction, exercise capacity, and dyspnea, as well as a decrease in the frequency of complex premature ventricular contractions (PVCs), compared to the placebo group.[29] Long-term follow-up also revealed a significant decrease in mortality rate in the berberine-supplemented group.[29]

Various known physiological effects of berberine, including inhibition of smooth muscle contraction, reduction of inflammation, platelet aggregation inhibition, and inhibition of ventricular tachyarrhythmias, may help explain its apparently potent effects on HF patients.[30,31] Nonetheless, there is some suggestion that long-term use of berberine may inhibit cytochrome P450, resulting in various potentially negative effects on the clearance of drugs and possibly other toxins; thus, caution is warranted until further study can elucidate the impact of such findings.[32]

External Counter-Pulsation Therapy

One therapy for congestive heart failure that is under recognized and rarely recommended is external counter-pulsation (ECP). Years ago, a device known as an intra-aortic balloon pump, or IABP, was developed to aid failing hearts. While effective, it required insertion of a catheter inside of an artery in order to provide a rhymical inflation of a balloon pump timed to the heartbeat. Soon, however, researchers in China and subsequently New York found they could duplicate the support of the failing heart with external pressure cuffs and external counter-pulsation (ECP) was developed. Although first developed for anginal chest pain in the setting of badly blocked heart arteries, studies extended the evaluation to include congestive heart failure.

A registry was published that involved treating patients with advanced heart failure with ECP. Article co-author, Dr. Joel Kahn, participated in those early studies. Data were retrospectively analyzed from 127 New York Heart Association class II-IV CHF patients. The patients were divided into three groups based on the pressures applied during ECP. In the year following ECP using the most intense therapy, all-cause mortality was only 1.9% (one of 54 patients), whereas over the same time period the mortality rate was 7-8% with the lower pressure ECP group. For the Low, Mid, and High ECP pressure therapy groups, respectively: 1) average left ventricular ejection fractions increased 23%, 20%, and 17%; 2) NYHA class declined 36%, 29%, and 29%; and 3) all-cause hospitalizations, including terminal admissions, were reduced 86%, 83%, and 57% in the year following ECP therapy from the prior year. Also of note, there were no adverse effects or withdrawals from the ECP therapy and no significant difference in sex-based outcomes.

H2RA Therapy

Another novel therapy of congestive heart failure is to utilize the H2 histamine antagonist (H2RAs) receptor system which exists on heart tissue.[33] Histamine H2 antagonists have long been suggested to have beneficial effects on congestive heart failure (CHF). However, full agreement about the cardioprotective effects of H2RAs is still not reached yet. A total of 10 studies (472 participants) were included in a meta-analysis. H2RAs exhibited significant negative inotropic and chronotropic effects to reduce heart rate. Furthermore, although H2RAs did not affect the blood pressure in healthy volunteers, they significantly decreased the blood pressure of CHF patients. Additionally, H2RAs were also associated with significant increase in pre-ejection period and the ratio of pre-ejection period to left ventricular ejection time. In summary, the findings showed that H2RAs exerted negative inotropic and chronotropic effects to reduce heart rate and blood pressure, which, similar to beta-adrenergic receptor blockers, might decrease myocardial oxygen demand and eventually result in improvement of CHF symptoms.

If you have CHF, and you decide with your doctor to try adding H2RAs to your treatment arsenal, please also be aware there is limited data suggesting the possibility that this class of medication may negatively affect B12 absorption. Therefore, it would be prudent to consider monitoring serum B12 and/or methylmalonic acid (MMA) levels if taking long term.

Case Study

AB is currently an 87-year-old woman who lives independently and enjoys friends and family events. Three years ago, she was admitted to a local hospital with shortness of breath, fatigue, and edema, and was diagnosed with congestive heart failure.

Exam and Treatment: An echocardiogram identified an enlarged heart, a reduced ejection fraction of 35%, and moderately severe mitral regurgitation. A coronary angiogram showed normal arteries. She was started on diuretics, carvedilol, and an ACE inhibitor. She was considered for a mitral valve clipping to reduce the amount of valve insufficiency. After several consultations for this new procedure, she consulted with Dr. Kahn, and a metabolic cardiology program was instituted on top of the standard medications. She began CoQ10 (400 mg/d), L-carnitine (500 mg BID), D-ribose powder (5 g TID), and magnesium taurate (250 mg/d).

Follow-up: AB remained free of dyspnea over the coming months, and a follow-up echocardiogram four months later demonstrated her left ventricle to be of normal size, her ejection fraction to be 60%, and only mild mitral insufficiency remaining. Now, almost three years later, she remains only on carvedilol plus the metabolic supplements, and is free of CHF.

Summary

Heart failure is a serious disorder that can cause great suffering and shorten life. Current therapies with lifestyle alterations and pharmaceutical agents are of value but can simultaneously worsen certain aspects of the problem and often leave patients limited in quality of life. A thorough and detailed understanding of myocardial bioenergetics, plus a decades-long history of data – some from rigorously performed studies – now exists, providing strong support for the use of nutraceuticals in support of this all too common condition. In fact, for these reasons, coupled with the fact that such therapeutic agents have almost no side effects and rarely interact negatively with pharmaceutical prescriptions, HF can be considered an ideal candidate for this therapeutic approach. In addition, advanced lab tests (eg, NT-proBNP)[34] along with ejection fraction testing afford physicians the means necessary to properly and objectively monitor patients and ensure they are experiencing a positive response to the prescribed therapies. As highlighted in the case study featured in this review, an integrated approach can garner excellent results, even in relatively severe cases, improving myocardial bioenergetics, left ventricular function, and quality of life.

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Therapeutic Application of Carnitine Derivatives in Integrative Treatment of Cardiovascular Diseases

by Jeremy Mikolai, ND

The amino acid L-carnitine is a natural treatment of primary import in the integrative treatment of cardiovascular disease. Carnitine and its closely related therapeutic derivatives are underappreciated for their impact on the treatment of multiple organ systems and the dysfunction linking them, especially when that primary dysfunction is of vascular origin. When applied in the appropriate context, several carnitine derivatives provide the practitioner an opportunity to address primary cardiovascular complaints and concurrent issues simultaneously.

Carnitine derivatives can address cardiovascular complaints and concurrent issues simultaneously.

Carnitine is a general term used to refer to the amino acid L-carnitine (LC) and its closely related metabolic counterparts, acetyl-l-carnitine (ALC) and propionyl-l-carnitine (PLC). L-carnitine is a non-essential amino acid that participates in several metabolic processes. Participation of LC/ALC in shuttling of fatty acids into mitochondria for beta-oxidation is well known and largely understood, as is the activity of carnitine in inhibiting the conversion of thyroid hormone from less active thyroxine (T4), to the metabolic driver (T3), triiodothyronine, but its participation – and that of its derivatives – in other body processes is less completely understood.

None the less, the therapeutic applications for these agents in diverse pathologies of any age and gender cannot be overstated. Their efficacy has been demonstrated on important pathologies of the cardiovascular system, peripheral vascular system, nervous system, in the redress of infertility in both males and females, and in several conditions that are specific to the male urogenital organs. It is important to bear in mind that L-carnitine, in particular, has a moderate ability to slow thyroid hyperfunction and interfere with thyroid hormone supplementation. By no means is this a warrant to avoid use of carnitine where appropriate; however, thyroid levels will need to be monitored and possibly medication adjustments made until the carnitine dose is stable.

A randomized, controlled trial (RCT) of carnitine in post-heart attack patients demonstrated attenuation of left ventricular dilatation similar to the effect expected for angiotensin-converting enzyme inhibitors and beta blockers, two standards of conventional treatment, (the so-called ventricular remodeling effect).[1]

A significant mortality benefit was demonstrated in NYHA class II, IV heart failure patients with lower rates of death from all causes in patients on carnitine at a dose of 2 grams daily.[2] Other studies of carnitine in heart failure have shown significant improvements in exercise capacity, maximum exercise time, peak heart rate, peak oxygen consumption, and improved hemodynamic and echocardiographic parameters.[3,4]

In heart failure, we use L-carnitine at 1 to 4 grams/day, orally, in divided doses. L-carnitine inhibits peripheral thyroid hormone conversion (T4 to T3) and is therefore indicated in hyperthyroid conditions and relatively contraindicated in hypothyroid conditions. L-carnitine can cause an increase in seizure activity and is therefore contraindicated in seizure disorders. L-carnitine does have drug interactions with warfarin, acenocoumarol/Sintrom, and thyroid hormone.

Propionyl-l-carnitine (PLC) is a derivative of l-carnitine. As a manufactured agent it is most often delivered as glycine propionyl-l-carnitine (GPLC). Its therapeutic effect has several important applications in the vascular system, including in the treatment of peripheral vascular disease (PVD), healing of ulcers from PVD, improved walk distance in intermittent claudication, congestive heart failure, and angina.[5-9] Repeated studies show PLC supplementation increases circulating nitrate.[10] That does not prove increased vasodilation or conclusively elucidate one or more mechanisms of action for the therapeutic effect of PLC, yet its effect on diseases of ischemic pathophysiology are demonstrable.

Vasodilator activity may explain the role of PLC in the improvement of male sexual dysfunction, erectile dysfunction, and some symptoms of decreased sex hormone production with age ("andropause"), but that mechanism does not seem to explain all of the effects of the combination of PLC and ALC in the treatment of these disorders. All the same, oral supplementation of 2 grams/day of each of ALC and PLC has demonstrated profound effects on male sexual function and associated symptoms.

As it pertains to treatment of underlying causes, many of carnitine's effects may be the result of improved endothelial function. The endothelium covers the inner surface of blood vessels. It is the interface between the elements of the blood and the walls of blood vessels and the body's largest paracrine organ. Endothelial signaling is responsible for changing blood vessel sizes and pressures; it is responsible for both the formation and the breakdown of clots and platelet plugs. Inflammation, oxidized cholesterol, endothelial damage and repair, connective tissue degradation and new deposition, atherosclerosis and acute cardiovascular events all play their part in the function and dysfunction of endothelial function and dysfunction. In health, the function and ultimate homeostatic balance of the endothelium maintains appropriate wall tension and permeability of the blood vessels, maintains an anti-coagulant, anti-thrombotic, profibrinolytic milieu, which also inhibits immune cell adhesion and activation; and it maintains and promotes appropriate vascular remodeling.

Given its ability to increase circulating nitrate, GPLC should be a primary endothelial rehabilitator since the decrease in circulating nitrate levels is the sine qua non of endothelial dysfunction. While there is no extant research to date demonstrating direct effects of ALC or PLC on endothelial function, we can safely infer this action given that we know the parent compound affects endothelial function, we know PLC results in vasodilation, and we know ALC/PLC results in improved peripheral vascular function.

Acetyl-l-carnitine (ALC) is derived from the acetylation of carnitine and is used as a coenzyme A (CoA) donor in the mitochondria as acetyl CoA combines with carnitine to form ALC, which is transported out into the cytosol leaving CoA available for further beta-oxidation of fatty acids.

ALC has diverse and complex action upon the nervous system ranging from antiapoptotic effects to nerve regenerative properties, including increased neuronal structural element synthesis and increased growth factor sensitivity to analgesic action that may be attributable to a reduction of glutamate in the neuronal synapse.[11]

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well as in chronic neuropathies such as diabetic neuropathy. A 2017 review of 711 patients with diabetic neuropathy by Veronese et al demonstrated that ALC oral supplementation resulted in improved pain perception as well as improved motor and sensory nerve conduction velocities and amplitudes. In other words, ALC resulted in improved nerve function.[12]

Our understanding of the impact of ALC directly on the vascular system, endothelial dysfunction, and vascular disease is coming into greater focus. Metabolomic evidence has been established for the role of acylcarnitines in the pathogenesis of venous thromboembolic events (VTE) such as deep vein thrombosis, pulmonary embolism, and stroke. Acylcarnitines have an ability to bind to clotting factor Xa and thereby inhibit clotting. Evidence demonstrates that case-control groups with the lowest circulating acylcarnitine levels have the highest rates of VTE.[13] Within the integrative concepts of cardiovascular diseases and their underlying causes, we often refer to this as "vulnerable blood," blood that is prone to clot formation. It is yet another manifestation of endothelial dysfunction and dysfunctional vascular biology.

Dysfunction in vascular biology by any etiology is the underlying cause of cardiovascular disease. While L-carnitine is a treatment of primary importance in integrative treatment of cardiovascular disease, the availability of carnitine derivatives applied in particular cases may give us an opportunity to address several pathological manifestations simultaneously.

Case Study

VE is a 68-year-old male with an extensive medical history and medication list. He presented to me primarily for subclinical hypothyroidism in the setting of significant peripheral vascular disease with ulcerations and long-term wound care with poor healing, severe left ulnar neuropathy of unknown etiology, erectile dysfunction, mild hypertension and hyperlipidemia, a history of controlled type 2 diabetes, and insomnia.

I choose this patient case, not because there are stories of success or follow up to demonstrate, but because this patient is the epitome of one in which to consider therapeutic use of ALC/PLC over L-carnitine. Obviously, the evidence supports the use of these agents in several of his conditions. They offer an opportunity to avoid likely interactions with his thyroid medication as they are less prone to interfere than is L-carnitine. They are also less prone to interfere with anticoagulants than is L-carnitine, which can interfere with warfarin and acenocoumarol. While GPLC and ALC are not free from potential interactions with anticoagulants, this is not an interaction that this author has ever seen clinically and one of which no documented case reports or evidence has been found. Again, this potential interaction is of moderate severity because of the medications involved and potential sequelae but is a matter which is promptly solved with monitoring and appropriate medication and supplement adjustment and therefore, a helpful alternative strategy when wading through a quagmire of polypharmacy.

The true power of natural medicine is in its ability to address underlying causes of disease and therefore, to improve multiple, seeming disparate complaints simultaneously. I posit that the improvement of multiple conditions at the same time through the most judicious use of interventions is our best indicator of whether we are executing our most important jobs well: Do No Harm, Address the Underlying Cause, Work with the Healing Power of Nature.

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Broken Hearts

by Jacob Schor, ND, FABNO

With the many rapid, recent, and near miraculous medical advances in cardiac catherization, angioplasty, stent placement and ablation, it's easy to view the heart's pathology as either a matter of bad plumbing, or bad wiring. We forget the old understanding that our heart is the seat of our emotions and that things other than atherosclerotic plaque can injure our hearts.

We know this intuitively; just look at the words we use to express emotions. I miss a dear friend, and this causes me heartache. I feel love; my heart feels full. Sadness tears at my heart. Other organs do not lend themselves to emotional states. My stomach doesn't love anyone…though admittedly it may crave certain foods from time to time. My bladder may feel full and even insistent, but it is just urine and is not a measure of either happiness or sadness. Sitting next to a friend can fill my heart. Their departure can leave a hole in that same organ. Thinking of lost loves breaks my heart. And there are people, hopefully no one we know, people unlucky in love, who may die of a broken heart.

I'm pondering this after reading a recent research paper on Takotsubo syndrome by Christian Templin and colleagues published in March 2019 in the *European Heart Journal*.[1] In a collaboration between neuroscientists and cardiologists, functional MRI brain scans were run on people who had survived Takotsubo syndrome in the past, comparing them with scans from healthy controls.

Takotsubo syndrome is perhaps the most critical physical expression of this idea that emotions are felt in the heart phenomenon. Takotsubo is a life-threatening reaction to intensely strong emotions. When someone dies of a broken heart, this is not just a figure of speech; this is Takotsubo syndrome.

Takotsubo syndrome was first described in 1991 by Japanese doctors who reported five cases of transient multivessel coronary spasm. The disease manifests predominantly in postmenopausal females. "In about 75% of cases there are emotional triggers such as severe physical or emotional stress, natural disasters such as earthquakes, unexpected death of relatives, acute medical illnesses…." The common scenario is that the stress is a romantic breakup or the death of a spouse. The symptoms mimic a heart attack: chest pain, shortness of breath, even congestive heart failure. This association has earned Takotsubo the nickname, 'broken heart' syndrome.[2] The heart muscle balloons into a distinctive shape resembling, at least to the Japanese doctors who came up with a name, the clay pots used in Japan to trap octopus. Takotsubo pots have a wide base and long narrow necks; and apparently once the octopus enters, it can't figure out how to get back out of the pot.[3]

This syndrome usually resolves on its own within a few weeks, but the acute period can be severe enough to cause heart failure, arrhythmia, and death. People literally do die of a broken heart.

Coming back to Templin's functional MRI study, the researchers recruited fifty-four subjects to study. Of these, 15 had survived Takotsubo syndrome within the past year or so. (the median survival time was 378 days). The other 39 subjects were matched healthy controls. Let's skip the details of how these brain scientists tested the subjects' neurologic function. Put simply, the researchers defined four different sets of brain regions that allowed for analysis of how and to what extent different areas of the brain were simultaneously activated and communicating with each other. These networks were compared in the Takotsubo survivors and healthy controls.

In the healthy volunteers, the parts of the brain associated with the sympathetic and parasympathetic ➤

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nervous systems lit up at the same time as emotions. The communication among those areas was relatively slight in the Takotsubo survivors. Parasympathetic- and sympathetic-associated subnetworks both showed reduced resting state functional connectivity in Takotsubo survivors compared with controls. The dimmed neuronal activity was most notable between the brain regions that control the sympathetic and parasympathetic nervous systems; the physiological calming that should occur after stress was apparently less likely to take place. In other words, broken heart survivors had greater difficulty telling their hearts to calm down after being stimulated by intense emotions.

These results may bring us closer toward an explanation of how emotions effect health, in particular cardiovascular health; so, while I tend to yawn when the term 'functional MRI' comes up, I'm paying attention.

Takotsubo syndrome is perhaps the most critical physical expression of this idea that the heart feels emotional pain. Takotsubo is a life-threatening reaction to intensely strong emotions; when someone dies of a broken heart, this is not just a figure of speech, this is Takotsubo syndrome.

The etiology of Takotsubo syndrome is still unclear, but the most plausible explanation is that the sudden release of stress hormones, such as norepinephrine, epinephrine, and dopamine, stuns the heart. This triggers changes in the cardiac myocytes that hinder coronary perfusion. Stress hormone levels have been measured in Takotsubo patients that are double or triple the normal range.[4] Such elevated hormones cause left ventricular contractile dysfunction.[5] Because the beta-adrenergic receptors are the most concentrated in the apical region of the heart, that area is most affected and that part of the heart changes shape more. Many of the Takotsubo features can be triggered just by giving high doses of catecholamines and beta-adrenergic agonists.[6] Following this line of thinking, betablockers are used in managing this syndrome.

Estrogen provides protection to the heart[7]; and since more than 90% of Takotsubo patients are postmenopausal women, this suggests that estrogen may provide some protection.[8] Studies have shown that lack of estrogen replacement therapy may predispose women to developing Takotsubo syndrome.[9,10]

Templin et al's findings suggest that broken heart syndrome may actually begin in the brain, the result of an uncontained overreaction to emotions and an inability to dampen them. Whether it is the stress that changes the brain or whether the brains of people who develop this syndrome started out different, predisposed to handle stress poorly, is not clear. Earlier, somewhat simpler studies have also suggested that these survivors possess a weakened parasympathetic ability to moderate the stress brought on by intense emotions.

Back in 2016, writing in the *American Journal of Cardiology,* Norcliffe-Kaufmann et al reported they had characterized autonomic function in 10 women with a history of Takotsubo comparing them with an equal number of healthy controls. These researchers used more traditional assessment tools including baroreflex stimulation (Valsalva maneuver and tilt testing), cognitive stimulation (Stroop test), and emotional stimulation (event recall, patients). Ambulatory blood pressure monitoring and measurement of brachial artery flow-mediated vasodilation were also performed. Their testing was performed an average of 37 months after the Takotsubo event. Even three years later, these Takotsubo survivors had excessive sympathetic responsiveness and reduced parasympathetic modulation of heart rate.[11]

A second paper published in 2016 used functional MRIs to monitor four Takotsubo survivors comparing them with eight healthy volunteers while they underwent various autonomic challenges. Similar conclusions were reached: "The central autonomic response to autonomic challenges is altered in patients with Takotsubo cardiomyopathy, thus suggesting a dysregulation of the central autonomic nervous system network…. [It is unclear] whether these alterations are causal or predisposing factors to Takotsubo cardiomyopathy."[12]

Lazzeroni et al reported similar findings in 2017 after comparing autonomic function in 24 Takotsubo patients, 25 healthy subjects and 22 post-MI patients. Once again, Takotsubo survivors "…showed a blunted parasympathetic reactivation after exercise, similar to that observed in post-MI patients, thereby suggesting that vagal control of heart rate after exercise is abnormal long after the acute presentation of TS [Takotsubo syndrome]." Thus, although "… exaggerated sympathetic stimulation plays a role in the development of Takotsubo syndrome (TS) during the acute phase," parasympathetic function continues to be weak long after the episode.[13]

The question remains. Is this weakened parasympathetic response the result of the Takotsubo episode or is it causal? Was it there prior to the episode? Does a weakened parasympathetic system leave these patients vulnerable to heart break? Or, did the heart episode cause the problem in function? Is the parasympathetic weakness we find just collateral damage from the experience?

This raises somewhat parallel questions about depression and cardiac disease.

Symptoms of depression are about three times more common in patients after an acute heart attack than in the general population, which strongly suggests a link between depression and heart disease.[14]

May et al for their 2017 report, followed 24,137 patients with coronary artery disease (CAD). At follow-up, 3,646 of their group (15%) were diagnosed as depressed. During the next decade about 40% of the larger cohort died. In those diagnosed with depression, 50% had died while only 38% of those not depressed had died. A depression diagnosis at any time following CAD diagnosis was associated with a two-fold

higher risk of death. Being depressed was the strongest predictor of death in those diagnosed with CAD, doubling risk of death.[15]

A study by Liu et al, published in April 2019, examined data from 32, 345 people in the US and evaluated how depression and anxiety affect risk of coronary artery disease (CAD). Here too, depression or anxiety were associated with double the risk of developing CAD. Treating and reducing the severity of either psychological disorder was associated with lower risk of CAD.[16]

There are hints that depressed cardiovascular patients differ deeply from those that aren't depressed. Williams et al reported in March 2019 that, "Depressed cardiovascular patients had higher serotonin receptor density, and significantly higher incidence of major and minor cardiac adverse events" than non-depressed cardiac patients. Something appears to shift in the very biology of these patients.[17] This may be why treating these depressed patients with serotonin reuptake inhibitors (SSRIs) is more effective at reducing major cardiovascular events than antidepressants that do not target serotonin pathways.[18]

Depressed cardiac patients may also look and feel depressed for another reason. "Patients with depressive symptoms directly after MI have a flattened diurnal serum cortisol profile. This is particularly expressed in patients with longer lasting symptoms."[19] It is a challenge even for an experienced practitioner to differentiate flattened cortisol from frank depression. Few cardiologists will probably even think to make the distinction between the two conditions.

These associations between cardiac disease and depression beg the question: Will treating the depression improve the cardiac disease prognosis? Rahmani et al reported in 2018 that it might help. They report that taking part in cardiac rehabilitation programs following coronary angiography is associated with less depression. Patients who do not take part in rehab are nearly 11 times more likely to be depressed.[20] One might reasonably argue, though, that the more depressed someone is, the less likely they will be to get around to attending these sessions.

There's another new paper suggesting that the post-cardiac disease depression is deep in the physiology. Bremner et al reported that there are specific changes in brain function in depressed cardiac patients. Depressed CAD patients had "…increased parietal cortex activation and a relative failure of medial prefrontal/anterior cingulate activation during mental stress compared to CAD patients without depression." The authors of this study consider their findings, "…consistent with a role for brain areas implicated in stress and depression in the mechanism of increased risk for CAD morbidity and mortality in CAD patients with the diagnosis of major depression."[21] In other words, they are thinking depression may lead to CAD.

Takotsubo syndrome is perhaps the most critical physical expression of this idea that emotions are felt in the heart.

A year earlier in 2018, the same authors, Bremner et al, describe a situation reminiscent of Takotsubo: "… mental stress-induced myocardial ischemia is associated with activation in brain areas involved in the stress response and autonomic regulation of the cardiovascular system. Altered brain reactivity to stress could possibly represent a mechanism through which stress leads to increased risk of CAD-related morbidity and mortality."[22]

A decade or so back this association between stress and heart disease was used to justify interventions that hoped to increase resilience in patients and so reduce the impact of heart disease. Dimsdale wrote in 2008, "There is nonetheless overwhelming evidence both for the deleterious effects of stress on the heart and for the fact that vulnerability and resilience factors play a role in amplifying or dampening those effects. Numerous approaches are available for stress management that can decrease patients' suffering and enhance their quality of life."[23]

Sadly, these attempts at stress management and other psychological interventions have not proven to be terribly fruitful in preventing heart disease. Machado et al, in an umbrella study published in 2018, (An umbrella study in case you wondered, is apparently the term used to describe a meta-analysis of prior meta-analyses) wrote: "A causal effect of depression on all-cause and cause-specific mortality remains unproven, and thus interventions targeting depression are not expected to result in lower mortality rates at least based on current evidence from observational studies."[24]

A 2017 Cochrane review was even less supportive of this approach of treating depression to reduce impact of CAD. The reviewers reported that for people with CHD, there was no evidence that psychological treatments had an effect on total mortality, the risk of revascularization procedures, or on the rate of non-fatal MI. However, there was some positive news: the rate of cardiac mortality was reduced and psychological symptoms (depression, anxiety, or stress) were alleviated.[25]

There is one additional 2016 study on Takotsubo patients that is worth mentioning. Marfella and colleagues in Naples treated 48 people diagnosed in their hospital with Takotsubo by giving them 600 mg per day of alpha lipoic acid or placebo after they were discharged. The patients were followed for 12 months. As in other studies, changes in heart function persisted long after the initial episode. However, treatment with lipoic acid improved the adrenergic cardiac innervation.[26] If lipoic acid helps prevent heart damage in Takotsubo, might it also ameliorate depression in CAD patients? This supplement is already suggested for treating CVD[27] and for depression in general.[28]

A trial published in early 2020 compared two groups of heart patients:

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68 who had experienced Takotsubo syndrome with 68 who had suffered myocardial infarction, assessing disease-related quality of life, depression, anxiety, chronic stress, social support, resilience, and life events prior to disease onset. Both groups displayed higher scores in depression and anxiety, higher levels of chronic stress, and lower scores in physical and mental quality of life in comparison to normal people, though no differences were seen between the two patient groups. Within both groups, the majority of patients (Takotsubo: 69.1%; MI: 60.3%) reported stressful life events prior to disease onset. In both patient groups, the number of events had a significant impact on long-term mental health and chronic stress.[29] The bottom line seems to be that both diseases have a lasting negative impact on mental health.

Depression in cardiac patients is common and a bad sign to see. This is one of those chicken and egg situations. Which came first? Does injury to the heart release chemicals that make people feel depressed, or does depression release chemicals in the body that promote heart injury? Or is it a two-way street that the poor chicken is trying to cross? At this point the science is unclear so we might as well look both ways. If we treat depression and improve heart function, it's not the end of the world. And if we treat cardiac disease and accidentally cheer people up, once again, it's not the end of the world. Though if you talk to people with heart disease, some of them will act as if it is the end of the world. Those are the depressed ones, those we need to worry about the most.

Jacob Schor is a 1991 graduate of National College of Naturopathic Medicine in Portland, Oregon, and practiced in Denver, Colorado, until June 2019. He also has a BS degree in food science from Cornell University in Ithaca, New York. This earlier training in food chemistry and nutrition has influenced and informed his approach to naturopathic medicine throughout his career as a naturopathic doctor. He is a past president of the Colorado Association of Naturopathic Physicians and the Oncology Association of Naturopathic Physicians and also served on the board of directors of both associations as well as the American Association of Naturopathic Physicians. He is an associate editor for the Natural Medicine Journal and a regular contributor to the *Townsend Letter*.

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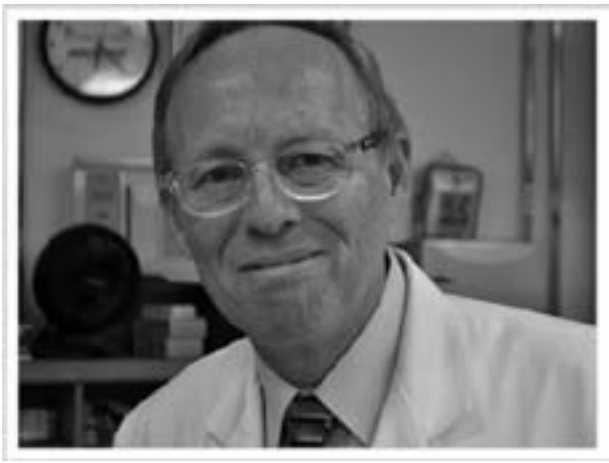

Effectiveness of Hyperbaric Oxygen Therapy for Traumatic Brain Injury

Interview of Paul G. Harch, MD

by Nancy Faass, MSW, MPH

In the treatment of traumatic brain injury, the research suggests that hyperbaric oxygen is the most effective therapy currently available at all levels of severity and all time points in the disease process. Mortality from acute, severe traumatic brain injury (TBI) can be reduced with just a few treatments, early on, documented in randomized clinical trials. One to three sessions of hyperbaric oxygen therapy (HBOT) in the acute phase can decrease mortality by 50% to 60%, the greatest reduction in mortality of any known therapy. There is also evidence that HBOT is therapeutic for acute concussion, as well as post-concussion symptoms. HBOT is the only therapy that directly treats the underlying disease processes in TBI wounds: decreased oxygen, decreased blood flow, swelling, constriction of blood vessels, acidosis, and anaerobic metabolism.

Significance. In the US, accidents are now the leading cause of death among every age cohort from childhood through age 44. Among the survivors, the official number of brain injuries is approximately two million annually in the US and ten million worldwide. This means that more than two million brain-injured individuals are added to our population every year. Over 75% of these brain injuries are concussions. Approximately half of all mild TBI patients develop persistent post-concussion syndrome, characterized by cognitive symptoms such as memory loss, headaches, mood swings (irritability, anxiety, depression, apathy), sleep disruption, fatigue, dizziness, and changes in personality.

In terms of potential therapeutics, two randomized trials have been conducted showing significant improvement in moderate to severe TBI in the subacute phase utilizing HBOT. There are multiple randomized trials on mild TBI in the chronic phase, showing the therapeutic benefit of HBOT.

History. Hyperbaric oxygen therapy has been described as a treatment for wounds in any location in the body and of any duration. Initially discovered in England in 1662, hyperbaric therapy was developed in its present form by the US Navy to treat the bends. In the 1950s, surgeons in the Netherlands innovated the use of high-pressure oxygen in the treatment of infections, cases of carbon monoxide poisoning, and during surgery. Today, we are in a new era. In terms of concussion, for example, persistent post-concussion syndrome is no longer considered a psychiatric disease. We now know that it is an organic injury, essentially a wound to the brain. Scientifically, TBI treatment is a logical application of hyperbaric therapy, which is widely acknowledged as an effective intervention for difficult-to-heal wounds. With emerging clinical trials, we have the evidence to confirm the specific effectiveness of HBOT for traumatic brain injury.

*Mechanisms of action***.** Scientific studies over the last 10 years have shown that one of the primary targets of hyperbaric therapy is the DNA. Based on molecular biochemical techniques, which became available in 2008, researchers were able to perform mass gene array analysis to determine which genes were upregulated or

downregulated by changes in pressure and oxygen. Research has found that a single hyperbaric treatment affects more than 40% of the protein-coding genes in our DNA – 8,101 of the 19,000 protein coding genes. Different pressures and different oxygen levels have independent, overlapping, and interactive effects on different clusters of genes. Those effects occur through oxygen and pressure signaling. Understanding these effects, the applications of this therapy can be extended to a wide array of health issues, including genetic syndromes that are not considered wound conditions.

Treatment

Hyperbaric oxygen therapy involves the use of increased atmospheric pressure and elevated levels of oxygen as medications to treat pathophysiology. Therapeutic effects occur through genetic expression and suppression of growth and repair hormone and anti-inflammatory genes, and by improving blood flow and metabolism. These effects are a function of dose and timing of the intervention in the disease process. HBOT doses of 203-304 kPa (kilopascals, equivalent to 29-44 pounds per square inch) are utilized for wound healing and to treat infection. Lesser doses have been used primarily for chronic neurological conditions.

Harch Protocols. Over the last 34 years, I have developed algorithmic approaches and flexible protocols to assess and treat different medical conditions with HBOT. One methodology for brain-based neurological conditions involves the following protocol:

- Pretreatment screening by phone
- Day 1. History, physical exam, and SPECT brain imaging to establish baseline
- Day 2. First HBOT session at a specific dose, followed in 3-4 hours by a second SPECT imaging
- Days 3 on: 39 additional daily HBOT sessions at that specific dose, five days per week, for eight weeks
- Periodic evaluation to check progress, response, and dosage

SPECT scans. We began doing SPECT scans in 1989. The protocol for SPECT scanning described above evolved from Dr. Richard A. Neubauer's use of SPECT imaging before and after a first HBOT in a stroke patient in the late 1980s. We adapted this to the first-ever use in divers with residual brain injury from decompression illness and in boxers, and over time, treated patients with more than 80 different neurological conditions. What we typically would see on the second scan after the very first HBOT was an improvement in overall brain circulation (blood flow), and an improvement in the pattern of blood flow, which becomes more normalized. (Note that the SPECT scan provides images of circulation or lack thereof that is a snapshot of brain blood flow at the time the SPECT dye is injected. In contrast, a PET scan documents activity involving glucose metabolism.)

Oxygen toxicity. The most extreme form of oxygen toxicity is a grand mal seizure. Although this is generally rare, I strongly recommend that HBOT is delivered by and under the supervision of a physician knowledgeable regarding HBOT. Lin, Tsai, & Lee (2008), treating moderate to severe TBI at 203 kPa, reported a 9% incidence of grand mal seizures. However, accumulating data and experience have demonstrated that toxicity can occur at lower pressures of oxygen. In 2018 four patients were reported to the FDA as part of a traumatic brain injury study who developed signs of oxygen sensitivity/overdosing with less than 40 treatments at 150 kPa. Children with seizure disorders can exhibit oxygen toxicity at far lower levels and with lesser numbers of treatments. In my own practice, when treating patients with a history of seizure disorder, I start at lower pressures and observe the patient carefully, adjusting the dose based on their response.

It is possible that hyperbaric oxygen therapy can worsen existing conditions. Having documented these cases over the course of 30 years in practice, my observation is that too much oxygen can result in deterioration. In the majority of these cases (both in the US and overseas), the treating facilities had no physicians involved and the technicians were operating under the precept that hyperbaric oxygen therapy could only improve patients, so negative effects were puzzling, often ignored, or commonly blamed on the patient. (I reported these results at the 2001 Symposium on Hyperbaric Oxygenation and the Brain Injured Child. The Proceedings are available from Best Publishing Company.)

Mild adverse effects. In terms of mild, temporary symptoms, in our study of mild TBI and PTSD (2017), six of thirty participants experienced mild, reversible middle-ear barotrauma (five of these patients had mild upper ➤

Table 1. Symptoms reported for 30 military personnel in study (Harch *et al*., 2017) and improvement after received HBOT for mild TBI.

Symptoms in Order of Prevalence	Percentage of Subjects' Symptoms better after 40 HBOT Treatments	Percentage of Subjects' Symptoms better at 6-Month Follow-up
Headaches	93%	86%
Depression	92%	87%
Cognition (100% of participants reported cognitive symptoms)	90%	96%
Short temper	90%	95%
Low energy	86%	93%
Mood swings	84%	96%
Short-term memory loss	83%	91%
Speech problems	78%	87%
Sleep disruption	73%	80%
Poor balance	65%	88%
Tinnitus	47%	56%
Hearing loss	10%	22%

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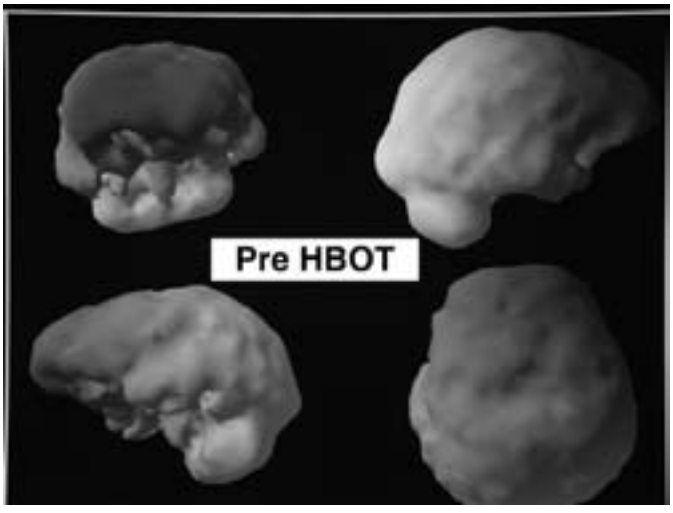
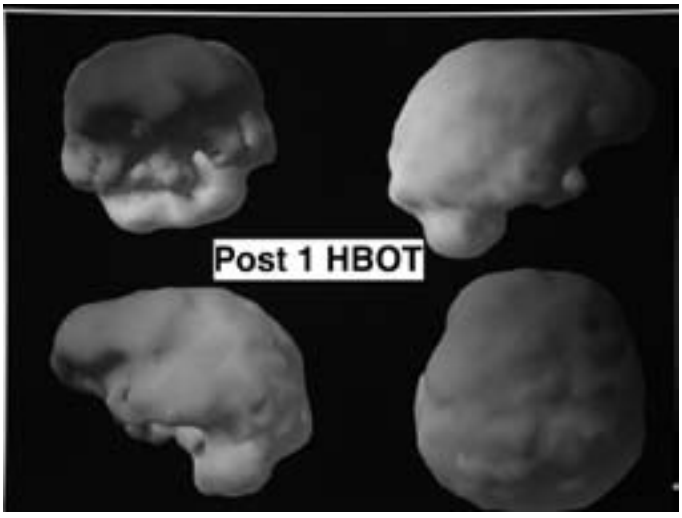
respiratory infections at the beginning of the study). Other patients, seven of the total 30 in the study, had a transient deterioration or re-expression of pre-existing symptoms approximately halfway through the 40-treatment course. These resolved over the next four to six HBOT treatments. In general practice, mild symptoms most likely to occur involve clearing pressure in the ears, or sometimes the sinuses.

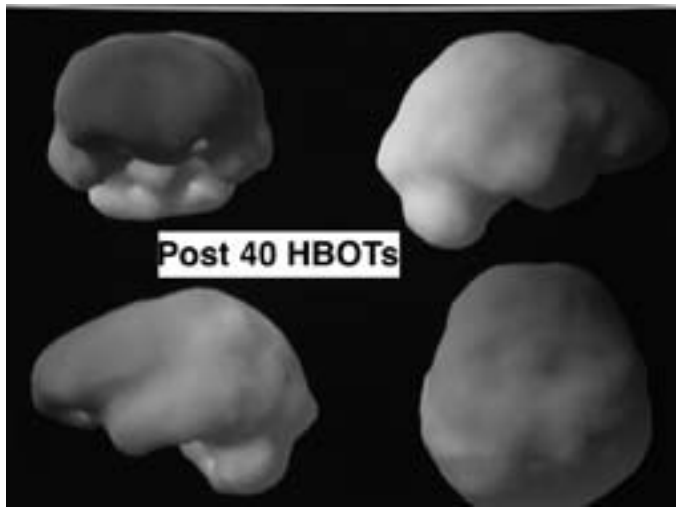
Therapeutic Effects Achieved with Delayed Treatment for TBI. In our previously mentioned study involving 30 military personnel with mild TBI, 90% reported improvement in cognitive symptoms after 40 HBOTs, and that improvement increased at the six-month follow-up. The veterans' post-concussion symptom scores (headaches, neurological, cognitive, vestibular, and emotional symptoms) decreased by 36%. (See Table 1.) Wechsler IQ testing was administered and showed on average a gain in IQ points of 14.2 points, which is exceptional. Evaluation of anxiety using the GAD-7 showed an average decrease of 5.4 points on a scale of 21. Similar improvements were documented in depression with an average 7.9-point decrease on a scale of 27 using the PHQ-9. Reductions in PTSD symptoms averaged 16.6 points on the 80-point Military PTSD Checklist. This represents the greatest improvement in PTSD symptoms in the shortest period of time for any clinical study on PTSD. Overall, there was a self-reported 95% improvement in emotional control. All findings had a *P*-value of 0.001 or better.

Case Study: Delayed Treatment of Traumatic Brain Injury

Rusty had been in the military and was back in civilian life, a family man, serving as a law enforcement officer, and running a small business in the Midwest, when 9/11 occurred. He was so outraged that he reenlisted and found himself in Iraq, age 45, escorting translators around the Green Zone. When his area was hit with more than 300 rounds of enemy mortar, he was knocked unconscious. He started having headaches and went to the sick bay, but no one there realized that he had been in an explosion. He continued to suffer from debilitating headaches, vertigo, short-term memory loss, and constant fatigue, was medically boarded out of the military, and diagnosed with a psychiatric disorder. One year later, a military caseworker happened to note his symptoms and called me. When I screened him over the phone and took a detailed history, I realized that he had a brain injury, so I entered him in our study, and he came to Louisiana. One aspect of the study was brain imaging







Figures 1-3: Four-view three-dimensional surface reconstructions of SPECT brain imaging of 45-year-old veteran with blast-induced persistent post-concussion syndrome, before, after 1 HBOT, and after 40 HBOTs. Figure 1. Imaging pre-HBOT shows deficits in brain blood flow that are registered as scalloped indentations on the surface of the brain. Note the asymmetric injury to the left side of the brain (in the left upper quadrant scan, the area of the brain on the right side of the image), the side from which the mortar blast occurred. The left frontal, temporal, and cerebellar lobes are severely affected. Figure 2. After the first HBOT intervention there is diffuse improvement in the brain blood flow, especially the most damaged left temporal, frontal, and cerebellar lobes. After 40 HBOTs, Figure 3, there is a near normalization of brain blood flow compared to the pre-HBOT study. Simultaneously, the veteran experienced improvement in his symptoms and cognition.

and the resulting images were dramatic, in fact, the most exceptional of any of the 30 participants in the study. The entire left side of his brain showed a reduction in brain-blood flow (the side on which the blast had occurred). He also complained about intense itching and periodic blistering on his left arm. We subsequently X-rayed his arm, which showed 16 pieces of embedded shrapnel. Here was the evidence that he had experienced a concussive blast injury. Over the course of treatment, his symptoms and imaging improved. He was eventually awarded a Purple Heart and now serves as liaison to the North Dakota State Legislature on matters related to veterans.

Achieving More Precise Diagnosis and Treatment with EEG

For much of my career, I have been searching for an effective means of pinpointing dose-response for the individual patient and for their specific disease process. Dosage was guided by cumulative case experience, my research, the research of others, and the use of pre- and post-treatment SPECT scans. However, we were unable to see what was occurring in the brain during actual treatment, until now. In the past four years, I have adapted our hyperbaric chambers to perform real-time qEEG in the chamber to monitor a patient's response to hyperbaric therapy. This process is patent pending.

Paul G. Harch, MD

Indications for Hyperbaric Therapy

I have two practices: one is an academic medical practice in which we treat reimbursable conditions, both outpatient and inpatient. At the present time, 13 indications are approved for the marketing of hyperbaric chambers by the FDA, and 15 are typically reimbursed. (The typically reimbursed indications appear in Table 2 identified by an asterisk.) The second practice is a private practice where I treat off-label indications, which for the most part are not reimbursable. It becomes a matter of discretionary income in order for ➤

Table 2. Indications for Hyperbaric Therapy (*typically reimbursable conditions)

Neurological Conditions
*Air or Gas Embolism
Anoxic Brain injury
Autism
Birth Injury
Brain Aneurysm, Post-Rupture
Brain Injury, Acquired (ABI) (lack of oxygen, exposure to toxins, pressure of a tumor)
Brain Injury caused by Substance Abuse
Brain Injury, Chronic Traumatic Encephalopathy Brain Injury from *Chemotherapy ("Chemo Brain")
Brain Injury from Radiation
Brain Injury¸ Traumatic
Brain Injury-Related Depression
Brain Insult, Concussion
Brain Insult, Post-Concussion Syndrome
*Central Retinal Artery Occlusion
Cerebral Palsy
Dementia
Dementia, Early (Mild Cognitive Impairment)
Drowning
Encephalopathy, Hypoxic Ischemic
Fetal Alcohol Syndrome
Hypoxia (Near-hanging)
*Intracranial Abscess
Migraine Headache
Post-Anesthesia Brain Injury
Traumatic Brain Injury
Traumatic Brain Injury with Post-Traumatic Stress Disorder
Shaken Baby Syndrome
Stroke

Toxic Exposure
*Carbon Monoxide Poisoning, Acute and
Acute Carbon Monoxide Poisoning complicated by Cyanide Poisoning
Carbon Monoxide Poisoning, Chronic

Injuries and Wound Healing
*Compartment Syndrome/Crush Injury/Other
*Delayed Radiation Injury (soft tissue and bony necrosis)
*Diabetic and Selected Problem Wounds
*Exceptional Blood Loss Anemia
*Skin Grafts and Flaps, Compromised
Spinal Cord Compression Injury
*Thermal Burns
*Traumatic Ischemias

Pain Conditions
Pain Syndrome, Complex Regional
Pain Syndrome, Fibromyalgia
Peripheral Neuropathy

Rehabilitation
Arthritis
*Decompression Sickness, Acute (The Bends)
Decompression Sickness, Chronic
Multiple Sclerosis
Recovery from Surgery
Sports Injuries and Sports Performance Recovery

Infectious Processes
*Actinomycosis
Chronic Infection
*Gas Gangrene
Lyme Disease
*Necrotizing Soft Tissue Infection
*Osteomyelitis (refractory)

Autoimmune Disorders
Chronic Fatigue Syndrome
Inflammatory Conditions

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patients to obtain this type of therapy. The additional problem is that the number of people who understand this therapy is relatively limited. Hospital facilities that have a chamber and have trained staff will not treat off-label indications such as neurological disorders because these conditions are not reimbursed at this high level. In addition, there have been threats in the past of disciplinary action by one of the medical societies against their members should they treat off-label conditions "for profit." This has inhibited many hyperbaric physicians from offering HBOT for off-label conditions in the hospital setting.

HBOT treats decreased oxygen, decreased blood flow, swelling, constriction of blood vessels, acidosis, and anaerobic metabolism.

Training Resources

One-year fellowship. These fellowships are currently offered at eight institutions across the US, which teach the treatment of 13-15 indications that have FDA-approval for marketing and third party reimbursement. Louisiana State University has the largest fellowship program in the country. Fellowship programs are also offered at Duke, State University in New York (SUNY), U.C. San Diego, and other universities. The problem is that no training is provided for off-label indications, which means that there is no training for the vast majority of neurological conditions that can be ameliorated with hyperbaric treatment. Additional information is available at acgme.org, using the search term "hyperbaric medicine fellowship."

Forty-hour CME training. There is a forty-hour continuing medical education introductory course provided nationwide, available online, and in other cases by physical attendance. The forty-hour course enables providers to bill Medicare, Medicaid, and insurance companies for hyperbaric therapy for reimbursable indications. This is essentially an entry point to the field. For an example of these CME offerings, see uhms.org/education/courses-meeting/introductory-courses.html.

Upcoming trainings. In the next year, we anticipate offering additional training in hyperbaric therapy via an educational platform. For updates on this training, please check HBOT.com periodically.

Annual meetings. Hyperbaric Medicine International (HMI) provides annual meetings focused on the science and applications, with information on dosing. See hyperbaricmedicineinternational.org.

Professional Organizations

Hyperbaric Medicine International (HMI). I am one of the founders of this association, which was originally called the International Hyperbaric Medical Society (IHMA, 2001), a nonprofit that supports research, education, and treatment. The website of HMI provides a wealth of information and resources for both medical professionals and patients. For more information, see hyperbaricmedicineinternational.org.

American College of Hyperbaric Medicine. The hyperbaric society originally founded by Dr. Richard Neubauer and colleagues in 1983 and now re-incorporated by another group, this society focuses primarily on reimbursable applications in wound care, certification, regulatory issues, practice protocols, quality assurance, and Medicare. See hyperbaricmedicine.org.

The Undersea and Hyperbaric Medical Society. The first of the hyperbaric medical societies, UHMS (uhms.org) holds national and regional meetings. The society established the majority of the current reimbursable diagnoses and continues to focus on the treatment of those 15 conditions.

Referring to Providers of HBOT

If you have patients with neurological disorders, and you wish to refer them within your metropolitan area for treatment, you must perform due diligence. Search for facilities in your area on the internet and then screen providers by phone and in person, just as you would any provider in an emerging field to whom you want to refer vulnerable patients. With hyperbaric therapy, the range of training, experience, and quality of care is so broad, this personal research is essential. Currently HBOT is provided by practitioners whose background varies from no training to 40 hours of CME to 20 or more years of training and experience. In other cases, HBOT may simply be provided as a business, by a technician who receives a prescription signed by a physician (the MD may be knowledgeable or not). This range in training and experience within the field explains why the outcomes are so varied.

These varied outcomes also characterize recent studies by the Dept. of Defense using HBOT, which were performed with what were judged to be highly sophisticated study designs. These designs instead revealed a fundamental misunderstanding of hyperbaric oxygen therapy, which is that HBOT is a dual-component drug composed of increased pressure and increased oxygen. Those patients in the control group received low-doses of hyperbaric therapy – low pressures and low doses of oxygen labeled as sham treatment. In fact, they turned out to be effective doses of hyperbaric therapy. Consequently, rather than comparing HBOT with sham therapy, the clinical trial inadvertently compared HBOT at different dosages. Unfortunately, the mixed outcomes from these federally sponsored studies have tended to prejudice other institutional healthcare providers.

The majority of hospitals in the US have SPECT brain scan capability but rarely perform this type of brain imaging. My recommendation is that you find a facility that frequently performs SPECT brain scanning and insist on a dedicated nuclear technologist and

radiologist to perform and interpret all of your patients' scans in order to minimize variability in technique and interpretation. Most radiologists are not familiar with or accustomed to seeing the type of changes evident on SPECT after HBOT. (More detailed suggestions are offered on numerous aspects of HBOT protocol in my book, *The Oxygen Revolution*, 3rd edition.)

Information Resources

Website. The website of Harch Hyperbarics features extensive patient videos, taken at various points in treatment, which provide an encouraging and realistic sense of what can be achieved in cases of traumatic brain injury. News articles and informational resources are also available on the site at HBOT.com.

Online. Interviews of a number of Harch Hyperbarics patients are also available on YouTube.com. Additional videos about HBOT on YouTube include Joe Namath speaking on hard-chamber treatment for post-concussion symptoms (years post-injury) and LeBron James describing how he uses soft-chamber HBOT for training and recovery.

Journal articles. My randomized study on use of hyperbaric therapy for the treatment of traumatic brain injury will be published in March/April in a peer-reviewed instant-access online journal. The journal will be announced at that time. The article will be easily accessed through any search engine using "Harch, traumatic brain injury, hyperbaric oxygen therapy." A number of journal articles are available for download in free full-text versions on PubMed, including our study of 30 military personnel with TBI, published in 2017.

Book. Paul Harch, MD, and Virginia McCullough. *The Oxygen Revolution, 3rd edition.* Hobart, NY: Hatherleigh Press, 2016, 310 pp. Written for both providers and patients, the book contains information and resources, with insight into numerous aspects of treatment, reimbursement, and healthcare policy. More than 100 pages are devoted to the treatment of TBI, birth injuries, strokes, autism, Alzheimer's, and alcohol abuse, as well as diabetes, bone and joint disorders, AIDS, and antiaging therapies.

Textbook. Kewal K. Jain, editor. *Textbook of Hyperbaric Medicine, 6th edition*. New York, NY: Springer; 2016.

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Paul G. Harch, MD

Paul G. Harch, MD is an emergency medicine and hyperbaric medicine clinician, clinical professor of medicine at Louisiana State University Health Sciences Center, New Orleans, and former director of the Hyperbaric Medicine Department and Hyperbaric Medicine Fellowship at Louisiana State University School of Medicine, New Orleans. He is a graduate of the University of California, Irvine (magna cum laude/Phi Beta Kappa), and Johns Hopkins University School of Medicine. Dr. Harch initiated and maintains both an academic and a private practice that have resulted in the largest case experience in neurological hyperbaric medicine in the world. In his private practice he has adapted the concepts of conventional hyperbaric oxygen therapy to disorders of the central nervous system. Working initially with brain-injured divers and boxers he treated the first case of chronic traumatic encephalopathy (CTE) in 1989 (boxer) and dementia (diver) then adapted this protocol to the treatment of traumatic brain injury in 1991, cerebral palsy in 1992, autism in 1995, and numerous other cerebral disorders. He has also seen the positive effects of HBOT firsthand through the treatment of patients with toxic brain injury, stroke, dementia, and learning disabilities. Dr. Harch has successfully treated U.S. servicemen with TBI and PTSD, and his clinical studies for brain-injured veterans have continued with a recently completed randomized trial funded by a Louisiana-generated congressional appropriation. He has also presented his clinical experience and research four times to the U.S. Congress and has been a semifinalist for the NIH Director's Pioneer Award. A founder and the first president of Hyperbaric Medicine International, Dr. Harch is active in conferences and training. His book, *The Oxygen Revolution, 3rd edition,* (2016) co-authored with Virginia McCullough, explains HBOT as an epigenetic therapy with potentially widespread application in medicine and neurology.

Dr. Harch's work focuses on the development of HBOT treatment protocols for neurological disorders, based on the needs of each specific patient, their disease process, and the patient's response to HBOT. SPECT scans and qEEG-directed dosing facilitate additional precision, but are optional. His patients are from all over the world, and all are treated at his center in New Orleans.

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Hashimoto's Thyroiditis, A Common Disorder in Women: How to Treat It – Part 2

by Thierry Hertoghe, MD

Editor Note: In Part 1 of this article (*Townsend Letter,* April 2020), Dr. Hertoghe discussed diagnosis, associated pathologies, and underlying causes of Hashimoto's thyroiditis. He also presented details about dietary measures that can reduce antithyroid antibody levels. Part 2 continues with more treatments for underlying causes.

Causes to Treat: Nutritional Deficiencies

Nutritional therapies to treat nutritional deficiencies may decrease thyroid antibody levels by 20-50%.

Selenium and vitamin D supplementations: Patients with Hashimoto's thyroiditis have been reported to have significantly lower selenium[315-316] and vitamin D[317-339] levels than controls without the disorder. On the other hand, daily selenium supplementation can decrease the levels of thyroid antibodies by 20 to 30%,[8,340-344] particularly if the level is low-normal or below the normal limit. The same is valid for vitamin D supplements.[345-354]

What are adequate doses? In adults, the dosage of selenium supplements should be at least 200 µg a day for several months and higher in the case of a noted deficiency in this trace element. Concomitant intake of 600 mg a day of myo-inositol has shown to increase the efficacy of selenium supplements to further reduce thyroid antibody levels.[8,343-344] Efficient doses of vitamin D3 supplements are 4000 IU a day at minimum, but higher doses between 10,000 IU (lean persons) to 20,000 IU a day (obese persons and intensive sports practitioners) may be more effective.

Iodine supplementation may be crucial, as about one-quarter of studies have shown that low iodine levels, especially within the thyroid gland,[355-356] have been associated with higher levels of antithyroid autoimmune antibodies.[355-360] At least one study has shown iodine supplementation to reduce the levels of antithyroid antibodies.[361] However, the majority of epidemiological studies have reported that high iodine intake increases the risk of autoimmune thyroiditis in iodine-deficient areas.[362-370] For this reason, if iodine is given alone, it may be better to limit the dose to 200 µg a day, as a study showed that in thyroxine-treated patients the production of thyroid antibodies was not induced at that dose.[371] If higher doses of iodine are requested, it should be administered along with selenium. Selenium appears to reduce markedly the risk of inducing autoimmune thyroiditis and other toxic effects on the thyroid with excessive iodine intake.[372-375]

Omega-3 polyunsaturated fatty acid supplements: Adding 2 g per day of fish oil for patients who don't regularly eat fatty fishes might help reduce autoimmune antibodies, just as fatty food consumption does. Daily omega-3 supplementation has been shown to prevent and reduce the autoimmune antibody production in mice with autoimmune lupus[376-377] and glomerulonephritis.[380] In vitro studies of dendritic cells show that adding docosahexaenoic acid, one of the major omega-3 fatty acids of fish oil, prevents development of experimental autoimmune encephalomyelitis (multiple sclerosis), another autoimmune disorder.[379]

Thus, after confirmation that nutritional levels are low, low-normal, or even average in laboratory tests (but not if these levels are high-normal or

Table 4: Nutritional treatments that reduce antithyroid antibody levels

Treatment	Dose First 6 months	Dose Next 6-18 months
• Selenium	2×200 µg/day of selenium methionine	200 µg per day
with Myo-inositol	600 mg/day	600 mg/day
• Vitamin D3	10,000 IU/day	6000 IU
• Iodine	200 µg/day	200 µg/day of iodine
	Or with selenium: 1-3 drops of Lugol 5% solution (6.25-9.75 mg of iodine/iodide)	Or with selenium: 1-3 drops of Lugol 5%
• Omega-3 fatty acids	2 g/day of fish oil	1-2 g/day of fish oil

above the upper limit), I suggest the intake of higher amounts of the above-mentioned nutrients the first four to six months. Table 4 gives an overview of the dosages of the nutritional treatments that can efficiently calm down the autoimmune thyroiditis.

Cause to Treat: Hormone Deficiencies

Hormone treatments can decrease thyroid antibody levels by 20-70%.

Thyroid treatment: Thyroid therapy is necessary not only to reduce thyroid antibodies[380-394] but to relieve the patient's hypothyroid symptoms and the risks and severity of psychological and somatic disorders that often accompany autoimmune thyroiditis. Many studies have shown the efficacy of thyroxine treatment. The best efficacy is reached when the dose is high enough to suppress the TSH level in the serum.

What is the best thyroid therapy? In most cases, I suggest desiccated thyroid extracts such as Armour, Erfa, or Nature Thyroid (doses between 30-180 mg/day) because they work better, in my experience, due to their high content in T3, T2, T1, and T0, which are not found in treatments with thyroxine alone. Be careful with patients who are allergic to pork. Most desiccated thyroid are of porcine origin and should be avoided by patients with pork meat allergy. In this case, synthetic T3-T4 combinations might be an acceptable alternative, but are not as efficient as desiccated thyroid. Triiodothyronine alone is not indicated because of the lack of persistence of beneficial effects over a 24-hour period (except if administered in 5 divided doses per day). Thyroxine alone may help on the condition that it clearly reduces the TSH level, even suppresses it. Titrate the dose up to just below the level that causes signs and symptoms of thyroid excess.

Glucocorticoid and DHEA treatments: One of the roles of cortisol is to prevent autoantibody production. In cases of cortisol deficiencies, such as Addison's disease, the risk of autoimmune thyroiditis considerably increases.[126] This explains why glucocorticoid treatments may significantly decrease the production of antithyroid antibodies but, in my experience, rarely eradicates it totally at physiological doses. Hydrocortisone (bioidentical cortisol) at doses of 15-35 mg/day in at least two divided doses (more in the morning, less at lunch) is recommended for patients with adrenal deficiency and autoimmune thyroiditis. When levels of antithyroid antibodies are high, treatment with a long-acting synthetic derivative of cortisol during the first six months may be more efficient to reduce the antithyroid antibody titers.[97,393-395] Prednisolone, which has more persistent effects over the next 24 hours than bioidentical hydrocortisone whose biological action fades after 6-9 hours, is then a good choice.

Add anabolic DHEA (dehydroepiandrosterone) to any glucocorticoid treatment in doses that are at least equivalent in milligrams to the dose of hydrocortisone that is given (15-35 mg/day). The anabolic actions of DHEA neutralize any excessive catabolism from the glucocorticoid treatment and have the additional benefit of further reducing antithyroid antibodies, as shown in women with Hashimoto's thyroiditis and premature ovarian failure. In these women, DHEA sulfate levels were found to be significantly lower than in women without Hashimoto's and normalized with a substantial reduction of anti-thyroperoxidase antibodies at 30 mg/day of DHEA. DHEA treatment also normalizes the natural killer cell toxicity, which is deficient, in patients with Hashimoto's thyroiditis.[397]

Testosterone therapy, along with female hormone treatments in women, has been reported to oppose the development of various types of autoimmune disease. In autoimmune thyroiditis, a significantly lower testosterone to estradiol ratio is found in men.[148] Testosterone therapy, on the other hand, has been shown to reduce antithyroid peroxidase and antithyroglobulin antibodies in men and animals with the disorder.[398-402] In women with autoimmune lupus, the testosterone is also significantly lower in the serum.[403]

Table 5 gives an overview of various autoimmune pathologies in which testosterone therapy was shown to reduce the autoimmune antibody levels.

As discussed above, the reason autoimmune thyroiditis is 5 to 10 times more common in women than men may be due to their much lower testosterone levels, which leave them less protected against autoimmune dysregulation. Regarding lupus, another autoimmune disorder, testosterone levels are lower in women who have lupus than in women without it, and testosterone therapy has been reported to reduce the production of autoimmune antibodies in women with lupus. I have not (yet) found studies on testosterone therapy for autoimmune thyroiditis in women.

However, if testosterone therapy is administered to a female patient, it should always be done in combination with sufficient female hormone therapies to avoid masculinization. Estrogen and progesterone therapy protect women against hair loss, body hair overgrowth, acne, and other undesirable effects of testosterone ➤

Because of its frequency and adverse impact, Hashimoto's thyroiditis should be systematically screened and treated in women.

Table 5: Autoimmune diseases that may improve (decrease) with testosterone therapy

- Autoimmune thyroiditis (men,[398] rats,[399-400] chickens) [401-402]
- Autoimmune encephalomyelitis (rats)[404-405]
- Autoimmune demyelinating disease (mice)[406]
- Autoimmune epilepsy (men)[407-410]
- Autoimmune disease in general (mice)[423]
- Sjögren syndrome (men,[411] mice)[412]
- Systemic lupus erythematosus (men, women,[413-418] mice)[419-420]
- Autoimmune orchitis (rats)[421]
- Autoimmune prostatitis (mice)[422]

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therapy administered alone. In case high amounts of intramuscular testosterone injections are administered, finasteride, a progesterone derivative, which reduces the conversion of testosterone to the masculinizing dihydrotestosterone, may have to be added and is safe only in presence of testosterone supplementation.

Table 6 overviews the most important hormone treatments for Hashimoto's thyroiditis.

Causes to Treat: Viral, Bacterial, and Yeast Infections

Infections by microorganisms have been reported to cause or contribute to the development of autoimmune thyroiditis. Both a leaky gut, which permits these microorganisms to penetrate into the body, and an inefficient immune system contribute to make a patient's thyroid gland prone to infection by viruses, bacteria, and yeast, which trigger autoimmune reactions. In autoimmune thyroiditis, two immune failures have been discovered: a defect in the number of CD8(+) suppressor lymphocytes[424] and a decrease in efficacy (toxicity) of natural killer cells.[425-426] CD8(+) suppressor lymphocytes are those that oppose the development of autoimmune thyroiditis. Natural killer cells serve to contain viral infections to provide time for the immune system to produce antigen-specific cytotoxic T cells that respond to the invaders and clear the infection. Natural killer cells control viral infections by secreting interferon γ and tumor necrosis factor α.

Viruses seem to trigger or amplify autoimmune thyroiditis more than other infectious agents.[427-429] Especially herpes viruses, particularly the Epstein-Barr virus[430-435] (also called herpes virus 4) that causes mononucleosis seems to be worst, but the herpes virus[436-437] that causes herpes labialis has also been incriminated as facilitating Hashimoto's thyroiditis. The hepatitis C virus,[438] parvovirus,[439] human T-cell lymphotropic virus type 1,[440-442] and HIV virus[443-444] have also been associated with autoimmune thyroiditis.

Yeast is another trigger of autoimmune diseases, and, therefore, potentially also of autoimmune thyroiditis. Patients with autoimmune diabetes, for example, are four times more likely to have anti-

Table 6: Hormone treatments that reduce antithyroid antibody levels

TREATMENT	INDICATION	DOSE	DOSE APPLICATION	EFFICACY**
Thyroid				
• Oral desiccated thyroid	Mild to severe HT*	30-150 mg/day	• Start at a low dose and gradually (every 10-14 days) increase the dose to the optimal level	+±
• T4/T3 combinations		10 µg		+
• Thyroxine		75-200 µg/day		+
Cortisol				
• Hydrocortisone (bioidentical)	Mild to severe HT	15-35 mg/day (women) 20-60 mg/day (men)	• Start directly at the useful dose (in rare cases of acute inflammation with very high antithyroid antibody levels, start at 50%-100% higher than the ideal dose [for 2-3 months], then decrease to the physiological dose) • Always with a sufficient amount of DHEA	+
• Or Prednisolone	Mild HT	2.5-5 mg/day		+
	Severe HT	5-10 mg/day (max. 2-3 months)		++
• Or Methyl-prednisolone (in overweight or hyper-tensive patients)	Mild HT	2.5-5 mg/day		+
	Severe HT	5-10 mg/day (max. 2-3 month		++
DHEA				
• DHEA	Mild to severe HT	Women 10-30 mg /day	DHEA should always be added to a glucocorticoid treatment	± to +
		Men 20-60 mg/day		± to +
Testosterone				
• Transdermal testosterone gel or liposomal cream	Mild HT	Women: 0.5%: 0.5 -1 g/day (2.5-5 mg/day) Men: 10%: 0.5 -3 g/day (50-300 mg/day)	• Always in combination with transdermal estradiol 1-3 mg/day and 100 mg/day of progesterone	+
• IM testosterone enanthate injections	Severe	50-100 mg/month	• Almost always with finasteride (2.5-7.5 mg/day) to block masculinization	+±

Note: *HT = Hashimoto's thyroiditis: **mild:** antithyroid antibody levels reach a maximum of 10 times the upper limit; **severe:** antithyroid antibody levels are far higher than 10 times the upper limit; ** efficacy to reduce autoimmune antibodies.

Candida (enolase) IgG antibodies,[445] and twice more likely to have Candida albicans overgrowth in the stools.[446] Furthermore, experimental autoimmune encephalomyelitis is considerably aggravated if mice are beforehand infected by Candida. Bacteria that have been associated with thyroiditis include streptococci, staphylococci,[437] Yersinia, Borrelia (Lyme disease),[448-449] and Helicobacter pylori.[435] Among the parasites that have been associated with autoimmune thyroiditis is Treponema gondii.[450]

demonstrated that thymosin-alpha-1 strongly opposes the development of autoimmune thyroiditis in mice prone to produce antithyroid antibodies. However, in mice, relatively resistant to autoimmunity, thymosin-alpha-1 may trigger a mild form of autoimmune thyroiditis.[456-457] For this reason, treatment with thymosin-alpha-1 is probably and mainly indicated in patients with high titers of thyroid antibodies.

Thymosin-alpha-1 activates the natural killer cell activity.[458-460] In vitro, it has been shown to inhibit the proliferation of the HIV virus in infection of macrophages and polymorphonuclear cells by activating CD8(+) cells so powerfully that some researchers think it makes a re-evaluation of the approach to antiretroviral therapy necessary.[461] In Lyme disease, this treatment is, in my experience, efficient in reducing the aggressiveness of the disease, much more than prolonged use of antibiotics that has toxic effects that thymosin-alpha-1 does not have. I cannot stress enough that the best therapies are those that treat the cause (the immune deficiency due to a lack of thymus hormones, for example) than the consequences (providing long-term antibiotics to kill intracellular microorganisms which install themselves when there is an immune deficiency). Furthermore, thymosin-alpha-1 is anti-inflammatory, a beneficial effect in autoimmune diseases.

Table 7 shows the most important immune-enhancing treatments for patients with Hashimoto's thyroiditis.

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Table 7: Treatments that reduce antithyroid antibody levels by improving the immune system

TREATMENT	INDICATION	DOSE	DOSE APPLICATION	EFFICACY**
Dietary adjustments See above table 3				
Vitamin D See above table 4				
Thyroid See above table 6				
Thymus hormone				
• Thymosin-alpha-1	Moderate to severe HT	0.05 mg/day (0.03-0.15 mg/day)	Start directly at the useful dose. The higher the dose the greater the efficacy.	+ to +±

How to avoid getting the thyroid invaded by microorganisms that trigger autoimmune thyroiditis? First of all, by blocking the passage of these microorganisms through a leaky gut by a healthy diet, as explained earlier in this article. Second, by restoring an optimal immune system by hormone and nutritional supplementations (of any deficiency) so that even if microorganisms pass through the gut wall, they are destroyed by the body's natural defenses. *Thyroid therapy* is a strong immune booster that has been shown to increase natural killer cell toxicity[451-453] and increase both the number of CD4 helper and CD8 suppressor cells.[454-455]

Moreover, daily subcutaneous injections of *thymosin-alpha-1,* probably the body's most potent immune-enhancing hormone may be an additional powerful tool. Research has

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Causes to Treat: Pollutants

Breathe pure air. Pollutants trigger the development of autoimmune thyroiditis by attacking the immune system and tissues. There are so many toxins that we cannot list them all, but let's cover some of them.

Smoking increases the risk of autoimmune thyroiditis.[462-463] High cadmium levels have been associated with autoimmune thyroiditis,[464] as has radioactive fallout from Chernobyl, for example.[465-466]

Endocrine disruptors such as polychlorinated biphenyls (PCBs), used as coolants and insulating fluids (transformer oil) for transformers and capacitors or plasticizers in cement, are so persistent that even if a baby receives them through breast-feeding from his mother, they remain in the body as a young adult. Young adults who were breastfed as babies have been reported to have higher levels of the various types of persistent PCBs and antithyroid peroxidase antibodies than individuals who were not breastfed.[467]

Table 8 provides an overview of the main recommendations to give to patients to lessen the toxic load.

Table 8: Pollution-free living to reduce antithyroid antibody levels

Globally, the recommendations include the following:

- **Drink lots of purified water** to evacuate pollutants in the urine.
- **Consume organic foods** and avoid toxic foods, including phthalates that leak into food from packaging.
- **Breathe pure air** by living and working in safe neighborhoods.
- If indoor air is not clean, use **indoor air filters**. Any odor of synthetic projects or exhaust gases should prompt an alarm to evacuate the area.
- **Stop smoking.**

Conclusion

Because of its frequency and adverse impact, Hashimoto's thyroiditis should be systematically screened and treated in women. The treatment of autoimmune thyroiditis (particularly Hashimoto's thyroiditis) is generally a combination of therapies consisting of, at least, dietary adjustments, nutritional therapies (selenium and vitamin D3 being the most important ones), and reduction of any toxic overload. However, in more severe or treatment-resistant forms with highly elevated antithyroid antibody levels, hormone therapies, such as thyroid, glucocorticoid, DHEA, and/or testosterone (associated with female hormone therapy in women), are generally inevitable, and optimization of the immune system might be necessary. In case the patient is hypothyroid, thyroid therapy becomes essential to treat the hypothyroidism and can considerably reduce antithyroid antibody levels too. ◆

Born in Antwerp, Belgium, Dr. Hertoghe practices his medicine in his clinic in Brussels. With his sister, Dr. Thérèse Hertoghe, they proudly represent the fourth successive generation of physicians working with hormonal treatments – and this since 1892 (after Eugène Hertoghe, former vice president of the Royal Academy of Medicine in Belgium, and Luc and Jacques Hertoghe, endocrinologists). Dr. Thierry Hertoghe devotes his life to the promotion of a better, patient-oriented, and evidence-based medicine.

Author of numerous books, Dr. Thierry Hertoghe also travels a lot to take part in numerous conferences and congresses throughout the world. He co-organizes many of these specialized gatherings and holds important positions in several international and national medical organizations (which usually tend to fight against aging). He is the president of the International Hormone Society (over 2500 physicians), and of the World Society of Anti-Aging Medicine (over 7000 physicians), as well as the supervisor of two important postacademic trainings for doctors.

http://www.hertoghe.eu

Full references for

"Hashimoto's Thyroiditis, A Common Disorder in Women: How to Treat It" Parts 1 and 2 by Thierry Hertoghe, MD

and

"An Integrative Medical Approach to Macular Degeneration" Parts 1 and 2 by Marc Grossman, OD, LAc

can be accessed at www.townsendletter.com

An Integrative Medical Approach to Macular Degeneration – Part 2

by Marc Grossman, OD, LAc

Editor Note: In Part 1, published last month (*Townsend Letter*, April 2020), Dr. Grossman describes age-related macular degeneration, its risk factors, conventional treatment, and the role of diet. In Part 2, the conclusion of this article, Dr. Grossman focuses on nutrients and other ways to preserve vision.

Recommended Nutrients

Essential nutrients for the retina and macula include the following:

Lutein (6 mg-30 mg per day)**.** Lutein and zeaxanthin are two similarly structured carotenoids that make up the macular pigment in the retina and help protect against damaging blue (visible) light as well as acting as powerful antioxidants. This pigment functions as a yellow filter to protect the light-sensitive photoreceptor cells (the cones) from light-originated (especially blue light) free radical damage. These carotenoids are also used in the lens of the eyes and act like internal sunglasses, protecting the eyes from damage against sunlight.[1-5] Lutein has also been shown to reduce eye inflammation.[6]

Zeaxanthin (2 mg-12 mg per day).[5] Lutein and zeaxanthin supplements are best taken separately from beta-carotene supplements because of competition for absorption. Lutein and zeaxanthin need fat to absorb well, so take them with food or a small amount of oil. The best time to take one of the dosages of lutein is before you go to bed.

Omega-3 fatty acids (2,000 mg-3,000 mg per day). Omega-3 fatty acids, found in fish, are a primary component of retinal photoreceptors and of the myelin sheath that surrounds nerve fibers in the eye. DHA (a type of omega-3 fatty acid) has been found to have antioxidative, anti-inflammatory, anti-apoptotic, and anti-angiogenic (limiting growth of new blood vessels) effects.[3,7-11]

Vitamin D3 (2,000 IU-5,000 IU per day). Numerous studies have found that low levels of vitamin D3 in the body can be connected to an increase in the presence of macular degeneration.[12,13]

Astaxanthin (6 mg-12 mg per day). Astaxanthin has been shown to be effective in protecting against damage from light due to its antioxidant effect.[14] Astaxanthin destroys the unstable molecules called reactive-oxygen species (ROS), commonly known as free radicals, and wards off their constant attack towards all parts of the body.[15] It helps slow down oxidative damage and protect the photoreceptors.[16] Astaxanthin is a fat-soluble carotenoid which, in its value as an antioxidant, is ten times more powerful than beta-carotene, lutein, or zeaxanthin, and from 60 times to 500 times stronger than vitamin E. It must be taken in through food or in a supplement since it is not made by the body. For best absorption, astaxanthin should be taken with a healthy fat. Some astaxanthin supplements contain some fat in their formulation. In addition, in one study subjects fed 2 mg astaxanthin also showed lower plasma C-reactive protein concentrations, demonstrating the anti-inflammatory action of astaxanthin in humans.[17]

Meso-zeaxanthin (10 mg) is a carotenoid in the lutein family.[18] Studies show that supplementing with meso-zeaxanthin helps protect central vision for those with AMD. One study done on 2007 found that levels of all three carotenoids (lutein, zeaxanthin and meso-zeaxanthin) increased blood and macular pigment density.[19]

Trans Resveratrol (175 mg per day). This potent antioxidant helps protect retinal cells from damage,[20] reduces oxidative damage,[21,22] and supports a healthy inflammatory response.[23] It also maintains the health of existing blood vessels while suppressing the growth of new ones (angiogenesis), similar to the neovascularization that can affect those with diabetic retinopathy. It reduces retinal pigment epithelial (RPE) cell degeneration, leading to malfunction of blood-retinal barrier and loss of vision.[24]

Grape seed extract (300 mg per day) helps to strengthen blood vessels, maintain healthy platelet function and other aspects of platelet responses,[25] and protects the central nervous system from reactive oxygen species.[26]

Vitamin C (buffered: 2,000 mg-3,000 mg per day). As an antioxidant, vitamin C scavenges free radicals in the body and protects tissues from oxidative stress.[27,28]

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Saffron. Studies have shown that saffron helps protect photoreceptor cells from damage and support healthy circulation in the retina.[29-34]

In addition to these essential nutrients, daily home sessions of microcurrent stimulation (MCS) are recommended. The MCS 100ile unit is the most researched unit used for retinal health related to AMD. Five research studies have been done to date showing MCS as a method to daily support retinal and photoreceptor health in three ways: 1) supports healthy circulation to the retina; 2) increases energy (ATP) production within the retinal cells; 3) helps the retina eliminate waste.[35-40]

Other very important nutrients for eye health include the following:

Bilberry (120 mg-180 mg per day). Bilberry extracts have been shown to help night time vision and have potent antioxidative properties that not only are neuroprotective, but they also help suppress photooxidative processes and have been shown to improve microcapillary circulation.[41,42] Bilberry is helpful for macular degeneration because it protects the retina against oxidative stress resulting from free radicals.[43]

Gingko Biloba (120 mg per day) helps support healthy circulation to the eyes and body overall and helps maintain the normal function and tone of blood vessels.[44-46]

Glutathione (500 mg – 900 mg per day) is a potent antioxidant shown to help protect retinal cells from damage. Best taken sublingually as it is not well absorbed through the capsules or tablets.[47,48]

Taurine (750 mg-1,000 mg per day) is a potent antioxidant found in the retina and essential in helping the eyes eliminate waste. It also enhances the rods and cones within the retina that serve as visual receptors.[49]

Lycopene (3 mg per day). Persons who had the lowest serum levels of lycopene, which is the most abundant carotenoid in the serum, were twice as likely to have macular degeneration when compared to those with the highest levels.[50]

Melatonin has prevented destruction of cells in the retina, including retinal neurons.[51] And a research study showed this combination of melatonin (3 mg), zinc (8.7 mg) and selenium (50 mcg) taken before bedtime helped stabilize AMD, with some remarkable improvement in the fundus of the eye after taking the supplement for six months. (This is all in the Dr. Pierpaoli formulation.)[52]

Helpful foods and nutrients include the following:

- Green tea (500 mg – 750 mg per day)[53,54]
- CoQ10 (100 mg – 200 mg per day)[55,56]
- Selenium (200 mcg per day)[57,58]
- Dietary enzymes to increase glutathione synthesis, which can prevent free radical-induced apoptosis (cell suicide) and may help prevent or treat AMD.
- Goji Berries: A study found that daily supplementation with the milk/goji formulation increased the levels of zeaxanthin in the blood and protected from additional drusen formulation or loss of pigmentation.[59]
- Quercetin (250 mg-500 mg per day) has been found to be helpful through its antioxidant capacity to protect retinal pigment from oxidative stress caused by solar radiation.[60] For wet macular degeneration, quercetin was also found to inhibit formation of extra blood vessels as well as improve blood flow in the choroidal layer of the retina.[60]
- Rutin (500 mg per day) combats inflammation and oxidative stress involved in the development of macular degeneration. Furthermore, research has indicated that rutin reduces leakage from the tiny retinal blood vessels and combats inflammation.[61]
- Curcumin (400 mg per day) has been shown to inhibit VEGF and cell death and is of great interest to researchers.[62] In other research, curcumin has been found to have a protective effect against cell damage in human retinal pigment cells caused by blue light. The conclusion was that curcuminoids may have potential in AMD treatment.[63] This research corroborates earlier studies.[64]
- Dark Chocolate (the darker the better) contains high amounts of epicatechin which has been found to have found to have three times the antioxidant value of green tea. One of the primary benefits of epicatechin is that it is a powerful antioxidant and because it can cross the blood-brain barrier it can be delivered directly to the brain (as well as reaching the optic nerve and retina.[65]

More About the Nutrients

Essential Fatty Acids (EFAs). EFAs are just as essential to a healthy balance of body chemistry and proper cell function as good food, vitamins, and minerals are. They are an integral component of nerve cells, cell membranes, and vital hormone-like substances called prostaglandins. Prostaglandins help regulate numerous body functions, including normal immune response during inflammation. Unsaturated fatty acids are also necessary for healthy skin, hair, and nails; they have a supportive effect on the circulatory system and can reduce blood cholesterol levels. Good food sources of unsaturated fats include nuts (almonds, macadamia nuts, hazelnuts, pecans, cashews), avocado, peanut butter, walnuts, sardines, seeds, salmon, olives, flaxseed, fatty fish (salmon, tuna, mackerel, herring, trout, sardines, anchovies), and other seafoods, including oysters and mussels. Olive oil (organic, first cold pressed) is a great oil for use in salads and dipping with bread. Olive oil can be used for cooking, but it should not be cooked at high temperatures. The best oils for cooking are saturated fats such as grass-fed butter, avocado oil, or coconut oil.

Oil	Omega-6 Content	Omega-3 Content
Safflower	75%	0%
Sunflower	65%	0%
Corn	54%	0%
Cottonseed	50%	0%
Sesame	42%	0%
Peanut	32%	0%
Soybean	51%	7%
Canola	20%	9%
Walnut	52%	10%
Flaxseed	14%	57%
Fish*	0%	100%

Vegetable oils have a much higher omega 6 to omega 3 ratio, which can

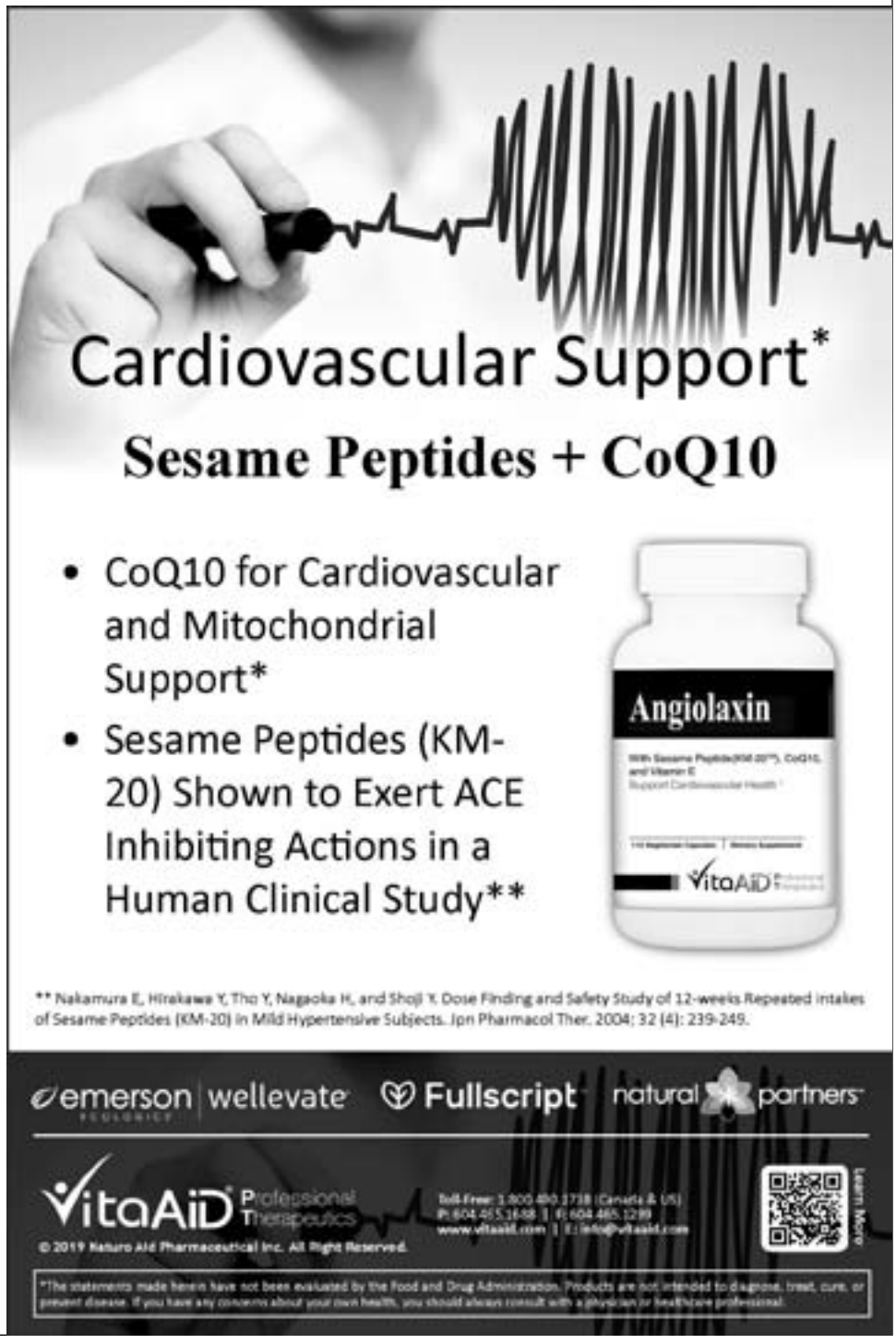
lead to health problems. Both omega 6 and 3 use the same conversion enzymes. So, higher levels of omega 6 from vegetable oils in the body convert to inflammatory eicosanoids and reduce the conversion of omega 3 to anti-inflammatory DHE/EPA.[66]

Omega-3 fatty acids are a specific type of EFA known to reduce inflammation and lower risk of chronic diseases such as heart disease, cancer, and arthritis. They are so essential to the retina that when omega-3 levels begin to fall, the retina begins to recycle them within the eye. Omega-3 fats are essential for nerve conduction in the retina and for reducing cholesterol; this keeps retinal blood vessels open, helping to maintain retinal nutrition. The typical American diet is deficient in omega-3 fatty acids, whereas omega-6 fatty acids are generally plentiful due to a high intake of vegetable oils and refined grains, which includes white breads, white rice, and white pasta. The best sources of omega-3 fatty acids are cold-water fish such as salmon, herring, and mackerel as well as black currant seed oil, flax seeds and flax seed oil, and chia seeds. Other plant-based sources of omega-3s are walnuts, fresh basil, grape leaves, spinach, cauliflower, arugula, Romaine lettuce, as well as Boston and bibb lettuces, certain beans (pinto, kidney and soy), Brussels sprouts, and tofu.

Vitamin D3. Researchers have found that low levels of vitamin D3 in the body are connected to an increase in the presence of macular degeneration. Studies also showed that supplementing with vitamin D could lower AMD risk in women who were younger than age 75.

Astaxanthin is a powerful antioxidant in the eyes that helps to prevent damage due to exposure to sunlight, reduces DNA damage, and in many ways is more powerful as an antioxidant than vitamin C, vitamin E, and beta-carotene. Other overall health benefits include being a natural anti-inflammatory, boosting the immune system, reducing the risk of certain types of cancer, protecting cardiovascular health, supporting skin health, and increasing muscle recovery after exercise. Foods that contain astaxanthin include krill, algae, sockeye salmon, red trout, red seabream, shrimp, crabs, lobster, crawfish and salmon roe.

Bilberry. Forty years of research confirm bilberry's benefits for the eyes. Nicknamed the vision herb, it contains high amounts of anthocyanins, which improve the delivery of oxygen and blood to the eye, help maintain the integrity of capillaries, and help stimulate the production of rhodopsin, needed for night vision. The antioxidants contained in bilberry strengthen collagen and promote the health of tissues. Studies show that the high content of anthocyanins are a class of compounds in the flavonoid ➤



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family (plant compounds that are found in almost all fruits and vegetables) with antioxidant benefits that are found in many fruits and vegetables and are responsible for giving these plants red, purple, and blue coloring. Bilberry is also rich in tannins, which are astringent in nature and have anti-inflammatory properties.

liver and heart contain more taurine than choice cuts of flesh.

Resveratrol is a powerful antioxidant that helps protect against oxidative stress in the eyes. It also has natural anti-inflammatory benefits and aids in protecting the endothelial lining of the arteries, both in the eyes and throughout the body. The trans-resveratrol version is easily absorbed and is often included with Japanese knotweed root in formulas. Resveratrol increases NAD+ which is important for mitochondrial health and ATP energy production (which is what fuels our cells).

Protecting eye health begins with good nutrition, regular exercise, and stress management.

Meso-zeaxanthin is a carotenoid similar to lutein and is found in the center of the macula where we get our most detailed vision. It is found in microalgae and sea creatures that consume the algae, such as salmon, shellfish, and krill. A 2015 study showed supplementing daily with meso-zeaxanthin significantly increased macular pigment (found to be compromised in those with macular degeneration).

Ginkgo biloba. A double-blind study conducted by scientists in France in 1986 found that Ginkgo biloba could help people with macular degeneration, possibly due to its benefit of improving blood circulation to the brain.

Taurine is an important amino acid needed for the maintenance of vision, and the regeneration of worn-out tissues of the visual system. Taurine is found in high concentrations in the retina, especially in the photoreceptor cells where it protects cells from ultraviolet damage. Studies have shown that when taurine is removed from food, animals develop retinal degeneration; this is reversed when taurine is replaced. Taurine is also an essential nutrient in helping to remove waste deposits formed in the back of the eyes from exposure to sunlight. Unfortunately, taurine concentration in our cells decreases as we age. Food sources of taurine include fish, such as salmon, tuna, sardines and shellfish, beef, lamb, dark chicken meat, eggs, most dairy products, seaweed, krill, and brewer's yeast, human breast milk, sea algae and plants. Organ meats such as

Selenium may help prevent oxidative damage in the retina along with zinc; it was included in the AREDS studies.

Zinc is found in high concentrations in the eye, especially the retina and its underlying tissues. Zinc helps bind the protective pigment layer of the retina to the underlying tissue. Supplementing with zinc has been shown in the AREDS studies to help support retinal health for those with AMD (along with other nutrients included in the study).[28] Zinc also plays a vital role in bringing vitamin A from the liver to the retina in order to produce melanin, a protective pigment in the eyes. Zinc is highly concentrated in the eye, mostly in the retina and choroid, the vascular tissue layer lying under the retina.

Green tea is high in antioxidants called catechins that along with vitamin C, vitamin E, lutein, and zeaxanthin help protect the delicate tissues in the retina. Catechins are classified as flavanols (a distinct group of compounds within the flavonoid family and the predominant flavonoids in tea, red wine and cocoa) and act as antioxidants in the body, helping to neutralize free radicals.

CoQ10. Researchers reported that CoQ10 may improve the function of cell tissue in the retinal pigment, which, in turn, improves the function of the retina in those with age-related macular degeneration. In another study, CoQ10 has been shown in research to increase visual acuity and to reduce drusen in those with macular degeneration.

Pycnogenol and/or grape seed extract contain powerful antioxidants called proanthocyanidins that are many times stronger than vitamin E and vitamin C. Proanthocyanidins have potent free radical scavenger ability. They also strengthen capillaries, arteries, and veins, improve circulation, reduce capillary fragility, and reduce nerve damage in the eye. In his 1993 book, *The Superantioxidant*, Richard Passwater mentions studies that have shown that pycnogenol improves visual acuity and the functioning of the retina, especially in cases of retinal damage caused by microbleeding of the eye capillaries due to blood sugar imbalances.

Vinpocetine is an extract from periwinkle seeds that helps increase retinal circulation. In one study, 100 people with atherosclerosis and eye disorders were given vinpocetine. Eighty-eight responded with increased retinal circulation and improved visual acuity. Vinpocetine has been shown to support healthy circulation to the retina and brain and is a popular herb used in Europe to help with stroke prevention and memory enhancement. By increasing blood flow and stepping up concentration of ATP (the energy carrying molecules of our cells), vinpocetine improves utilization of glucose and oxygen in the brain and the retina. It also inhibits abnormal platelet aggregation, improves red blood cell elasticity, and inhibits an enzyme (GMP) that causes arterial constriction and blood flow reduction. Thus, arteries relax, blood pressure normalizes, and blood flow increases. In clinical studies, vinpocetine has been shown to benefit depression, headaches, short-term memory, inner-ear conditions, tinnitus, vertigo, menopausal symptoms, insomnia, speech impairment, stroke, and eyesight disorders.

Vitamin A (also called "Retinol") is required for the formation of the photoreceptor rhodopsin, which acts as a regulator of light-activated

photochannels (essentially enabling us to see by providing a mechanism for the photoreceptor cells to pass light to the optic nerve which then goes to the visual cortex in the brain.)

Vitamin E. Studies indicate that vitamin E reduces the progression of both AMD and cataract formation. Vitamin E also plays a significant role in the immune system, the health of cell membranes, DNA repair, and in other metabolic processes. Vitamin E can be found in high amount in nuts, fortified cereals, and sweet potatoes.

Note: Many eye doctors recommend taking an AREDS formula, based on two studies showing that supplementing with this formula can reduce the risk of advancement of macular degeneration. The Age-Related Eye Disease Study (AREDS), sponsored by the federal government's National Eye Institute, determined that taking high levels of antioxidants and zinc could reduce the risk of developing advanced age-related macular degeneration (AMD) by about 25 percent. Research has shown that targeted, therapeutic doses of certain supplements not only reduce the risk of macular degeneration but may prevent additional vision loss as well as potentially improve vision in people who already have AMD. As many of these formulas contain food coloring and fillers, we prefer these nutrients, instead, be ingested as part of a whole food, organic multivitamin.

Although genetic testing is available now to help determine one's risk of getting AMD, there has been some controversy among eye care professionals as to whether genetic testing should be done in at-risk patients with certain genetic polymorphisms (CFH and ARMS2). It has been suggested that certain patients actually do worse with zinc supplementation. In 2015, the recommendations from Bascom Palmer stated there was not enough evidence to recommend routine genetic testing when considering supplementation. Dr. Stuart Richer, lead researcher in the last lutein antioxidant supplementation trial, recommends the use of lower amounts of zinc (less than 50 mg daily), determined to be as effective as taking higher doses. He also recommends that genetic testing be offered to monocular patients (where one's two eyes see near far distance differently) to avoid the possibility of a zinc hyper-immune response that is a possibility in one out of seven of all high-risk AMD patients.

Good food sources for zinc include red meat, seafood, poultry, eggs, pumpkin seeds, wheat germ, mixed nuts, black-eyed peas, tofu, and baked beans.

Self-Help Tips

The following are additional measures to prevent or treat macular degeneration. ➤

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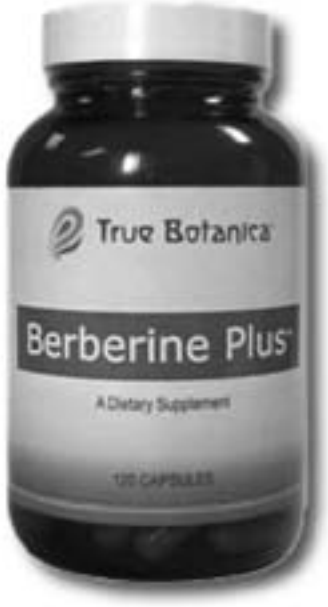

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Sunglasses should be worn outside in bright sunlight. They should be 100 percent UVA and UVB blocking lenses with wrap-around sides. Amber or brown lenses are the most effective colors for neutralizing the blue light spectrum, which is as potentially damaging as UVA and UVB light. Wear sunglasses along with a three-inch brimmed hat.

Aspirin thins the blood, so some doctors recommend it for improving blood flow to the retina; but several studies have shown that aspirin actually can cause macular degeneration through retinal hemorrhages.[67] Therefore, try to avoid aspirin, particularly if you have a family history of macular degeneration.

Exercise regularly; exercise does so many things to affect the health of the body, including increasing circulation, which promotes the health of the body, including the eyes.

Eat a diet moderate in fats; high fat levels can disturb proper balance of gut bacteria essential for proper digestion and overall health.[68,69] This also includes keeping polyunsaturated fats to a minimum,[70] limiting refined carbohydrates,[71] and eliminating any trans-fatty acids in one's diet, which can increase cholesterol levels and inflammation, both of which affect the eyes' blood vessels.

Drink plenty of water. Adequate hydration is essential. Spring water without chlorine or fluoride is the best. Sip water throughout the day and do not rely on feeling thirsty before drinking.[72] At the point you feel thirsty, your body is already dehydrated. Neurological changes start occurring when there is a 2% drop in total body water. Also, certain eye diseases as well as general health diseases have been associated with dehydration.[73]

Avoid or severely restrict sources of non-native electromagnetic frequencies (nn-EMF). Higher EMF exposure leads to hypoxia, dehydration and inflammation.[74] Turn off all wifi signals at night such as the internet modem, wireless printers, cell phones, computers, etc.

Reduce exposure to artificial blue light from computer screens, cell phones, LED and fluorescent lighting. Even low levels of blue light (400-470 nm) exposure may induce photoreceptor and retinal pigment epithelial cell damage. Light-induced damage also increases with age due to a decrease in protective enzymes such as superoxide dismutase (SOD). Artificial blue light exposure appears to be more damaging at night than during the day.[75] Dr. Celia Sanchez-Ramos from Madrid's Complutense University found that blue light permanently damages retinal cells and once damaged they cannot regenerate. Replace all fluorescent and LED lighting with incandescent bulbs. Other lighting options include beeswax candles and Himalayan salt lamps. There are blue light blocking glasses (ex: Blu-Tech lenses or complete protection BP1550 tints) that can be worn while on the computer, as well as blue light blocking programs that can be downloaded onto your computer and phone.

Ground yourself as much as possible by allowing any part of your body (bare skin) to directly touch the ground. This allows access to the earth's abundant negative ions that can protect us from free-radical-induced inflammation and cellular damage. Grounding connects you to the Earth's magnetic field and can protect your body from man-made EMF, UV light, and cosmic radiation.[76]

In summary, proper diet, nutritional supplementation, and lifestyle improvements, including regular exercise and stress management are fundamental to any preventive program, or as an adjunct to treatment, for macular degeneration. Macular degeneration is a difficult disease to control, and we need to incorporate the best of current knowledge in nutritional support and supplementation.

In addition to the measures presented in this article, there are several adjunctive treatments that can help people protect their vision, including eye exercises, Chinese herbal medicine and acupuncture, homeopathic formulas, essential oils, chelation therapy, hyperbaric oxygen therapy, and IV nutrient therapy. These topics and more are covered in my book *Natural Eye Care: Your Guide to Healthy Vision and Healing.* ◆

Since 1980 Dr. Marc Grossman has helped many people maintain healthy vision and even improve eyesight. He is best described as a holistic eye doctor, dedicated to helping people with such conditions ranging from myopia and dry eyes to potentially vision threatening diseases as macular degeneration and glaucoma. His combined multi-disciplinary approach using nutrition, eye exercises, lifestyle changes and Chinese medicine provides him with a wide array of tools and approaches to tackle difficult eye problems.

Dr. Grossman founded the Rye Learning Center in 1980, a multidisciplinary center for learning problems, in 1996 co-founded Integral Health Associates in New Paltz, New York, and in 1999 co-founded Natural Eye Care, Inc.

His background includes degrees in optometry, biology, physical education and learning disabilities, coupled with yoga, bioenergetics, nutrition, Chinese medicine and acupuncture, the Alexander technique and Feldenkrais. This orientation provides the foundation for an integrated approach to vision and its influence on the body, mind and spirit of each patient.

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References for parts 1 and 2 can be accessed at www.townsendletter.com

Letter to the Editor

Riordan Clinical Study of Continuous Intravenous Vitamin C in Cancer Treatment

Vitamin C (IVC) therapy is widely used in naturopathic and integrative oncology. Preclinical studies of large doses of ascorbic acid (vitamin C) have been reported to show significant anti-cancer effects in animal models and tissue culture investigations.

Studies on understanding the biological activities of ascorbate have led to a number of hypotheses for mechanisms of anti-cancer activity, such as the generation of significant quantities of hydrogen peroxide by the autoxidation of pharmacological concentrations of ascorbate, changes in the metabolic activity, and stimulation of the enzymes that have a cofactor requirement for ascorbate. In addition, high dose ascorbic acid may improve the anti-cancer action of chemotherapeutic agents, boost immune cell functioning, and inhibit angiogenesis.

Many case studies demonstrated the effectiveness of intravenous vitamin C, with various degrees of success. Clinically published IVC case studies report efficacy against a variety of cancers in humans, including pancreatic cancer, bone metastases accompanying breast cancer, non-Hodgkin's lymphoma, liver carcinoma, colon carcinoma, and ovarian cancer.

Several Phase I and Phase II clinical trials have been conducted in the last ten years to test safety and efficacy when IVC is used as an adjuvant with chemotherapy. The results of these trials confirm that IVC can be administered safely.

Most practitioners administer IV ascorbate to cancer patients by bolus infusions two-to-three times per week.

There have been two clinical trials that used continuous IVC infusions. Cameron and Pauling performed a clinical trial in 100 terminal cancer patients. The protocol included an initial 10-day course of IV ascorbate, at a relatively low daily dose of 10 g/day given by continuous infusions, followed by daily oral intakes of 10-30 g/day, in divided doses. Their results showed increased survival time and improved the quality of life of the patients compared with patients who had not received IVC.

The ideas of Linus Pauling were extended in the model developed by Dr. Hickey. He described "The dynamic flow model" that proposes restoring human physiology by administering excess ascorbate, over and above the amount normally absorbed, spread throughout the day, so a consistent supply is achieved.

The second trial of the treatment of cancer patients by continuous infusions was conducted by Dr. Riordan. In this Phase I clinical trial, patients were administered continuous infusions using an infusion pump. In the Riordan Clinic trial, patients were treated by continuous infusion, which was administered over much longer periods of time than bolus intermittent treatments. For most patients the duration of the continuous infusion was at least 20 hours, as the duration of bolus infusion is from one hour to three hours depending on the dosage.

It was an eight-week trial that involved terminal patients with poor prognosis; 24 subjects were given continuous IVC at doses between 150 and 710 mg/kg/day (10-50 grams per day). Most of the patients had colon cancer with liver and lung metastasis and three patients had pancreatic or liver cancer. All patients had several chemotherapy/radiation treatments before entering the study. Seventy-nine percent of the patients had a metastatic tumor. ➤

Letter to the Editor

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In our recent publication, we analyzed previously unpublished parameters from this clinical study, including blood chemistry and blood count parameters that are reportedly related to patient prognosis and degree of inflammation. This included absolute neutrophil and lymphocyte counts and the neutrophil-to-lymphocyte ratio; lactate dehydrogenase, an enzyme involved in tumor initiation, metastasis, and recurrence; creatinine, the depletion of which is associated with cachexia; and glucose, as hyperglycemia is common in cancer patients.

The most obvious effect of IVC therapy was to increase patient vitamin C levels. Consistent with other reports, the plasma ascorbate measurements conducted in this trial showed that vitamin C depletion in cancer patients is common. In fact, ten out of twenty-four subjects entered the trial with plasma ascorbate concentrations undetectable by the colorimetric ascorbate assay used at that time with another four having ascorbate concentrations below the normal range. IVC infusion increased plasma levels to the order of 1 mM. This likely replenished depleted tissue ascorbate stores as well.

As lymphocytes and neutrophils have important roles in tumorigenesis and carcinogenesis, we analyzed the effect of the treatment on these parameters. As the result of chemotherapy, neutrophil and lymphocyte counts typically decrease in cancer patients, with the effect being more severe for lymphocytes.

Analysis of white blood cell counts for patients in this trial indicated the potential for IVC to increase lymphocyte and neutrophil counts for patients in whom these numbers are below normal while reducing neutrophil counts in patients for whom neutrophil counts are elevated.

It was particularly important for lymphocyte counts. Lymphopenia commonly occurs in cancer patients who had chemotherapy and high levels of oxidative stress, induced by treatment, predicting a poor prognosis.

In our study population, about half of the patients who started intervention had lymphocyte counts lower than normal range. For patients with severe lymphopenia, who completed treatment, the median improvement in the lymphocyte count was 69% and for all patients with lymphocyte levels lower than normal range the median improvement was 22%. These data proved that continuous IVC can improve immune function of cancer patients by increasing the level of lymphocytes, especially in patients with low lymphocyte count. Our data also indicated that lower doses are more favorable for the improvement of lymphocyte count.

As absolute neutrophil counts and neutrophil-to-lymphocyte (NLR) ratios are useful prognostic factors in a variety of cancers, with higher values of NLR indicating lower survival times, the effect of continuous injections on these parameters was analyzed. For cancer patients in general, increased neutrophil counts are consistent with systemic inflammation, and a neutrophilic response is associated with poor prognosis, as it can inhibit the immune system by suppressing the cytotoxic activity of T cells. For most of the patients, the tendency during treatment was for normalization of the neutrophil counts, i.e. improvement of neutrophil counts at the low level of this parameter and decreasing for the higher values.

The present analysis of neutrophil-to-lymphocyte ratios (NLR) also demonstrated the regulatory effect of IVC. NLR has been used to assess inflammatory response and has been suggested as a prognostic factor in a variety of cancers. In particular, cut-off values ranging between 2.0 and 4.0 were associated with a significant increase in all-cause mortality. As NLR may reflect the balance between the activation of the inflammatory pathway and the anti-tumor immune function, elevated NLR due to neutrophilia is linked to accelerated tumor development.

In the present study, most of the patients entered the trial with NLRs well above this cut-off. Continuous IVC therapy tended to decrease the rate of growth of NLR. Moreover, we were able to confirm the predictive potential of NLR. Our data demonstrated the relationship between the survival of patients and the rate of growth of NLR, as NLR increases correlated with lower survival times of the patients.

We examined the rate of change in this ratio (ΔNLR) for each patient before and after therapy. The comparison of the trend in the change of NLR measured for periods one week before treatment and during treatment demonstrated that the rate of change was decreased. This suggests that IVC may reduce NLR levels, thus improving prognosis. Since the rate of increase in NLR for patients with initially elevated values decreased during IVC therapy, ascorbate may be decreasing inflammation in these subjects.

As activation of glycolytic metabolism is a significant characteristic of tumor cells, and since lactate dehydrogenase (LDH) is an important coenzyme in glycolysis, elevated levels of serum lactate dehydrogenase may be useful prognostic biomarkers. Lactate dehydrogenase is elevated in many types of cancers; it has been linked to tumor growth, maintenance, and invasion.

The rate of increase of LDH was calculated before and after treatment. The value of this parameter (LDH rate of growth) was decreased in 38% of the patients, increased in 28.6% and was not changed in 33.4% of patients. The result that LDH decreased in 38% of the subjects is remarkable considering their illness.

The median survival time for the all participants with initial LDH higher than normal range (LDH>245 U/L) was 95 days. In contrast, the median survival time for all subjects with normal initial LDH values was 173 days.

Hyperglycemia is another prognostic factor in cancer patients. It is common in cancer patients and represents a challenge during therapy. For example, about 70% of pancreatic cancer patients have impaired glucose tolerance. Moreover, there is a link between the lowering of blood glucose concentration and remission of malignancy. In one study, patients under insulin coma

therapy for six months (for psychosis) were reported to become free of large tumor burdens considered incurable by their oncologists.

Hyperglycemia was common in our cancer patients. Two thirds of the patients in our study had above normal blood glucose concentrations. There were changes in blood glucose during IVC therapy. Blood glucose levels were decreased for patients with the concentrations higher than normal range during IVC therapy.

Several clinical trials have established that IVC can be administered safely. In the continuous IVC infusion trial from which data for the present analysis are obtained, side effects were mostly minor and the criterion for stopping the clinical trial (two or more Grade 3 or higher adverse events at a given dose at least possibly related to the treatment) was never reached. Briefly, blood chemistry parameters that serve as indicators of renal function (BUN, creatinine, and uric acid) remained relatively stable or in the case of uric acid, decreased during therapy. Only four subjects experienced BUN increases during therapy.

The most common side effects were nausea (11 subjects), injector port occlusion (10 subjects), dry skin or mouth (7 subjects), edema (7 subjects) and fatigue (6 subjects). These were generally minor (Grade 1). Most of the Grade 3 events involved hypokalemia, which is considered possibly related to the ascorbate therapy.

In summary, the present analysis demonstrated the regulatory and normalizing effect of continuous IVC infusions on lymphopenia, neutrophil-to-lymphocyte ratios, and absolute neutrophil counts. Despite the very poor health status of patients, continuous IVC treatment had positive effects on the important parameter that characterized tumor metabolism (lactate dehydrogenase) and blood glucose concentration.

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Ask Dr. J

by Jim Cross, ND, LAc
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Would You Rather Fight Than Switch?

From 1963-1981, the Tareyton cigarette company ran an incredibly successful advertising campaign for their brand of cigarettes: "I'd rather fight than switch brands from Tareyton."[1] They would have people with black eyes in their commercials swearing by the above slogan. This campaign was very successful as it targeted an important group of cigarette smokers: the rebels in our society. They would rather fight than switch and give up any portion of their lifestyle, especially cigarette smoking. Now, in 2020, we have entered a similar arena with our patients: would they rather fight our dietary recommendation to intermittently eat (remember, use the term *intermittent eating not intermittent fasting* as fasting implies being deprived) and continue suffering through their chronic diseases, or will they see the light of the positive changes that occur with intermittent eating and switch their food habits to begin the long, nutritional road back to a functional life?

Before I dig deeper into the actual switching mechanism involved, why is food so important again? Our patients tend to have short memory spans, so we need to continuously update them on the importance of choosing food that will nurture and nourish their bodies. (Unfortunately, we practitioners sometimes also have similarly short memory spans!) A 2019 study in the *Lancet* found that one in five deaths globally are the result of an unhealthy diet.[2] As many people have already thoroughly proven, we don't lack access to nutritious food. Farmer's markets have sprouted in almost every locale across the United States. Unfortunately, the nutrient-rich food at these markets is financially out of reach for many Americans. Thus, most people are eating food that overfeeds them but under nourishes their bodies leading to various chronic diseases, two of which are the focus of this month's *Townsend Letter*: cardiovascular health and metabolic syndrome.

Another 2019 study showed that one in eight Americans is metabolically healthy. Being metabolically healthy means having ideal blood levels of glucose, triglycerides, and HDL plus ideal blood pressure and waist circumference *without the use of pharmaceuticals*.[3] Wow, first off, how scary is that fact. Second, I wish my mother and Father Becker hadn't instilled in me a moral inflexibility, or I would be investing in pharmaceutical companies on the stock market. Finally, thankfully, I have made the positive dietary changes in my life so that I am proudly part of the one in eight! So, how can we as practitioners functionally improve that 1:8 ratio through intermittent eating?

Nutritionally, scientifically, what is the big deal about switching to intermittent eating? Basically, it boils down to a metabolic switch, mTOR or Mammalian Target of Rapamycin.[4] mTOR is essentially a master switch that can move our cellular biochemical reactions in the on/biogenesis direction or the off/autophagy direction. It is thus a key regulator of metabolic homeostasis or balance for all approximately 50 trillion cells of our body. Readers of my past columns will also remember that the organ physiologically accomplishes nothing. The cells do the heavy lifting. If they're happy and functioning at a tip top level, the organ will then be also, as will the organism as a whole.

For me, the key here is autophagy. Why is autophagy so important? Essentially autophagy is the major intracellular recycling system by which older, less functionally active cytoplasmic macromolecules and organelles are delivered to and degraded by the lysosome into building-block organic

molecules like amino acids and fatty acids. These metabolic breakdown products by the lysosome are then reused by the cell in a dynamic recycling system that produces new macromolecules and organelles for continual cellular renovation and maintenance of cellular homeostasis.[5,6] Wow, I want some of this in my body! So, how do I turn on autophagy, and do I want it continually turned to the "on" direction?

First off, all dietary strategies are simply meant to mimic what happens in the natural environment. Our ancestors cycled continuously between mTOR being turned on or turned off. When mTOR is turned off, autophagy or intracellular housekeeping kicks in and our cells rid themselves of cellular organelles and macromolecules that have become defective. This protects our cells from damaged proteins and organelles, misfolded/faulty proteins, and other cellular components that can contribute to suboptimal functioning of our cells. Remember, homeostasis or balance is key to an organism that is functionally humming like a 1969 Rambler. Thus, we also need mTOR turned on at appropriately timed intervals so that cellular biogenesis can occur and so that our cells can utilize the recycled materials generated by autophagy to make sure that cellular metabolism can proceed full speed ahead! What a body we inherited. It not only self-analyzes itself and removes worn-out parts but also recycles the material from the worn-out parts to synthesize and replace what was removed in a continuous back and forth! Why can't we humans figure out this seemingly simple system for use in real life?

Because mTOR is a nutrient-sensing pathway, it can be deactivated by intermittent eating and/or severe calorie restriction.[7] Unfortunately modern food consumption paradoxically leads to the availability of unlimited quantities of very quickly digestible foods. This consumption process points the needle in the "on" position for far too many hours of the day. By intermittently eating, we can teach our patients how to deactivate mTOR and activate autophagy daily for a more extended period of time. This will allow cellular R & R (repair and regeneration) adequate time to perform their activities and continually remodel our cells in a positive, health-promoting direction.

Essentially then, one of the best ways to turn off mTOR and turn on autophagy is through intermittent eating. This is what happened before grocery stores. Our ancestors went long periods of time without food, which turned on autophagy and allowed their bodies to assess any damage that had occurred and correct the issue. Then, when they found food, mTOR could be turned back on with cellular repair correcting any damage that had occurred during intracellular housecleaning.

So, let's end with a quote by the esteemed Benjamin Franklin: "Life's tragedy is that we get old too soon and wise too late." In learning about mTOR or the switch, I've gained an insight into true cellular biochemistry and how to move my body and my patients' and family's bodies in a direction that mimics these wise words: *Hopefully the older you become, the healthier you've been*!

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Pediatric Pearls

by Michelle Perro, MD

Dieting Dilemmas and Diabetogens in Children

There is no worse feeling than coming back from lunch to see a slumped teen in a chair in the waiting room, texting, scowling at mom, and wearing a hoodie to hide their largesse. My usual post-prandial wave went into reverse, I put pep in my step and brought JB into the room.

As customary, teen visits start with the parent present, then mom/dad are asked to leave so that my 14-year-old patient could have an opportunity to talk without parental editing. JB had just been diagnosed with type 2 diabetes at a local university hospital, and the endocrinologist wanted to begin pharmacologic intervention along with dietary caloric restriction.

Both JB and his mom did not want to start medication. They sought my help for alternatives to pills and dieting. JB reported that he'd been obese since he was five years old and dieting never worked for him in the past. His medical history was remarkable for being born via C-section, bottle-fed soy formula when he didn't tolerate dairy, and had experienced a few episodes of viral-induced wheezing and environmental allergies. In addition, his dad left when he was three years old, and JB hadn't seen him since. His mom worked two jobs and smoked cigarettes and marijuana both inside and outside their apartment. His ethnicity was a mix of white and Latino.

Their environmental exposure history was a recipe for a disastrous toxic soup. Mom was brought up in Texas near an oil-processing facility. She moved from Texas to California when JB was three. They lived in an apartment on a heavily trafficked street. She didn't buy organic foods and microwaved many of their meals in plastic containers. JB brought a plastic water bottle to school and ate the school meals two times a day, which were also wrapped and heated in plastic. They used non-stick cookware, which was worn and scratched. Canned foods, take-out, and quick-fix processed meals comprised most of both of their diets.

So where to begin?

We start with all the potential sources of metabolic disruption in JB's story, and in his case, environmental exposures were the first go-to.

1. Mom's own inherited epigenetic profile and ensuing environmental exposures of living in a toxic region can induce epigenetic changes transgenerationally.
2. Toxic exposures in utero from endocrine-disrupting chemicals are obesogens, which can alter metabolic control and set the child up for long-term obesity and its sequelae.[1] Twenty-five percent of children with obesity can have impaired glucose tolerance, and 4% have silent type 2 diabetes. An increased prevalence of type 2 diabetes among children and adolescents parallel the epidemic of childhood obesity.
3. Cesarean birth: Lack of inoculation from the vaginal microbiome impairs the initiation of innate immune function. Certain microbial ratios are associated with obesity, such as a decrease in Bacteroidetes.
4. Failure to breastfeed is also linked with obesity.[2]
5. Soy formula can be estrogenic and likely derived from GMOs and their associated herbicides…which can be endocrine disruptors.[3]
6. The early departure of his father may be the cause of stress and up-regulation of cortisol, as well as other inflammatory markers. This can be a set-up for future chronic disease states (think ACEs: Adverse Childhood Experiences, written about in a previous Pediatric Pearls in *Townsend Letter*; December 2019; 437; 84-5).
7. Exposure to polycyclic aromatic hydrocarbons from smoking, or even the oil industry, has been linked to diabetes.[4]
8. JB and mom's diet relied on take-out burritos of rice and beans. Rice is a known source of arsenic, which decreases insulin production.

9. Persistent organic pollutants (POPs) are known to induce diabetes, the worst offenders being BPA (bisphenyl A) and phthalates. JB's meals were daily doses of POPs from his food, water and personal care products. (An interesting note is that people with obesity who do not have elevated POPs do not have an increased risk of diabetes.)[5]
10. Teflon (PFOA) exposure is associated with diabetes.[6]
11. Twenty percent of Latino teens were reported to have type 2 diabetes from 2006-15 from the SEARCH study[7]; a lower risk was reported in White teens.

Positive laboratory findings included low vitamin D, zinc, and magnesium levels. Gamma glutamyl transferase (GGT), a sensitive test to evaluate POPs,[8] was elevated. As to whether the etiology of the elevation in GGT was due to POPs is presumptive and not definitive. JB also had an elevated cholesterol, low HDL, high HA1C and fasting blood glucose levels. His insulin level was slightly increased as well, possibly from the BPA, but again, could not be definitively determined. There may have been elements of both decreased insulin production and/or insulin resistance.

Deconstructing the Toxic Soup

Armed with the ingredients to change the soup recipe, JB and his mom began a program together to reverse his diabetes as well as her own toxic coping mechanisms. Initial work was very involved to change eating and shopping habits. They switched to an organic diet and got off processed foods. An iPhone dietary tracker was used, which JB enjoyed, as a means to get him involved with the plan. The journey wasn't always easy or a coast downhill, but initial early successes were a key motivating factor for JB.

The next strategy was to remove the environmental toxins, including cleaning supplies, personal care products, cigarettes and plastics from their home. They purchased water and air filters for their apartment and switched to glass containers. The Teflon cookware was replaced with stainless steel.

JB's treatment plan was carefully crafted so that he wouldn't lose weight too quickly since toxicants are stored in fat and may be released and overload detoxification pathways. Avoidance of toxics is still the best treatment; however, we began to treat with an array of integrative tools after new habits were being incorporated on a regular basis. JB began to lose weight, his centrally located adiposity decreased, lab tests improved; and not only his mood but his personality and self-confidence began to shift.

Some of the therapeutic strategies utilized included N-acetyl cysteine to increase glutathione production, pectin fiber as a binder, liver/kidney/liver homotoxicologic drainers, as well as vitamins C and D. We eventually added in probiotics, a multivitamin with minerals, omega 3 fatty acids and a constitutional homeopathic remedy which was *Lycopodium* 200c (with a nod to all my homeopathic colleagues). We tried to keep costs down and focus on organic foods as medicines. We explored foods that improve detoxification such as cilantro in his smoothies and foods that improved glucose control such as cinnamon. These simple modalities were tools that their family could easily incorporate without feeling overwhelmed.

After one year, JB had lost 30 pounds and his type 2 diabetes was reversed. His labs completely reverted to normal, and he was discharged from the endocrine clinic. Attempts to talk to his doctors about which strategies worked for JB were unsuccessful. Although getting Western-minded practitioners, particularly those that are collaborators on our mutual patients, to consider the available science on the effects of environmental toxins and metabolic health is often difficult, it is still worth pursuing. The larger message and mission for all health providers is to work cooperatively to eliminate the root cause and culprit of metabolic chaos, which are often environmental pollutants.

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Dr. Michelle Perro has recently co-chaired and lectured at this year's Environmental Health Symposium in Scottsdale, Arizona. She is presently working with the National Association of Environmental Medicine (https://www.naturopathicenvironment.com) on pediatric guidelines and environmental toxicity.

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Environmental Medicine Update

by Marianne Marchese, ND
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Perchlorate Toxicity – The Curious Case of a 10-Year-Old Boy

Introduction

Perchlorate is a chemical used in fireworks, road flares, explosives, gun powder, adhesives, batteries, and rocket fuel. In the past it was in fertilizer used for tobacco and was used as a medication to treat an over-reactive thyroid gland. It also forms naturally in the environment in some area of the country like western Texas. Perchlorates can also form naturally in the atmosphere, leading to trace levels of perchlorate in precipitation. It can be found in both ground and surface water and the soil. Perchlorates are colorless salts that have no odor. There are five perchlorate salts that are manufactured in large amounts: magnesium perchlorate, potassium perchlorate, ammonium perchlorate, sodium perchlorate, and lithium perchlorate.[1,2] Most people are exposed to perchlorate through food, drinking water, bleach, before and after watching a firework show or lighting off fireworks at home, living near factories that make fireworks and explosive devices, using certain cleaning products and pool chemicals, and through chewing tobacco.[1,2]

Food and water are the main sources of exposure, and it can be found in breast milk. In 2005-2006, the FDA's Total Diet Study revealed that 74% of the 208 foods analyzed had at least one detected perchlorate.[1] Cow milk products, eggs, fruit and dark green vegetables account for most of the food exposure.[2] Drinking water and water used in agriculture is another common source of exposure. In an analysis of drinking water samples from 3,262 residences of National Health and Nutrition Survey (NHANES) subjects, perchlorates were identified in 83% of samples.[2] The current California regulatory standard for perchlorate in water is 6 ppb or ug/L. This standard is based on evidence from a human intervention trial linking perchlorate to decreases in iodide uptake into the thyroid gland and hypothyroidism.

In August 2019 the EPA proposed to establish a maximum contaminant level (MCL) and a health-based maximum contaminant level at 56 ppb. The proposed safe level published by the EPA under the Obama administration was 15 ppb, much lower than the current EPA MCL recommended amount.[3] Since there is no federal acceptable level of perchlorate in the drinking water, it is up to the states to monitor and set maximum contaminant levels. Only a few states have done so, and the levels vary considerably. California and Massachusetts have set the perchlorate MCL at 6 ug/L and 2 ug/L respectively based on research linking that level of exposure to thyroid disease. The following states have set a public health goal: Arizona at 14, New York at 5, Maryland at 1, New Mexico at 1, New Hampshire at 1. Three states have set an enforceable action levels: New York 18, Nevada 18, Texas 4. This means if the action level is exceeded, the water treatment company is obliged to take appropriate action to reduce the concentration below action level.[4]

Perchlorate is rapidly eliminated from the body through the urine with half-times of approximately 8-12 hours in humans. So, one might conclude it can't be detected in the body with such a short half-life. However, recent studies have shown widespread exposure to low levels of perchlorate by the general population, detected via urine test. In almost 2700 participants of the NHANES national survey, the geometric mean in urine was 2.63 ug/L and 3.04 ug/L creatine corrected. Urine is the best method of testing, but it might be detectable for only a few days after exposure because it leaves the body so rapidly. This makes testing difficult to capture the exposure.

The health effects of perchlorate are mainly on the thyroid gland where it inhibits iodine uptake leading to hypothyroidism. It may also affect brain development and cause birth defects and developmental delays.[5,6] It is especially a concern for pregnant women unknowingly exposed through the drinking water as perchlorate is passed in utero affecting fetal health. [5,6] The Agency for Toxic Substances and Disease Registry cite thyroid as the main organ affected by perchlorate exposure as well as hematological, renal, and hepatic effects in humans from oral exposure.[7]

Given the widespread exposure to perchlorate through drinking water and food sources, the prevalence of hypothyroidism, and recent urine studies showing large scale exposure to perchlorate, doctors and other health care providers may want to start screening patients for exposure. It is important to educate patients on avoidance and know how to mitigate the health effects of perchlorate. The following case example highlights perchlorate testing and treatment options.

Case Example

In April of 2019, a 10-year-old boy developed left side, unilateral areola swelling. His mother, a physician, and his pediatrician did a thorough exam and found no mass or breast bud development, no dermatological changes to the area, no signs of an infection, and no lymph node swelling. The area was tender with palpation compared to the right areola, but otherwise it did not hurt. There was no preceding trauma or injury; however, he is an active boy and could have easily bumped the area. Two years prior he had the exact same symptom, left areola swelling. At that time, he was seven years old; and he went to his pediatrician had a complete exam and blood work-up that included CBC, CMP, TSH, FT4, FT3, iron, ferritin, prolactin, FSH, LH, SHBG, testosterone and estradiol. All the tests were normal for a seven-year-old boy and after three-to-four months the symptom eventually self-resolved without any treatment. ➤

Urinary Perchlorate (2011 – 2014)

CAS Number 14797-73-0

Geometric mean and selected percentiles of urine concentrations (in µg/L of creatinine) for the US population from the National Health and Nutrition Examination Survey.

Categories (Survey Years)	Geometric Mean (95% conf. interval)	50th Percentile (95th conf. interval)	75th Percentile (95th conf. interval)	90th Percentile (95th conf. interval)	95th Percentile (95th conf. interval)	Sample Size
Total population (2011 - 2012)	2.96 (2.79-3.14)	3.01 (2.82-3.24)	5.46 (5.11-5.71)	9.11 (8.46-9.78)	12.8 (11.4-14.2)	2467
Total population (2013 - 2014)	2.63 (2.44-2.83)	2.62 (2.40-2.83)	4.76 (4.51-5.08)	7.92 (7.26-8.57)	10.6 (9.43-11.9)	2644
Age 6-11 years (2011 - 2012)	3.89 (3.45-4.39)	4.31 (3.67-4.86)	6.41 (5.88-7.17)	10.6 (8.97-12.8)	14.9 (12.7-18.5)	394
Age 6-11 years (2013 - 2014)	3.37 (2.96-3.84)	3.56 (2.95-4.14)	5.61 (4.95-7.08)	9.38 (7.52-10.6)	12.1 (9.70-21.2)	398
Age 12-19 years (2011 - 2012)	2.77 (2.33-3.30)	2.82 (2.39-3.60)	5.15 (4.28-5.71)	7.54 (6.40-8.68)	10.8 (7.54-13.3)	384
Age 12-19 years (2013 - 2014)	2.86 (2.42-3.37)	2.91 (2.47-3.45)	5.11 (3.96-6.69)	9.43 (7.11-10.9)	13.9 (9.83-24.0)	449
Age 20+ years (2011 - 2012)	2.90 (2.71-3.10)	2.95 (2.75-3.14)	5.30 (4.94-5.73)	9.16 (8.46-10.3)	12.8 (11.2-14.6)	1689
Age 20+ years (2013 - 2014)	2.53 (2.34-2.73)	2.44 (2.25-2.76)	4.57 (4.22-4.93)	7.67 (6.94-8.29)	10.2 (8.93-11.7)	1797
Males (2011 - 2012)	3.32 (3.05-3.61)	3.43 (3.22-3.67)	5.79 (5.45-6.58)	10.3 (8.70-10.8)	13.3 (11.7-14.6)	1251
Males (2013 - 2014)	2.73 (2.47-3.03)	2.85 (2.58-3.13)	4.84 (4.53-5.33)	8.18 (6.97-9.43)	11.0 (9.87-13.4)	1313
Females (2011 - 2012)	2.65 (2.44-2.87)	2.64 (2.40-2.96)	4.86 (4.20-5.52)	8.21 (7.78-8.86)	11.7 (10.6-13.2)	1216
Females (2013 - 2014)	2.53 (2.36-2.70)	2.40 (2.21-2.62)	4.67 (4.14-5.21)	7.81 (7.00-8.45)	9.83 (8.73-11.7)	1331

Urinary Perchlorate (creatinine corrected) (2011 – 2014)

CAS Number 14797-73-0

Geometric mean and selected percentiles of urine concentrations (in µg/g of creatinine) for the US population from the National Health and Nutrition Examination Survey.

Categories (Survey Years)	Geometric Mean (95% conf. interval)	50th Percentile (95th conf. interval)	75th Percentile (95th conf. interval)	90th Percentile (95th conf. interval)	95th Percentile (95th conf. interval)	Sample Size
Total population (2011 - 2012)	3.38 (3.17-3.60)	3.28 (2.99-3.60)	5.55 (5.21-5.92)	9.20(8.31-10.1)	14.7 (11.1-17.4)	2465
Total population (2013 - 2014)	3.04 (2.80-3.30)	2.98 (2.76-3.24)	4.89 (4.56-5.37)	7.96 (7.25-8.67)	10.7 (9.68-12.4)	2643
Age 6-11 years (2011 - 2012)	5.56 (5.05-6.12)	5.25 (4.75-5.95)	8.57 (7.57-9.95)	14.3 (10.4-17.4)	18.0 (13.8-27.3)	393
Age 6-11 years (2013 - 2014)	5.06 (4.43-5.76)	4.83 (3.90-5.66)	7.71 (6.31-9.13)	11.0 (9.14-15.4)	15.4 (11.5-28.3)	398
Age 12-19 years (2011 - 2012)	2.70 (2.50-2.91)	2.87 (2.46-3.22)	3.92 (3.80-4.14)	5.73 (4.55-7.76)	8.14 (5.97-8.43)	384
Age 12-19 years (2013 - 2014)	2.59 (2.32-2.89)	2.41 (2.05-2.74)	4.06 (3.34-4.56)	6.33 (4.96-8.11)	8.66 (7.05-12.0)	449
Age 20+ years (2011 - 2012)	3.31 (3.07-3.56)	3.16 (2.85-3.54)	5.53 (5.16-5.88)	9.16 (8.03-10.2)	14.8 (10.1-18.8)	1688
Age 20+ years (2013 - 2014)	2.95 (2.72-3.20)	2.92 (2.72-3.19)	4.71 (4.45-5.21)	7.59 (6.77-8.35)	10.4 (9.02-11.7)	1796
Males (2011 - 2012)	3.12 (2.84-3.42)	2.99 (2.64-3.34)	4.84 (4.41-5.41)	8.38 (7.08-9.92)	14.7 (9.56-20.0)	1250
Males (2013 - 2014)	2.74 (2.51-2.99)	2.65 (2.32-2.91)	4.48 (4.00-4.81)	7.09 (6.32-8.25)	10.6 (8.16-13.1)	1312
Females (2011 - 2012)	3.64 (3.41-3.89)	3.61 (3.18-3.96)	6.10 (5.66-6.45)	9.86 (8.80-10.9)	14.7 (11.6-17.7)	1215
Females (2013 - 2014)	3.36 (3.08-3.67)	3.36 (3.06-3.60)	5.54 (4.86-5.92)	8.59 (7.75-9.45)	11.4 (10.0-13.1)	1331

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Now at age 10 he once again developed the same areola swelling and as stated above was examined by both his pediatrician and mother who is a physician. He is 80-85% for his height, 60-65% for his weight and physical exam was unremarkable. He eats a primarily organic diet, including some meat and dairy, and is gluten free. He eats organic fruits and vegetables daily, and on school days drinks veggie/fruit/protein smoothie with his breakfast. The drinking and cooking water were filtered with reverse osmosis, which can remove perchlorate, and there is a whole house carbon water filter in place. Basic lab tests were all normal and included CBC, CMP, TSH, FT4, FT3, iodine, estradiol, and ESR. An in-depth environmental medicine history revealed household mold exposure for years (from age 4-7). The mold exposure was a slow leak in a bathroom with visible mold. This was remediated in 2017. On February 24, 2019, prior to the onset of the symptom, the patient had a mycotoxin test done, which was negative.

Since the basic labs and exam were normal and this was the second time this symptom had appeared, toxin profiles and immune profiles were ordered to look for potential environmental sources of hormone modulation. A May 19, 2019, organic acid test was normal, and a toxicant panel test revealed *a perchlorate level of 152 ug/g creatine.* Based on the NHANES reference ranges listed above for this age group, anything above 15 ug/g creatine is in the 95% percentile. His urine level was extremely elevated. Although it is not known for perchlorate to cause areolar swelling, it was the only test that came back positive. Perchlorate is a known hormone disruptor mostly affecting thyroid function, yet this patient's thyroid function at this time was normal.

Given the very high level of perchlorate in his urine, we looked for a source of exposure and started treatment to mitigate the effects of perchlorate exposure. The initial treatment consisted of avoiding the unfiltered water at his school and taking three supplements: cholestyramine for past mold exposure, a supplement that provided cofactors to support liver phase one and two metabolic reactions, and an herbal product to cleanse the liver and gall bladder that consisted of milk thistle, beet root, dandelion, burdock and artichoke. The cofactor support product contained vitamin A, vitamin D3, vitamin K1, vitamin B1, vitamin B2, vitamin B3, vitamin B5, vitamin B6, vitamin B12 (as methylcobalamine), vitamin C, vitamin E, biotin, folate (5-methyl-tetrahydrofolate), calcium, chromium, copper, iodine, magnesium, manganese, molybdenum, potassium,

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In terms of a source of perchlorate exposure, he mostly eats organic, does not use bleach, was not around fireworks and his home water is filtered with reverse osmosis. He was drinking the water at school which was unfiltered. Due to several factors it took four months for the water to be tested. A home and school water test were run through National Testing laboratory September 2019 and both were normal. The water at home was reverse osmosis and perchlorate was normal at less than 0.05 ug/l. The water at school was unfiltered and was 0.91 ug/l. These levels are well below even the strictest minimal acceptable level in Massachusetts of 2 ug/l.

After avoiding the water at school and taking the three supplements, he repeated the urine perchlorate test on October 8, 2019; and the *perchlorate level dropped to 4.8 ug/g creatine.* This is within the NHANES normal reference range of 50% for his age group. But at what level we see health effects remains an area for further investigation. Some studies indicate anything over the 50% percentile will over time lower iodine and lead to thyroid disease.[8] Of course, this patient never had thyroid disease, he had left areola swelling, which resolved by August 2019.

Discussion

This was the first patient I had encountered with elevated perchlorate levels; 152 ug/g creatine is well above all labs reference ranges and NHANES percentiles. This case was challenging because the patient did not have a known source of exposure. Food and water are the most common sources of perchlorate exposure. Both his home and school water were tested and within the normal acceptable levels. Since both water tests were done four months after discovering the elevated urine level, it is theorized that he was exposed to perchlorate through the unfiltered water at school in early 2019 but not captured on the testing done months later. It is the only possible source of exposure.

Another interesting part of this case is the patient's thyroid hormones and iodine levels were normal and his presenting symptom, areola swelling, is not a known common effect of perchlorate. However, in animal studies perchlorate exposure from food and water does cause breast gland dysplasia and other histopathological lesions in the mammary gland.[9] Other studies do show that absorbed perchlorate, regardless of the route of exposure, will distribute to the breast tissue and mammary epithelium. Perchlorate is also detected in human breast milk showing an affinity for breast tissue deposition.[9] Therefore it is theorized that the very high level of perchlorate in the urine of this nine-year boy, 152 ug/g creatine, may have created breast areola swelling. After three-to-four months of avoiding the unfiltered drinking water at school and taking the three supplements described above, the urine perchlorate level was normal, 4.8, and the areola swelling resolved.

Summary

Perchlorate is a hormone-disrupting toxicant that is often overlooked by doctors and health care providers. It is a common contaminate in water and food, and people are exposed to it on a daily basis at low levels. Over time, it is known to disrupt thyroid function and is linked to fetal brain development and other health effects. It is important to educate patients and the public on avoidance and know how to mitigate the health effects of perchlorate.

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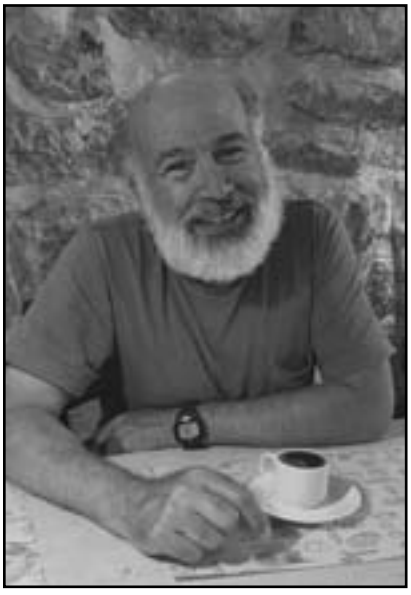

Curmudgeon's Corner

by Jacob Schor, ND, FABNO
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Eggs, Choline, TMAO, and Cardiovascular Disease

A paper published in February 2019 has me rethinking my opinion about eggs and leading me down a rabbit hole of recent research trying to understand how we might better prevent cardiovascular disease. Eggs were originally singled out years ago as a prime suspect in causing heart disease because they contain large amounts of dietary cholesterol. But then they were cleared of blame as researchers repeatedly failed to find a positive association between eggs eaten and disease incidence. Now a new study by Victor Zhong et al has again cast suspicion on eggs.

I originally posted an article suggesting that eggs posed little risk of cardiovascular disease (CVD) to our website in April 2005, over a dozen years ago: http://www.denvernaturopathic.com/news/eggs.html.

I wrote a second article that was published in the *Natural Medicine Journal* in July 2018 (https://www.naturalmedicinejournal.com/journal/2018-07/egg-consumption-may-lower-risk-cardiovascular-disease), reviewing the research by Qin et al, which actually concluded that egg consumption was associated with a significantly lower risk of cardiovascular disease.[1]

This new Zhong study contradicts the earlier information that we thought had answered this question with a degree of certainty. In the Zhong study, data was pooled from half a dozen prospective cohort studies done in the United States with a median follow up of 17.5 years. Taken together, there were 29,615 adults followed in their research. Each addition of 300 mg of dietary cholesterol eaten per day, that's about two eggs worth, significantly raised CVD risk by 17%. This same amount of cholesterol increased risk of all-cause mortality by 18%.[2] This is not what we were expecting to read.

What should we do with this information? We could just ignore it and assume it's wrong. Observational studies such as those used to source these data are prone to error. The data on egg consumption was only gathered at the start of the studies by having participants fill out questionnaires. Such data are less accurate than most researchers like, and inaccurate starting data could have led to inaccurate outcome results. That would be the easy way to deal with ideas we don't like. Another approach is to reconsider our starting assumptions and see if they still seem reasonable.

The initial idea that dietary cholesterol consumption was linked with heart disease risk came out of Framingham Study. Eggs are an excellent source of dietary cholesterol and so were early on identified as suspects that could increase risk of cholesterol deposits leading to plaque formation. Eggs are also an excellent source of dietary choline, in particular phosphatidylcholine; but no one was thinking about choline back then. The role phosphatidylcholine and dietary choline play in cardiovascular disease has only recently begun to be appreciated.

In 2009 two Swedish scientists, de Faire and Frostegård, reported that they had studied the immunoglobulins that target phosphatidylcholine and their association with CVD. Such immunoglobulins are protective for individuals with high blood pressure reducing their risk of heart disease. At the time, these Swedes were experimenting with what we might describe as phosphatidylcholine vaccines to prevent or treat atherosclerotic plaques.[3] By 2010 deFaire's research was suggesting that measuring these anti-PC immunoglobulins might be predictive of risk; people in the lowest quartile of anti-PC antibodies were at nearly double the risk for heart disease of those in the highest quartile. Did I get that right? The antibodies are good because they lower PC.[4] As a side note, low levels of PC antibodies also increase risk of stroke.[5]

Skip ahead a handful of years and the mechanism of action to explain phosphatidylcholine's role in increasing CVD is clearly established. The explanation is that bacteria in the large bowel

convert phosphatidylcholine (and also the choline-containing compounds betaine and l-carnitine) into trimethylamine (TMA), which is then oxidized in the liver to trimethylamine n-oxide (TMAO). This chemical TMAO has a pronounced atherosclerotic effect.[6,7] The degree phosphatidylcholine (and also betaine and l-carnitine) is converted to TMA and eventually TMAO varies with the types of bacteria living in the colon.

Eggs are our highest dietary source of choline, about 126 mg/egg. Other foods also high in choline include peanuts, beets, meat, and fish.[8] Few of us would group these together as heart risky foods, and we could imagine that studies that examined dietary data for disease associations would have easily overlooked choline intake and failed to associate levels with risk.

The obvious assumption would be that cutting back on dietary choline and other substrates for TMAO would lower CVD risk. Given that choline is an essential nutrient, we have to wonder whether reducing dietary intake would be worth the risk of deprivation and possible deficiency. More important it is unclear whether choline restriction will even have the desired effect reducing TMAO levels.

There is another source of phosphatidylcholine that gives me pause as I read these studies. Many of the liposomal encapsulated supplements use phosphatidylcholine to form the micellular nano-particles. My favorite brand of curcumin certainly does, and I've taken this myself, not to mention sold a fair bit to patients. These liposomal capsules deliver daily levels of phosphatidylcholine that are no different from what a person obtains from eating a dozen eggs a week. We frequently have suggested l-carnitine and beet powder to patients with heart problems. Could we have done a disservice to patients by inadvertently increasing their risk for CVD?

So far, the research hasn't supported such concerns; choline depletion doesn't appear to lower TMAO levels. Lemos et al in their 2018 study found that dietary choline has little impact; TMAO levels remained the same in study participants who ate three eggs, or took 400 mg of choline a day, compared to people who received no choline.[9] A 2017 meta-analysis that looked at dietary choline and CVD did not find any association with disease or mortality. The authors had identified six studies, comprising 18,076 incident CVD events, 5343 CVD deaths, and 184,010 total participants. Incident CVD was not associated with choline or betaine intake. A separate 2017 trial found that eating three eggs a day, while it did increase plasma choline, still did not affect TMAO levels.[10] Thus, while the idea that eating eggs or swallowing those liposomal phosphatidylcholine-containing supplements might increase TMAO seems logical, the evidence suggests we need not worry, at least for the moment.[11] It's all about the colonic bacteria and whether they convert the choline to TMA or not.

Various diet, nutrient and drug approaches to block the bacterial fermentation processes that produce TMA and then TMAO are being considered.[12,13] Interventions as simple as caloric restriction in combination with exercise reduce TMAO production. Reducing visceral adiposity also reduces production.[14] A day-long water fast drops TMAO levels by almost half.[15] Reducing red meat consumption lowers TMAO production, though this may be the result of reduced l-carnitine, another precursor to bacterial production, or just as likely, it shifts bacterial populations.[16] Two contradictory papers published in Spring 2019 informed us that adherence to a Mediterranean style diet may[17] or may not lower TMAO.[18]

The bottom line is that TMAO concentrations seem to be little changed by what someone eats.[19] Egg, fish, or meat consumption did not affect TMAO in one study, though dairy still may increase it.[20]

At the same time, nothing in the current literature suggests that we should discount the phosphatidylcholine-TMAO connection to heart disease. In fact, each new publication makes the theory all the more compelling.

A 2018 meta-analysis reported a significant association between TMAO levels and cardiac events and overall mortality. "Higher circulating TMAO was associated with a 23% higher risk of CVEs [cardiac vascular events] …and a 55% higher risk of all-cause mortality…"[21]

A January 2019 Chinese study comparing urinary TMAO levels reported patients in the upper quartile were nearly twice as likely to suffer from coronary heart disease than patients in the lower quartile. Diabetic patients whose TMAO was above the mean group were six-fold more likely to suffer from coronary heart disease than those below the mean.[22]

TMAO impacts a range of other conditions including cancer and diabetes.[23] The consensus is that if this pathway can be modulated, we could see wide ranging benefits. Modulation of the pathway should be possible through shifting the gut biome.

Granted that the impact of the gut microbiome is the fad of the moment and is now getting blamed for all things that might ail humans. In the case of CVD, this is possibly true; the biome research published in the last few years is compelling.

Specific families of bacteria are now associated with both higher heart disease risk and other families of bacteria are associated with lower risk. Some of the data are troubling as they don't support our routine generalizations of which bacteria are good or bad; some of our probiotic favorites such as *Lactobacilli* and *Bifidobacteria* are associated with increased heart disease risk.[24] (And while you might think so, that was not a typo.) Obscure bacteria such as *Bacteroides vulgatus* and *Bacteroides dorei* are associated with lower risk. These are not sold at your local health food store. Cardiac patients who consume 'resistant starches' do however increase levels.[25,26]

Fecal transplantation in mice can transfer atherosclerotic propensities.[27] Antibiotic treatments have then countered these tendencies. Recent research has sought to identify bacteria species that might serve as probiotics to lower conversion of choline to trimethylamine n-oxide.[28,29]

The Cleveland Heart Lab now tests for trimethylamine n-oxide (TMAO).[30] ARUP Laboratories offers testing for phosphatidylcholine antibodies.[31] With easy testing we see a growing list of studies that test various interventions that impact TMAO.

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Nora Kalagi and colleagues in Australia conducted a systematic review of these trials, which was published in May 2019: "A spectrum of antibiotics and other therapeutic strategies have been employed to test their potential to modulate TMAO concentrations, assuming the gut microbiome to be the key source of TMAO." Their paper provides the most comprehensive list of potential interventions seen to date.[32]

What's fascinating is that much of the findings contradict our starting assumptions of what is good and what is bad for heart disease.

Three studies have assessed the effect of taking metformin on TMAO levels and reported inconsistent results. TMAO levels increased in two studies, Huo in 2009 and Cadeddu et al in 2013.[33,34] Yet metformin had no effect for Velebova et al's study in 2016.[35]

Because they shut down bacterial conversion, antibiotics certainly lower TMAO, at least in the short term. In one study, TMAO levels increased in forty healthy subjects who ate two hard boiled eggs per day but when the subjects were pretreated for one week with a combination of metronidazole and ciprofloxacin, TMAO was undetectable when the same egg challenge was repeated.[36] There's a US patent for using enteric coated aspirin accompanied by a claim that it significantly reduces TMAO levels in study participants also eating two hard boiled eggs per day.[37] In these few studies, the common denominator is that eating hard boiled eggs is a bad idea.

A January 2019 publication in *Nutrients* reported that a grape pomace, which supplies resveratrol, lowered TMAO levels in humans by about 10% compared to placebo.[38] Another study published in the same issue reported that a fermented apple pomace reduced TMAO by 63%.[39]

There is a traditional food eaten in Greenland that is made by fermentation of shark meat. This Greenland Shark's flesh is toxic because it contains exceptionally high TMAO levels and also triethylamine (TMA) that occur naturally in the animal. Yet "hákarl" the traditional fermented shark meat preparation eaten in Greenland is apparently safe for humans; the fermentation process reduces the toxic levels of these constituents rendering the shark meat safe.[40] Scientific details aside, I suspect this food is still an acquired taste.

Oral berberine, long employed to shift gut biota, reduces TMAO production in mice[41] and also increases *Akkermansia* levels.[42] Specific strains of *Lactobacilli plantarum* have been reported to reduce TMAO production, also in mice.[43] Treating aging mice with antibiotics lowered their gut microbiome populations, reduced TMAO levels and improved endothelial function.[44] Inulin has been tried but failed to reduce TMAO levels in "… sedentary, overweight/obese adults at risk for T2DM" (fat human pre-diabetics).[45] Eating a Mediterranean diet also failed to change TMAO production.[46]

If dietary intake of choline has less impact on TMAO production than what types of bacteria are fermenting the choline, then perhaps worrying about dietary choline is wasted effort.

What one eats today may have little impact on TMAO production compared to what you were eating yesterday and the weeks leading up to the 'study.' Long-term dietary patterns may shift the gut biome. For example, a study published this past January fed radioactive tagged l-carnitine, (l-carnitine is also linked to increased TMAO levels via bacterial fermentation as choline is) to 32 vegetarians and 40 omnivores. "The transformation into atherosclerotic compounds occurred in both groups but occurred to a 'markedly lower extent, in vegans/vegetarians.'"[47]

So back to eggs. The truth is I don't know if we should worry about them or not. In a trial of fifty people by Missimer et al published in 2018, eating two eggs per day was more effective than oatmeal at lowering CVD markers and made no difference in TMAO levels.[48]

Even if TMAO is ultimately proven a significant problem, to date we aren't seeing strong evidence that getting excessive dietary choline makes a difference. If I had to make a guess, my prediction is that certain people because of their gut biome have a greater tendency to produce TMAO; and for them, the unlucky among us, choline and phosphatidylcholine consumption might play an undesirable role. How should we identify them? Perhaps by testing. We could use TMAO serum levels to screen and identify patients who might benefit from choline restriction and then watch individually if restriction lowers their TMAO. We also might try 'rearranging' their gut biome and see if that has an effect. Should we worry about eggs, liposomal supplements, or for that matter, l-carnitine or even beets? Perhaps, but at this point there are plenty of better things that deserve our attention.

In an ideal world, researchers will identify a strain of bacteria that lowers TMAO production and then really clever individuals will realize that these same bacteria are already present in some desirable food stuff, for example in blue cheese, and our problems will be solved. Add that to my list of ideal scenarios in the ideal world of my imagination.

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Curmudgeon's Corner

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Nutritional Treatment of Coronavirus

by Andrew W. Saul, Editor
Orthomolecular News Service

Abundant clinical evidence confirms vitamin C's powerful antiviral effect when used in sufficient quantity. Treating influenza with very large amounts of vitamin C is not a new idea at all. Frederick R. Klenner, MD, and Robert F. Cathcart, MD, successfully used this approach for decades. Frequent oral dosing with vitamin C sufficient to reach a daily bowel tolerance limit will work for most persons. Intravenous vitamin C is indicated for the most serious cases.

Bowel tolerance levels of vitamin C, taken as divided doses all throughout the day, are a clinically proven antiviral without equal. Vitamin C can be used alone or right along with medicines if one so chooses.

Dr. Robert Cathcart advocated treating influenza with up to 150,000 milligrams of vitamin C daily, often intravenously. You and I can, to some extent, simulate a 24 hour IV of vitamin C by taking it by mouth very, very often. When I had pneumonia, it took 2,000 mg of vitamin C every six minutes, by the clock, to get me to saturation. My oral daily dose was over 100,000 mg. Fever, cough and other symptoms were reduced in hours; complete recovery took just a few days. That is performance at least as good as any pharmaceutical will give, and the vitamin is both safer and cheaper. Many physicians consider high doses of vitamin C to be so powerful an antiviral that it may be ranked as a functional immunization for a variety influenza strains.[1]

Dr. Cathcart writes:

> The sicker a person was, the more ascorbic acid they would tolerate orally without it causing diarrhea. In a person with an otherwise normal GI tract when they were well, would tolerate 5 to 15 grams of ascorbic acid orally in divided doses without diarrhea. With a mild cold 30 to 60 grams; with a bad cold, 100 grams; with a flu, 150 grams; and with mononucleosis, viral pneumonia, etc. 200 grams or more of ascorbic acid would be tolerated orally without diarrhea. The process of finding what dose will cause diarrhea and will eliminate the acute symptoms, I call titrating to bowel tolerance.
>
> The ascorbate effect is a threshold effect. Symptoms are usually neutralized when a dose of about 90% or more of bowel tolerance is reached with oral ascorbic acid. Intravenous sodium ascorbate is about 2½ times more powerful than ascorbic acid by mouth and since for all practical purposes huge doses of sodium ascorbate are non-toxic, whatever dose necessary to eliminate free radical driven symptoms should be given.

The coronavirus, in acute infections, may be expected to be just as susceptible to vitamin C as all of the other viruses against which it has been proven to be extremely effective. There has never been a documented situation in which sufficiently high dosing with vitamin C has been unable to neutralize or kill any virus against which it has been tested.

Even the common cold is a coronavirus. A "new" opportunistic virus is a not a big surprise. History is full of them.

Flu Pandemic of 1919-1920

About 10 million soldiers were killed in World War I (1914-1918), charging machine guns and getting mowed down month after month. There were nearly a million casualties at the Somme and another million at Verdun. A terrible slaughter went on for four years. Yet, in just the two years following the war, over 20 million people died from influenza. That is more than twice as many deaths from the flu in one-half the time it took the machine guns.

With a century's worth of accumulated scientific hindsight, we must today ask this: Was a lack of vaccinations really the cause of those flu deaths, or was it really wartime stress, and especially war-induced malnutrition, that set the stage in 1918? And now, once again, we have an alarming and rather similar scenario: between nutrient-poor processed convenience foods, McNothing meals and TV news scare stories, we have the basic ingredients for an epidemic.

Influenza is a serious disease, and historically, has been the Reaper's scythe. There is no way to make light of that. It warrants a closer look at how the medical profession and government have approached different types of influenza.

Swine Flu

In the mid-1970s, there was the colossal swine flu panic. Here is what the government of the United States said about the infamous swine flu vaccine, in a 1976 mass-distributed FDA Consumer Memo on the subject: "Some minor side effects - tenderness in the arm, low fever, tiredness - will occur in less than 4% of (vaccinated) adults. Serious reactions from flu vaccines are very rare."

Many will remember the very numerous and very serious side effects

of swine flu vaccine that forced the federal immunization program to a halt. So much for blanket claims of safety.

As far as being essential, in the same memo the FDA said this of the same vaccine:

"Question: What can be done to prevent an epidemic? Answer: The only preventive action we can take is to develop a vaccine to immunize the public against the virus. This will prevent the virus from spreading."

This was seen to be totally false. The public immunization program for swine flu was abruptly halted and still there was no epidemic. If vaccination were the only defense, one might expect that tens of millions of Americans would have been struck down with the swine flu, for a large percentage of the population of the US was not vaccinated.

> "Vaccines are being used as an ideological weapon. What you see every year as the flu is caused by 200 or 300 different agents with a vaccine against two of them. That is simply nonsense." – Tom Jefferson, MD, epidemiologist[2]

Bird Flu

Robert F. Cathcart, MD, writes:

> Treatment of the Bird Flu with massive doses of ascorbate would be the same as any other flu except that the severity of the disease indicates that it may take unusually massive doses of ascorbic acid orally or even intravenous sodium ascorbate. (Why the dose needed is somewhat proportional to the severity of the disease being treated is discussed in my paper published in 1981, Titrating to Bowel Tolerance, Anascorbemia, and Acute Induced Scurvy.) I have not seen any flu yet that was not cured or markedly ameliorated by massive doses of vitamin C but it is possible that the bird flu may require even higher doses such as 150 to 300 grams a day. Additionally, this flu could be primarily respiratory. This means that hospitalization might be necessary. If massive doses of ascorbate are not used, they may not be adequate. Most hospitals will not allow adequate doses of ascorbate to be given.
>
> Initial oral doses of ascorbic acid should also be massive. I would suggest like 12 grams every 15 minutes until diarrhea is produced. Then, however, doses should be reduced but not much. Listen to your body. If there are many symptoms, keep taking doses that cause a little diarrhea. You do not want constant runs because it is the amount you absorb that is important, not the amount you put in your mouth.[1,3]

SARS

The coronavirus outbreak in China seems to be due to a virus similar to SARS (Severe Acute Respiratory Syndrome), which was also a coronavirus. You may remember SARS from 2002. I most certainly do, as I was in Toronto, Canada, at the time, smack in the middle of it. I took a lot of vitamin C preventively and had zero symptoms.[4]

Waiting for a Vaccine?

"We have set up a situation where a fear is created, and then we try to create the treatment for this fear. The public gets the idea that the flu is going to kill them and the vaccine will save them. Neither is true." (Marc Siegel, MD, author of *False Alarm: The Truth About the Epidemic of Fear*)[2]

Robert F. Cathcart wrote:

> All this talk about a vaccine is too late; a waste of time now, especially when we know how to cure the disease already. Every flu I have seen so far (since 1970) has been cured or ameliorated by massive doses of ascorbate. All of these diseases kill by way of free radicals. These free radicals are easily eliminated by massive doses of ascorbate. This is

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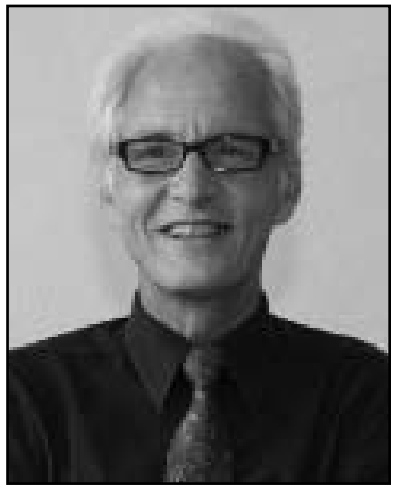
Paradigm, Practice, and Policy Advancing Integrative Health



The Journal of Alternative and Complementary Medicine is pleased to announce that John Weeks, *JACM* Editor in Chief, was the recipient of the 2020 Leadership Award from the Integrative Healthcare Symposium (IHS). The award honored and acknowledged Weeks' many accomplishments over the course of his 36-year career as an organizer, chronicler, executive, columnist, and editor in the field of integrative health and medicine. The Symposium is the largest annual conference in the field, typically drawing 1200-1600 attendees.

Weeks was presented the award on February 21st by his colleague of 20 years, long-time integrative medicine leader Ben Kligler, MD, MPH, a past chair of the Academic Consortium for Integrative Medicine and Health who presently serves as acting director of the Veterans Administration "Whole Health" initiative through its Office of Patient Center Care and Cultural Transformation. Kligler says of Weeks: "John has played a critical and unique role in moving integrative healthcare forward over the past three decades. His commitment, his insight, and his (sometimes brutal) honesty have kept us on track as a movement and continue to push us toward new and innovative ways of thinking and doing as we continue to grow our influence and impact on American health care."

After the award presentation, Weeks delivered a keynote entitled "Hope Is a Verb: 50 Years in the Furrows for Integrative Health and Medicine."

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a matter of chemistry, not medicine. The time has come to stop hiding our ability to treat these acute infectious diseases with massive doses of ascorbate.

Ideally, however, in serious cases this disease should be treated first with at least 180 grams or more of sodium ascorbate intravenously every 24 hours running constantly until the fever is broken and most of the symptoms are ameliorated. If after a few hours that rate of administration does not have an obvious ameliorating effect, the rate should be increased.[5]

What Dosage?

Vitamin C fights all types of viruses. Although the dose should truly be high, even a low supplemental amount of vitamin C saves lives. This is very important for those with low incomes and few treatment options. For example, in one well-controlled, randomized study, just 200 mg/day vitamin C given to the elderly resulted in improvement in respiratory symptoms in the most severely ill, hospitalized patients. And there were 80% fewer deaths in the vitamin C group.[6]

But to best build up our immune systems, we need to employ large, orthomolecular doses of several vital nutrients.[7,8] The physicians on the *Orthomolecular Medicine News Service* review board specifically recommend at least 3,000 milligrams (or more) of vitamin C daily, in divided doses. Vitamin C empowers the immune system and can directly denature many viruses. It can be taken as ascorbic acid (which is sour like vinegar), either in capsules or as crystals dissolved in water or juice. It can also be taken as sodium ascorbate, which is non-acidic. To be most effective, it should be taken to bowel tolerance. This means taking high doses several (or many) times each day.

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Nebulized Hydrogen Peroxide

Thomas E. Levy, MD, wrote:

> Viral syndromes start or are strongly supported by ongoing viral replication in the naso- and oropharynx. When appropriate agents are nebulized (into a fine spray) and this viral presence is quickly eliminated, the rest of the body "mops up" quite nicely the rest of the viral presence. The worst viral infections are continually fed and sustained by the viral growth in the pharynx. Probably the best and most accessible agent to nebulize would be 3% hydrogen peroxide for 15 to 30 minutes several times daily.[9]

An example of successful treatment by ascorbate:

> Chikungunya is a viral illness characterized by severe joint pains, which may persist for months to years. There is no effective treatment for this disease. We treated 56 patients with moderate to severe persistent pains with a single infusion of ascorbic acid ranging from 25-50 grams and hydrogen peroxide (3 cc of a 3% solution) from July to October 2014. Patients were asked about their pain using the Verbal Numerical Rating Scale-11 immediately before and after treatment. The mean Pain Score before and after treatment was 8 and 2 respectively (60%) ($p < 0.001$); and 5 patients (9%) had a Pain Score of 0. The use of intravenous ascorbic acid and hydrogen peroxide resulted in a statistically significant reduction of pain in patients with moderate to severe pain from the Chikungunya virus immediately after treatment.[10]

Additional Recommended Nutrients

Magnesium: 400 mg daily (in citrate, malate, chelate, or chloride form). Many people are deficient in magnesium because modern agriculture often does not supply adequate magnesium in the soil, and food processing removes magnesium. It is an extremely important nutrient that is essential for hundreds of biochemical pathways. A blood test for magnesium cannot correctly diagnose a deficiency. A long-term deficiency of magnesium can build up in the body that may take six months to a year of higher than normal doses to replete.

A very cheap and highly beneficial adjunct for any acute infection, especially viral, is oral magnesium chloride. Amazingly, just as intravenous vitamin C has been shown to cure polio, an oral magnesium chloride regimen has been shown to do the same thing, as or even more effectively than the vitamin C.[11-13]

Mix 25 grams $MgCl_2$ in a quart of water. Depending on body size (tiny infant to an adult), give 15 to 125 ml of this solution four times daily. If the taste is too salty/bitter, a favorite juice can be added.

Vitamin D3: 2,000 International Units daily. (Start with 5,000 IU/day for two weeks, then reduce to 2,000). Vitamin D is stored in the body for long periods but takes a long time to reach an effective level. If you are deficient (e.g. if you haven't taken vitamin D and it's near the end of winter when the sun is low in the sky) you can start by taking larger than normal doses for 2 weeks to build up the level quickly. The maintenance dose varies with body weight, 400-1000 IU/day for children and 2000-5000 IU/day for adults.

William Grant, PhD, says: "Coronaviruses cause pneumonia as does influenza. A study of the case-fatality rate from the 1918-1919 influenza pandemic in the United States showed that most deaths were due to pneumonia. The SARS-coronavirus and the current China coronavirus were both most common in winter, when vitamin D status is lowest."[14-18]

Zinc: Zinc is a powerful antioxidant and is essential for many biochemical pathways. It has been shown to be effective in helping the body fight infections.[19,20] A recommended dose is 20-40 mg/day for adults.

Selenium: 100 mcg (micrograms) daily. Dr. Damien Downing says:

> Swine flu, bird flu and SARS (another coronavirus) all developed in selenium-deficient areas of China; Ebola and HIV in Selenium-deficient areas of Sub-Saharan Africa. This is because the same oxidative stress that causes us inflammation forces viruses to mutate rapidly in order to survive. "When Se-deficient virus-infected hosts were supplemented with dietary Se, viral mutation rates diminished and immunocompetence improved."[21]

B-complex vitamins and vitamin A: A multivitamin tablet *with each meal* will supply these conveniently and economically.

Nutritional supplements are not just a good idea. For fighting viruses, they are absolutely essential.

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CALENDAR

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APRIL 23-26: ACUPUNCTURE MERIDIAN ASSESSMENT (AMA) TRAINING For Doctors, Dentists & Health Professionals: Detecting Parasites, Dental & Fungal in St. Louis, Missouri. Simon Yu, MD, CONTACT: www.preventionandhealing.com**.** 314-432-7802.

APRIL 25-27: AMERICAN ASSOCIATION OF NATUROPATHIC PHYSICIANS DC FLI – Leadership and Advocacy Training in Washington, DC. CONTACT: https://naturopathic.org/page/2020FLI

APRIL 27-29: FREQUENCY SPECIFIC MICROCURRENT CORE MODULE 2 – NEURO & VISCERAL in Denver, Colorado. Also, **JULY 10-12** in Philadelphia, Pennsylvania; **NOVEMBER 6-8** in Chicago, Illinois; **DECEMBER 3-5** in San Francisco, California. CONTACT: 360-695-7500; https://frequencyspecific.com/

APRIL 28-MAY 1: INTEGRATIVE CONGRESS ON INTEGRATIVE MEDICINE AND HEALTH in Cleveland, Ohio. CONTACT: http://www.icimh.org/#home

MAY 1: LYME DISEASE, TICK-BORNE ILLNESS, & MENTAL ILLNESS with Robert Bransfield, MD, in Easton, Maryland. CONTACT: 410-726-4573; https://www.eventbrite.com/e/lyme-disease-tick-borne-illnesses-and-mental-health-tickets-69337717981

MAY 2-3: MORPHOGENIC FIELD TECHNIQUE SEMINAR FOR BIOTICS (INTERMEDIATE/ADVANCED) in Waltham, Massachusetts. CONTACT: https://www.morphogenicfieldtechnique.com/

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MAY 14-16: AMERICAN ACADEMY OF OZONOTHERAPY ANNUAL MEETING in Denver, Colorado. CONTACT: 1-888-991-2268; https://aaot.us/?

MAY 14-16: 28th ANNUAL A4M/MMI SPRING CONFERENCE in Orlando, Florida. CONTACT: 561-997-0112; https://www.a4m.com/

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MAY 15-17: FREQUENCY SPECIFIC MICROCURRENT CORE MODULE 1 – PAIN/INJURY MODULE in Raleigh-Durham, North Carolina. Also, **SEPTEMBER 18-20** in Chicago, Illinois; **OCTOBER 16-18** in Anaheim, California.; **DECEMBER 6-8** in San Francisco, California. CONTACT: 360-695-7500; https://frequencyspecific.com/

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MAY 20-24: AUTISM-ONE CONFERENCE – Where Science, Hope, and Recovery Meet in Chicago, Illinois. CONTACT: https://autismoneconference.com/

MAY 22-24: GENETIC METHYLATION SERIES 1 with Marc Harris, MD, ND, in Hermosa Beach, California. Also, **OCTOBER 23-25** in Chicago, Illinois; **DECEMBER 4-6** in Las Vegas, Nevada. CONTACT: 800-890-4547.

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Women's Health Update

by Tori Hudson, ND
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Hawthorne and the Heart: Modern Research on a Medicinal Tree

Introduction

Cardiovascular disease is the most common cause of office visits, hospitalizations, and death in the US. About 647,000 people die per year of heart disease, one in every four.[1] Heart disease is the leading cause of death for both men and women.

As a whole, it is the leading cause of death in women worldwide. You might be surprised to know that more women die from CVD than from all cancers, tuberculosis, HIV/AIDS, and malaria combined. In the United States, rates of CVD are declining due to prevention, diagnosis, and treatment; but still, in 2014, over 399,000 women died from heart disease, which accounted for half of all deaths in women. Regardless of ethnicity, one in three women will die of heart disease in the US, and the death rates from CVD in women exceed those of men.

From the CDC: Heart disease is the leading cause of death for people of most racial and ethnic groups in the United States, including African American, American Indian, Alaska Native, Hispanic, and white men. For women from the Pacific Islands and Asian American, American Indian, Alaska Native, and Hispanic women, heart disease is second only to cancer.[1]

Below are the percentages of all deaths caused by heart disease in 2015, listed by ethnicity, race, and sex.[1]

Percentages of all deaths caused by heart disease in 2015 by ethnicity, race, and sex.

Race of Ethnic Group	% of Deaths	Men, %	Women, %
American Indian or Alaska Native	18.3	19.4	17.0
Asian American or Pacific Islander	21.4	22.9	19.9
Black (Non-Hispanic)	23.5	23.9	23.1
White (Non-Hispanic)	23.7	24.9	22.5
Hispanic	20.3	20.6	19.9
All	23.4	24.4	22.3

Practitioners of all philosophies and training must advance their skills in assessing risk factors, diagnostic work-ups, prevention strategies, and therapeutic interventions. While the list of lifestyle and natural therapies pertinent to cardiovascular issues is long with standouts such as smoking cessation, weight management, stress reduction, Mediterranean diet, fish oils, niacin, co-enzyme Q10 and so many more, one botanical with a rich historical tradition as well as modern research is worth a studious look to further understand how and when to best use it, as well as its limitations.

Hawthorn (*Crataegus oxyacantha*)

Crataegus species are mostly thorny shrubs and small trees whose berries, flowers, and leaves have been used in traditional herbal medicine throughout Europe and the US for generations and is widely used in Europe as a cardiotonic and for congestive heart failure.

The principle active components in hawthorn leaves, berries, and blossoms are flavonoids. One of these flavonoids, proanthocyanidin, has especially important cardiovascular effects.

Various hawthorn preparations have been researched with the majority using a proprietary preparation from the leaf and flowers (L1 132; WS 1442). Other preparations include leaf and flower combinations with and without the berries, aqueous extracts, methanolic extracts (L1 132; Faros), ethanolic extracts (Esbericard, Crataegutt), dried blossoms and a flavonoid extract (Crataemon).

The exact mechanisms of action for hawthorn and cardiovascular disease is uncertain, but it is thought that the primary activity is its ability to increase coronary arterial blood flow, perhaps due to dilation of the coronary arteries. The inotropic effects may be due to inhibition of myocardial

sodium/potassium ATPase. Hawthorn also appears to slightly increase the strength of the cardiac muscle contractions and decrease blood pressure, resulting in increased exercise tolerance and its role in congestive heart failure. Hawthorn has also been shown to exhibit antioxidant activity in a number of studies, which is likely due to its flavonoid and procyanidin constituents.[2,3,4]

Numerous randomized placebo-controlled clinical trials report hawthorn's ability to improve exercise capacity and alleviate symptoms of cardiac insufficiency, and its effectiveness in the treatment of mild to moderate congestive heart failure (CHF), the area of the most research.

The largest, recent and well known study, "The efficacy and safety of Crataegus extract WS 1442 in patients with heart failure: The SPICE trial," was designed to assess the safety of *WS 1442* and its effects on morbidity and mortality in patients with New York Heart Association (NYHA) class II and III CHF in addition to optimal standard care.[5] The randomized, multicenter, double-blind, placebo-controlled study enrolled 2681 patients and was performed at 156 centers in 13 European countries. Patients with NYHA class II and III CHF and reduced left ventricular function (≤35 %) were randomized to either the study medication (450 mg twice daily) or placebo in addition to conventional treatment for 24 months.

Crataegus extract had no impact on the primary endpoint, (composite endpoint of cardiac mortality, nonfatal myocardial infarction, and hospitalization due to progression of heart failure) with a rate of 27.9% in the treatment group vs 28.9% in the placebo group. In addition, after 24 months, there was no difference in the rates of cardiac mortality or sudden cardiac death between the two groups. However, in a subgroup analysis of patients with a left ventricular ejection fraction ≥ 25%, sudden cardiac death was lower in the WS 1442 group.

A meta-analysis of studies using hawthorn for chronic cardiac failure provided evidence of its efficacy in chronic heart failure.[6] This systematic review of controlled trials revealed that hawthorn extract was significantly more beneficial than placebo for maximal workload. Symptoms of dyspnea and fatigue also decreased significantly with hawthorn compared with placebo.

In 1996, a systematic review was done of seven controlled trials that met the New York Heart Classification (NYHA) Class I or II heart failure.[7] These studies demonstrated clinical improvement with hawthorn extract with decreases in symptoms and objective evidence of efficacy most often proven by exercise ergometry.

Additional randomized studies have also been positive. In a 1974 study, 10 mg hawthorn berry and 30 mg hawthorn leaf was added to a nitrate.[8] In 25% of the patients, the combination drug treatment and hawthorn was superior to drug treatment alone in patients with severe coronary sclerosis and congestive heart failure. A 1984 trial was done with hawthorn extract using 360 mg-1600 mg/day.[9] Overall cardiac performance was improved in the hawthorn group (77%) vs placebo (49%). With further analysis, only those patients with NYHA Class II CHF showed significant improvement. No improvement was seen with NYHA Class III CHF.

Another randomized placebo-controlled multicenter trial involved 143 adults with NYHA Class II heart failure.[10] Thirty drops three times per day of hawthorn extract tincture of fresh berries or placebo was given for eight weeks. After eight weeks, a significant increase in exercise tolerance and a non-significant improvement in blood pressure and heart rate were seen in the hawthorn group compared with placebo, but no differences were seen in cardiac symptoms.

In an uncontrolled trial, one tablet, twice a day, of a 450 mg hawthorn extract WS 1442 was given for 24 weeks to 1011 patients with NYHA Class II CHF.[11] Edema of the ankle completely resolved in 83% of patients and nocturia in 50%. Ejection fractions improved, and two-thirds of the patients felt subjectively better after the hawthorn extract.

Direct comparison trials using hawthorn vs conventional medications are limited, but one compared hawthorn to captopril in the treatment of CHF.[12] CHF patients received either 900 mg/day in three doses or 37.5 mg/day of captopril. Cardiac performance significantly increased, and symptoms decreased in both groups with no statistically significant difference. Another direct comparison study compared a homeopathic preparation of hawthorn vs an ACE inhibitor plus diuretic.[13] This nonrandomized cohort study included 212 adults with NYHA Class II heart failure. After eight weeks, there was no statically significant difference between the groups in 15 measurable variables, and only a greater reduction in blood pressure in the standard therapy group.

Hawthorn preparations are modestly effective in reducing blood pressure,[14] in the prevention and treatment of atherosclerosis, lowering cholesterol, and preventing the oxidation of LDL.[15] Hawthorn preparations improve the blood supply to the heart, by dilating the coronary arteries, increase the force of contraction of the heart muscle, and regulate cardiac rhythm.[16]

Studies on hypertension are limited. A 2006 study of 79 diabetic hypertensive patients who received 1200 mg hawthorn vs. placebo for 16 weeks found modest reductions in diastolic measures in the active treatment group.[16] Mean diastolic blood pressure reduced from 85.6 mmHg at baseline to 83.0 mmHg after 16 weeks, and the placebo group was an average of 84.5 mgmHg at baseline and 85.00 mmHg post treatment. There was no group difference in systolic blood pressure reduction from baseline for either hawthorn or placebo. Two randomized control trials found decreases in both systolic and diastolic measures when treatment was administered for about three months.[17,18]

Individuals suffering from angina may also benefit from hawthorn. In one small study, 100 mg three times daily of Crataegus extract or placebo was given for four weeks.[19] Angina decreased in 91% of patients in the hawthorn group vs only 37% in the placebo group, and 45% of the patients in the hawthorn group completely stopped their nitroglycerine compared with 25% in the placebo group.

Side effects of hawthorn are minimal with doses in the range of 180-900 mg of extracts from leaf with flower, leaf with flower and fruit, and fruit preparations. Some rare adverse effects

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that have been reported include gastrointestinal disorders, palpitations, headache, and dizziness. Drug interactions with hawthorn are worth paying attention to with some reports that it can potentiate the effects of cardiac glycosides,[20] potentiate barbiturate-induced sleeping times,[21] and increase the coronary artery dilating effect induced by theophylline, epinephrine, adenosine, papaverine and caffeine.[22]

Summary

Hawthorne has a long historical tradition of use for cardiac disorders, including congestive heart failure in particular, but also hypertension and angina. Practitioners should be encouraged about the modern research in these areas, although more robust high quality randomized controlled trials would be welcomed and a worthy addition in our ability to help stem the tide of the impact of cardiovascular disease on the lives of men and women. For the ever growing number of patients who refuse and even loathe the aggressive prescribing of statins, even for those with no history of cardiac events nor diabetes, and for those who refuse or do not tolerate prescription anti-hypertensives, hawthorne can be important tools especially in the context of comprehensive lifestyle changes and other nutraceutical/botanical interventions to reduce life threatening or debilitating cardiovascular disease.

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Surviving a Flu Epidemic: The Naturopathic Approach **by Dr. Heather Herington**
150 pages (approximate)
Electronic release: May 2020; Expected printing date: June 15, 2020
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Excerpt

On Oct 18, 2019, Johns Hopkins University ran a simulation of a possible pandemic; it was a disaster.

The question of why humans are unprepared for a pandemic lies in part with the lack of hospital beds and medical personnel, but more importantly, many of us do not realize the body has the ability to heal itself. Furthermore, we don't understand how to stimulate this healing ability.

Our collective immunity has been lowered with the daily assault of toxic environments, poor quality food, stress, toxic emotions, lack of exercise, dehydration, and smoking to name a few. In addition, we lack the knowledge of how natural therapies and remedies can support our body's healing ability. People are unfamiliar with the benefit of fever and detoxification in acute illness and don't realize aspirin and other fever suppressants can have deadly consequences.

Our immune system is an exquisitely orchestrated series of steps. It knows how to support healing. There is much we can do to enhance its function through therapies like fasting, hydrotherapy, homeopathy, herbs, Traditional Chinese Medicine and more. We must learn about these natural means to support optimal immune functioning in a time when we are exposed to many things that dampen our immune system.

This book, *Surviving a Flu Epidemic: The Naturopathic Approach*, comes at a time when influenza continues to outnumber any other virus in terms of sickness and death. This book informs us about the innate healing ability of humans and how to engage that process. ◆

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level of 2.0. However, there are two reasons that even a small reduction in gastric pH (to around 4.0) could be beneficial. First, the relationship between gastric pH and pepsin activity appears to be bimodal, with activity peaks at 2.0 and 4.0.[2] Second, at a pH around 4.0, gastric juice may kill a wide range of microorganisms, some of which appear to play a role in the pathogenesis of certain medical conditions (e.g., rosacea). Therefore, rejection of the observed clinical benefits of HCl solely on theoretical grounds does not appear to have been justifiable.

Potassium Magnesium Aspartate for Fatigue

In the 1950s, it was hypothesized that fatigue is caused in part by inefficient energy metabolism at the cellular level, and that metabolic efficiency could be increased by enriching the cellular environment with appropriate substrates and cofactors. Among the physiological compounds that were available at the time, the potassium and magnesium salts of aspartic acid (potassium magnesium aspartate) were considered likely candidates to enhance metabolic efficiency. Potassium regulates basic aspects of cellular functioning (i.e., transmembrane electrical potential and intracellular ionic strength) and is also involved in muscle contraction. Magnesium is required for the synthesis of the energy-yielding compound, adenosine triphosphate (ATP), and also enhances potassium transport into cells. Aspartic acid is converted *in vivo* to oxaloacetate, which is a substrate for the Krebs cycle.

In several randomized controlled trials conducted in the early 1960s, 75-94% of patients experienced an improvement in fatigue after treatment with potassium magnesium aspartate (usually 2 g per day), whereas only 5-27% of patients given placebo improved.[3] Adverse effects were uncommon and consisted mainly of mild gastrointestinal symptoms. While this treatment is largely unknown in the mainstream medical community, I have found potassium magnesium aspartate to be one of the most effective treatments for chronic fatigue.

Desiccated Adrenal Cortex for Nausea and Vomiting of Pregnancy

In the 1930s, a practitioner developed the hypothesis that nausea and vomiting of pregnancy is caused by a temporary, relative adrenocortical insufficiency induced by the stress of pregnancy. This hypothesis was based on observations that the maternal adrenal cortex undergoes hyperplasia during pregnancy, that the earliest signs of adrenocortical insufficiency in experimental animals are anorexia and vomiting, and that the earliest manifestations of Addison's disease (hypoadrenalism) in humans are anorexia and morning sickness. According to this hypothesis, nausea and vomiting resolve when the compensatory hyperplasia of the adrenal cortex becomes sufficient to meet the increased metabolic demands of pregnancy (around the end of the first trimester).[4,5]

Based on this hypothesis, eight women with nausea and vomiting of pregnancy were treated with a desiccated adrenal cortex preparation at a dose of 6-18 grains per day. Marked improvement was seen after three-to-four days.[5,6] Following this initial report, data were compiled on a total of 202 women with nausea and vomiting of pregnancy who were treated with adrenal cortex preparations by 47 physicians. Of those treated, 86% experienced complete relief or definite improvement. Treatment was usually discontinued by the end of the first trimester.[4] Other investigators in the 1940s and 1950s also observed beneficial effects of adrenal cortex extracts.[7,8]

A midwife who learned about this treatment from my writings began recommending it for clients with nausea and vomiting of pregnancy. Of 20 women who received an adrenal preparation, 18 experienced improvement, usually within 24-48 hours. The product used (Desiccated Adrenal; Standard Process, Inc.; Palmyra, Wisconsin) contained approximately 2 grains (130 mg) of desiccated whole adrenal per tablet. The initial dose was two tablets, three times per day. After improvement occurred, most of the women were able to maintain the improvement by taking one-to-two tablets per day and eating frequent, high-protein meals. The women were usually able to discontinue treatment by the 14th week of gestation.[9]

Conclusion

It is important to remember that many of the doctors and scientists of yesteryear were smart, wise, and innovative, and that there is much we could learn from them. Many treasures lay buried in the archives of the medical library.

Alan R. Gaby, MD

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Old Research Is Not Necessarily "Outdated"

When I speak at medical conferences, the course director usually provides me a summary of the evaluation forms that attendees filled out to rate the quality of the various presentations. While most of the comments tend to be positive, it is not uncommon for someone to criticize my presentation for citing old and "outdated" research. It is true that I sometimes cite research that is more than 50 years old. However, I would submit that old research is not necessarily outdated research.

"Outdated" is defined by the Collins English Dictionary as "old-fashioned and no longer useful or relevant to modern life." The belief that old research is outdated depends on the assumption that follow-up studies have been conducted in the modern era. Such is often not the case. Medical history is replete with examples of effective treatments that fell by the wayside because they did not fit into the belief systems or practice styles of the day. Below are examples of treatments that appear to be beneficial in clinical practice, even though they are based only on old research.

Hydrochloric Acid for Chronic Diarrhea

A 73-year-old, previously healthy woman presented with a three-month history of persistent diarrhea, which had resulted in progressive weight loss. Evaluation by her family doctor had failed to identify a cause, and she had been treated symptomatically with antidiarrheal medication. When she came to see me, questioning revealed that the onset of diarrhea had followed an episode of acute gastroenteritis. It has been reported that acute gastroenteritis sometimes leads to transient hypochlorhydria that can persist for months, and that hypochlorhydria can in some cases cause persistent diarrhea. So, I asked the patient about other symptoms that are commonly associated with hypochlorhydria, such as bloating after meals and a feeling of fullness in the stomach even after a small meal. She reported that these two symptoms had also developed around the same time as the diarrhea.

Many treasures lay buried in the archives of the medical library.

Based on the clinical picture, the possibility of post-infectious hypochlorhydria was considered. The patient was advised to undertake a therapeutic trial with hydrochloric acid (HCl), in the form of one or two 600-mg capsules of betaine hydrochloride with each meal, depending on the size and the protein content of the meal. She was also told to stop treatment or reduce the dose if it caused heartburn. HCl treatment resulted in immediate improvement in the diarrhea and the other gastrointestinal symptoms. The patient was able to discontinue HCl after two months. During the following year, symptoms recurred occasionally, but again responded to HCl therapy. So, where did I get the idea that chronic diarrhea can be caused by hypochlorhydria and that HCl can be an effective treatment? It came from a study published in the *Journal of the American Medical Association* in 1902.[1]

Dilute HCl for the treatment of hypochlorhydria was popular 100 years ago. It was said to be effective not only for various gastrointestinal symptoms, but also for conditions such as chronic urticaria, asthma, and rosacea. However, by the 1940s, the treatment had fallen out of favor, largely on theoretical grounds. It was argued that HCl could not possibly be effective because the dosages used in clinical trials were far below the amount needed to decrease gastric pH to a normal

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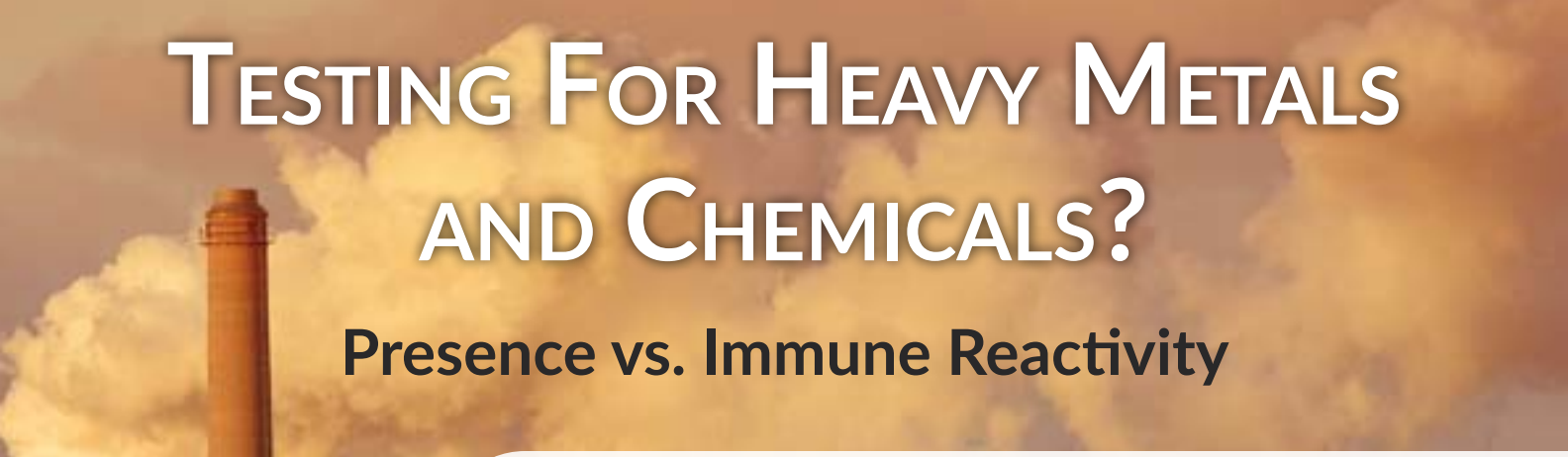

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Presence vs. Immune Reactivity

Toxic heavy metals and chemicals are all around us, in our environment and in our foods.

EVERYONE is constantly exposed.

It is not a question of **"if"** these toxins enter our body, but **"when"** they cause an immune reaction.

EVERYONE will find these toxins and chemicals in their urine at some point or another. BUT, what makes the difference is if the immune system reacts to those toxins and start binding to human tissue proteins.

When bound to tissue, the chemicals/metals will not necessarily be found free-floating in the blood, or excreted out in urine or hair. Yet they may be more destructive than their presence alone!

Testing **Levels** indicate exposure
Testing **Antibodies** indicate body burden*

Exposure → Absorption → Storage/Misfolding → Antibody Production → Cross Reactivity → Autoimmunity

Other Laboratory Tests

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**Body burden is an indication that the body is storing chemicals and unable to excrete them.*

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† This statement has not been evaluated by the Food and Drug Administration. This product is not intended to diagnose, treat, cure, or prevent any disease.

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Article

MDPI

A Novel Self-Emulsifying Drug Delivery System (SEDDS) Based on VESIsorb® Formulation Technology Improving the Oral Bioavailability of Cannabidiol in Healthy Subjects

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tract: Cannabidiol (CBD), a phytocannabinoid compound of *Cannabis sativa*, shows limited oral vailability due to its lipophilicity and extensive first-pass metabolism. CBD is also known for gh intra- and inter-subject absorption variability in humans. To overcome these limitations a l self-emulsifying drug delivery system (SEDDS) based on VESIsorb® formulation technology porating CBD, as Hemp-Extract, was developed (SEDDS-CBD). The study objective was to te the pharmacokinetic profile of SEDDS-CBD in a randomized, double-blind, cross-over design

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