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October 2020
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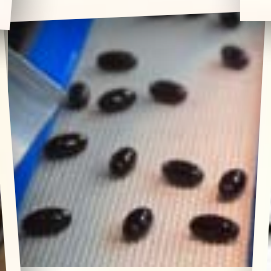
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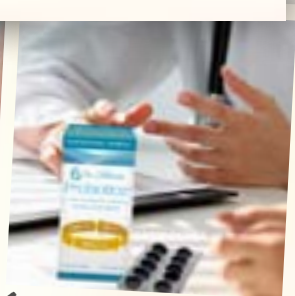
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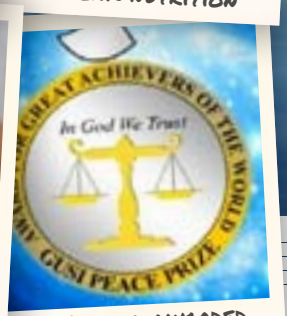

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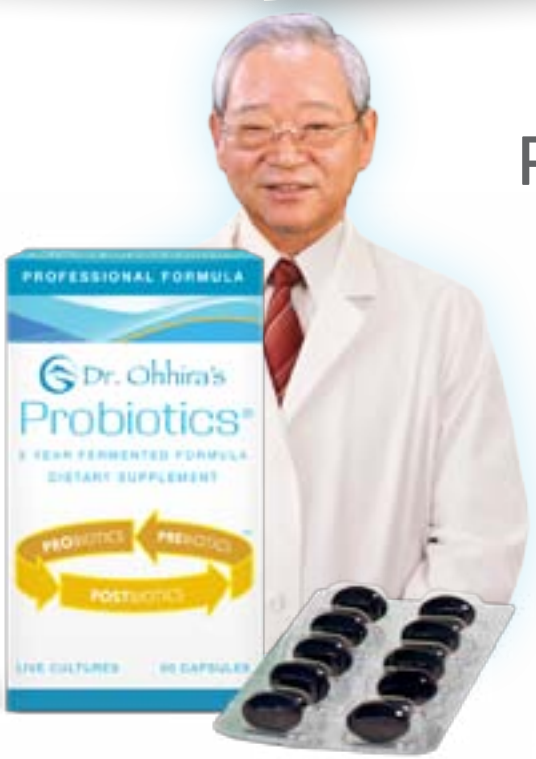

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From the Publisher

Another Pandemic Casualty – Medical Freedom of Speech

In the wildly ribald movie of the 1990s, *The Big Lebowski*, John Goodman's character, Walter, who is remembered for quipping a few dozen hilarious but off-color lines, defends his oafish loud-mouthed conversation to the waitress in a diner stating "the Supreme Court roundly rejected prior restraint." Goodman asserts a valid legal concept, "prior restraint," incorrectly to reject the waitress' attempt to ask him to quiet down. The humor aside, the first amendment protects our right to free speech. Preventing a book or an article from being published are examples of prior restraint – forbidden by the US Supreme Court. Once the work has actually been published and distributed, banning it would no longer be prior restraint but censorship. Censorship is also forbidden by the first amendment, but censorship has taken a new turn during the COVID-19 pandemic. A new standard has been sanctioned without legal basis to censor any speech or writing

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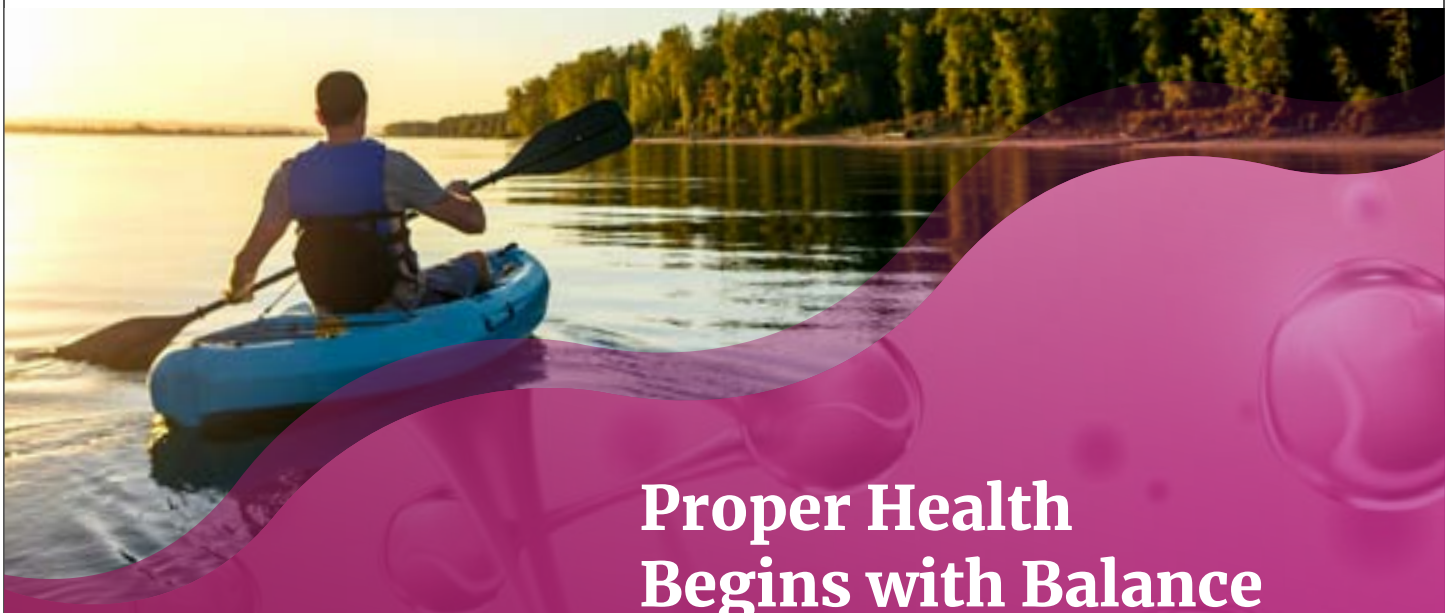

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Letter from the Publisher

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or information that is tacitly assumed to be false information regarding the diagnosis and treatment of the virus. Medical authorities, media, and social media platforms have arbitrarily decided that general consensus statements and protocols of the WHO, CDC, the FDA and other public health authorities are valid and any contrary or opposing opinions are not only invalid but must be censored.

Outrageously, some smug commentators deny that it is censorship and label such activities as "moderating" – as in social media platforms are "moderating" content being posted. Seriously, moderating! Censorship is censorship. Facebook, Twitter, YouTube under pressure from medical authorities are now removing all information posted that contradicts mainstream understanding of how the virus is to be prevented, treated, and controlled. Unlike removal of disinformation created by trolls, the information now being removed includes opinions by health professionals who do not accept the status quo and voice opposing viewpoints. The most frequently censored item is undoubtedly any discussion that asserts that wearing masks does not effectively prevent spread of the virus. In August, information touting the benefit of the drug hydroxychloroquine in treating COVID-19 was loudly decried as disproven by several large placebo-controlled studies; physicians who asserted patient benefit were denigrated and their reports were censored. When the social media platforms censor a posting, that information is essentially impossible to find by Google search. It has not yet come to the place where book publishing is being banned but stand by. Given the essential universal dependence of academics and public on information found on the web, censorship is a big problem and a very worrisome problem.

What will be next? Can we expect public health authorities to demand prior restraint of information posted on the web? We do see this prior restraint in communist and autocratic countries. While we do have the US Supreme Court decision, we are beginning to slide down a slippery slope where information is being withheld under the faulty thinking that public health safety demands the elimination of any medical opinions that argue against the mainstream. It has reached the level that doctor is now battling against doctor. Physicians who demand that the only acceptable medicine is "evidence-based" medicine are becoming belligerent against doctors who use best clinical judgement in treatment of their patients. Medicine is becoming so polarized that the "red/blue" division is entering into the hospital and clinic; physicians are beginning to wage degradation campaigns against physicians who do not stick absolutely to the "standard of care."

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Letter from the Publisher

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Readers of the *Townsend Letter* who are naturopathic physicians as well as functional medicine doctors are interested in keeping abreast of evidence-based medicine. Nevertheless, most practitioners have been interested in incorporating alternative diagnostics and therapies. The patients who seek alternative care do not want to be restricted to guidelines set down by the FDA and CDC. Despite the constant drumbeat of the media that the only way to be able to end the pandemic is through the entire population being vaccinated, there is a vast, visceral repulsion among patients and practitioners to agree to vaccination for COVID-19. We should assume that the social media platforms and general media will be censoring any anti-vaccine information. Will network operators that license individual websites begin to sanction those that post information considered to be contrary to public health consensus? Will medical boards initiate investigations of physicians who voice opposing opinions? Will the web be policed by some new superagency that will become the ultimate arbitrators of what is acceptable and what is not acceptable medical information?

In *The Big Lebowski*, Jeff Bridges who plays "the dude" protagonist quips, "Yeah, well, you know, that's just, like your opinion, man." Yes, it is just the doctor's opinion but that opinion is important and deserves to be articulated. Dissemination of that opinion is guaranteed by the first amendment and must not be abridged by medical authorities and social media platforms.

How Do We Treat Our Collective Angst?

Writing this in early August, it would take a soothsayer to know where we will be in October with the pandemic. Many schools and universities are electing to have in-person classes; not a smattering of them are shutting down after students and staff tested positive for COVID-19. However, most are choosing to teach online, much to the chagrin of parents and children. It does not take a soothsayer to see that isolation and social distancing is playing havoc on our lives – that we are becoming separated and lonelier. That may be the most critical ingredient impacting our mental health, responsible for making us not just irritable but anxious and depressed.

Jule Klotter reviews Johann Hari's book, *Lost Connections: Uncovering the Real Causes of Depression – and the Unexpected Solutions* in this issue. The psychiatric model has always separated anxiety and depression, but what if they are "twinned," two sides of a coin? Psychiatrists have begun to consider this duality often prescribing anti-depressant medications in the treatment of anxiety. The trouble with anti-anxiety medication, especially the benzodiazepines, is their addictive characteristic; even those that pose less of a dependency risk often yield difficult alterations in our mood and mentation. Hari wants us to consider a non-pharmaceutical approach to both anxiety and depression. Klotter's review of Hari asks us to consider the "disconnection from meaningful work, from other people, from meaningful values, from childhood trauma, from status and respect, from the natural world, and from a hopeful or secure future." Especially

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Sovereign Laboratories

Better Mental Health Requires a Healthy Gut Microbiome

Social isolation and chronic stress can lead to mental health issues, such as anxiety, depression, substance abuse, and suicidal ideation.[1] In trying times, it's even more important to address the problem head on. Our new-found knowledge about the gut microbiome and brain health illustrates the significance and benefit of taking good care of these microbes, nutritionally speaking. The trillions of microbes that call the human G.I. tract home are influential in the secretion of neurotransmitters and metabolites that act upon the brain. If dysbiosis and leaky gut develop, systemic inflammation increases. Inflammation has been identified as a primary cause of depression, but bovine colostrum can help lower inflammation. Blood tests prior and after colostrum use show decreases in inflammatory markers.

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[1] Czeisler MÉ, Lane RI, Petrosky E, et al. Mental Health, Substance Use, and Suicidal Ideation During the COVID-19 Pandemic - United States, June 24-30, 2020. *MMWR Morb Mortal Wkly Rep*. 2020;69(32):1049-1057. Published 2020 Aug 14.

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Letter from the Publisher

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now when we may be faced with another lockdown and be obliged to quarantine because of a positive test, it is needful to address our lost connections. We have to move beyond reaching for a pill and look for new means to connect and create meaningfulness.

Would Ultra-Low Frequency Electromagnetic Fields Be the Balm for Anxiety and Depression?

Everyday life in the 21st century is awash in devices employing electromagnetic fields. We wouldn't think of leaving home without our cell phone or remaining off-line by not connecting our computers to WiFi, both technologies using EMF. From a health perspective our EMF exposure is thought to be a minimal health risk – "high tech" dismissing its pathogenicity. Still not a few patients are extremely sensitive to EMF and make extensive efforts to limit their exposure, some even living "off the grid." No one questions the major risk that ultra-high frequency EMF poses – imagine if we were physically placed within a microwave oven! However, we have largely ignored the health benefits that may accrue by exposure to extremely-low frequency EMF. In fact, this technology is now widely available and is generally known as pulsed electromagnetic field devices or PEMF. PEMF may be the preferred alternative to pharmaceuticals for treating anxiety and depression as well as other psychiatric disorders. In this issue's cover article, Dr. William Pawluk, Nancy Faass, and Jerry Stine explore the role PEMF devices play in managing neurological disorders.

Theoretically it makes sense that the brain should respond well to PEMF treatment. After all the frequency of alpha, beta, delta, gamma, and theta brain waves match PEMF frequencies – 0.1-100 Hz (cycle/second). Unlike the extremely high frequency EMF of cell phones, short wavelength energy that is readily absorbed by the body, the ultra-low frequency energy of PEMF passes through the body without being absorbed. The impact is entirely different between the two; cell phone EMF absorption has a high potential for damaging the body, while non-absorbed PEMF energy can synchronize with brain waves, restoring normal brain rhythms and functioning. PEMF has the potential for reducing inflammation, pain, ischemia, and neurochemical imbalance. While PEMF has some contraindications, for example, in patients with pacemakers or during pregnancy, it poses little risk for most patients. This may be an ideal tool for the stressed individual suffering with insomnia and intractable pain.

The Stressed Brain

In the late 1970s while transitioning to integrative medicine, I became fascinated reading the work on calciphylaxis by the Canadian physiologist, Hans Selye. His book of the same title published in 1962 discusses experimentation he employed to create animal models of the condition. In one experiment with mice he examined the toxic effect of varying doses of chemical agents on the adrenal glands. With lower doses used over shorter periods of time, the "stress" of the chemical led to increasing hyperplasia of the adrenals. As the stress increased by lengthier exposure to the chemical, the adrenals maximized in size; and then when a certain stage was reached, the adrenal gland became exhausted and involuted to sub-normal size. Of course, he demonstrated this with autopsied photos of the mouse adrenal glands – the dramatic anatomical changes were so vivid that they persist in my mind's eye. Selye pursued his work on stress through the rest of his academic life. A 1997 paper, "Stressors, Stress, and Neuroendocrine Integration of the Adaptive Response," is an excellent overview on stress and its diverse interrelationships through all systems in the body.

In this issue Jonathan Prousky, ND, MSc, professor and chief naturopathic medical officer at the Canadian College of Naturopathic Medicine, begins a series of articles delineating the role of stress on the brain and its regulation. While we all acknowledge the role of stress in exacerbating psychiatric and physical illness, we generally give short shrift on how stress disrupts neurochemistry as well as hormone and immune functioning. Prousky's paper focuses on the physiological concept of allostasis, which differs from homeostasis. While the latter refers to the body's physiologic mechanisms necessary to maintain survival, allostasis is the general adaptation to stress that an individual mounts over one's lifetime. Allostatic adaptation may or may not work to the individual's benefit and counterintuitively may be counter survival. Prousky notes that there is an allostatic load that the brain must adapt to and that load may become an overload. As one might imagine allostatic load will cause the brain to dysfunction leading to "psychological distress" manifesting as anxiety, depression, insomnia, helplessness, hyperactivity, addictive behavior, suicidal ideation, delusional thinking, obsessive behavior, and more. Prousky's paper provides a framework to understand the brain's maladaptive response leading to the breadth of psychiatric conditions and illnesses. Future work will discuss treatment avenues for addressing the stressed and inflamed brain.

Jonathan Collin, MD

Shorts

briefed by Jule Klotter
jule@townsendletter.com

Aluminum and Neurodegenerative Disease

How much aluminum in the brain is too much, and are there ways to reduce aluminum levels? Those are the questions that aluminum expert Christopher Exley and colleagues have looked at over the past years. Aluminum is neurotoxic. In animal studies, too much aluminum produces neuropathological and neurodevelopmental effects that resemble human neurodegenerative diseases. Exley and colleagues have documented high aluminum levels in people who died with Alzheimer's disease (AD), multiple sclerosis (MS), and autism spectrum disorder (ASD), but they were criticized for presenting the information without a control.

For a 2020 *Scientific Reports* study, Exley and Elizabeth Clarkson were able to acquire brain samples from 20 people, mostly elderly, with "no overt neurodegeneration, no diagnosis of a neurodegenerative disease but some age-related changes in the older donors." Over 80% of the 191 tissue samples from this control group had an aluminum content below 1.0 μg/g dry weight of tissue. In earlier studies, Exley and colleagues found that brain samples from people with familial Alzheimer's disease contained aluminum concentrations that ranged from 0.01 to 35.65 μg/g dry wt; 11 out of 12 donor brains had at least one tissue in which aluminum content was ≥3 μg/g dry wt, which is considered "pathologically significant." Aluminum content in brain samples from people with MS ranged from 0.01 to >50 μg/g dry wt, and all 14 donors had at least one pathologically significant sample. All five donors with ASD also had at least one sample with an aluminum content of ≥3 μg/g dry wt; aluminum levels in these samples ranged from 0.01 to 22.11 μg/g dry wt. The researchers compared the control samples to those who died with Alzheimer's, MS, or autism: "The aluminium [sic] content of brain tissue in the control group was significantly lower than [sporadic Alzheimer's] ($P=0.0006$), [familial Alzheimer's] ($P=0.0020$), ASD ($P=0.0123$) and MS ($P<0.0001$)." Because most of the control samples came from elderly people, these results show that aluminum brain levels are not an inevitable result of aging despite its ubiquitous presence in food, water, personal products, and medications.

In addition to these quantitative studies, Exley and other researchers have used aluminum-specific fluorescence microscopy and other forms of microscopy and found that aluminum tends to accumulate in locations specific to the disease. In ASD, aluminum deposits were primarily intracellular and associated with non-neural cells (eg, microglia). Familial AD brains, on the other hand, had mostly extracellular deposits that were closely related with amyloid β. Aluminum deposits in those with MS appeared in both intracellular and extracellular locations and were associated with plaque-like structures and corpora amylacea (granular bodies, abundant in neurodegenerative disease, that amass waste products).

I have heard Exley state in interviews that Alzheimer's disease cannot occur without aluminum. While the jury is still out on that, the high aluminum levels found in the brains of people with these neurodegenerative diseases and the metal's established neurotoxicity indicate it would be prudent to follow the basics of environmental medicine: reduce/eliminate the source and find non-toxic ways to remove the metal from the body. Aluminum is found in processed foods and food packaging. The metal is also used in water purification, medicine (eg, antacids and buffered aspirin), vaccines, antiperspirants, and cosmetics. Reducing aluminum intake is step one.

One method for reducing aluminum (Al) levels already in the body is to drink silicon-rich water. In a 2012 article, Exley and colleagues reported that drinking up to one liter of silicon-rich mineral water each day for 12 weeks increased excretion of aluminum in people with Alzheimer's disease and in their caregivers or partners, who acted as the control group. In addition to reducing Al body burden, at least three of the 15 AD patients showed "clinically relevant" cognitive improvements at the end of the 12 weeks. In another study, 15 people with secondary progressive MS drank up to 1.5 L of silicon-rich mineral water (Malaysian mineral water Spritzer) daily for 12 weeks after a 12-week baseline period that acted as the control. Urinary Al excretion significantly increased from an already high baseline level of 135.2 nmol/

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mmol creatinine to 349.0 nmol/mmol creatinine at the study's end. Both studies are preliminary; much longer studies are needed to see if drinking silicon-rich water slows progression of neurodegenerative diseases linked to aluminum. For those who want to add silicon-rich drinking water to their health regimen, Exley says, "What you need to look for is a minimum concentration of 30 mg/L or 30 ppm written as 'silica' on the label." Fiji and Volvic are two options.

Exley C. Why everyone should drink silicon-rich mineral water. The Hippocratic Post. March 15, 2017.
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Jones K, et al. Urinary Excretion of Aluminium and Silicon in Secondary Progressive Multiple Sclerosis. *EBioMedicine.* 2017;60-67.

Iron Chelators and Parkinson's

Large randomized clinical trials are investigating the use of conservative iron chelation as a long-term treatment for neurodegenerative disease. Iron accumulation, causing oxidative damage, is a characteristic of both Parkinson's disease (PD) and amyotrophic lateral sclerosis (ALS). In PD, labile iron levels are increased in the substantia nigra (basal ganglia structure in midbrain associated with reward and movement). In early ALS, iron accumulates in corticospinal motor pathway neurons and, then later, in microglia. Conservative iron chelation does not remove iron from the body, which could result in iron depletion and anemia. Instead, the chelating agent attaches to labile iron and recycles it "back into circulation via the physiological carrier transferrin."

Several possible chelating agents have been tested in the laboratory, but deferiprone (DFP) is the only one that has been used in clinical trials. DFP is able to cross the blood brain barrier and chelate cellular labile iron in brain tissue. A 2015 French study, led by G. Grolez, tested deferiprone (15 mg/kg/day in the morning and evening) in a randomized trial with 40 patients. One group took DFP for 18 months; the other group took a placebo for six months and then took DFP for the remaining 12. DFP treatment reduced iron levels in the substantia nigra and improved motor scores. Patients with the AT genotype group with the D544E polymorphism had a greater response than the AA genotype.

DFP has a good safety profile, according to Devos et al; but weekly blood counts are needed during the first six months to monitor for "reversible neutropenia that could occur in 1-3% (agranulocytosis in 0.8%) of patients." Iron levels remained normal in PD patients who maintained DFP treatment for 18-24 months and in three ALS patients and one PD patient who received DFP (compassionate use) for four-to-five years.

Conservative iron chelation with DFP is the subject of a nine-month, phase II European multicenter, randomized, controlled clinical trial involving 372 de novo PD patients (www.fairpark2.eu). DFP's effect on 240 newly diagnosed ALS patients is the subject of a 12-month French multicenter randomized, controlled trial.

Although DFP is the most promising iron chelator at this point, Carlos Perez and colleagues at the University of Massachusetts point out that some plant polyphenols, such as quercetin and curcumin, are iron chelators. Moreover, several polyphenols, like those in green tea, have documented neuroprotective effects that are beneficial for people with PD.

Devos D, et al. Conservative iron chelation for neurodegenerative diseases such as Parkinson's disease and amyotrophic lateral sclerosis. *J Neural Transmission.* 2020;127:189-203.
Grolez G, et al. Ceruloplasmin activity and iron chelation treatment of patients with Parkinson's disease. *BMC Neurology.* 2015;15:74.
Perez CA, et al. Iron Chelators as Potential Therapeutic Agents for Parkinson's Disease. *Curr Bioact Compd.* October 1, 2008;4(3):150-158.

Mental Suffering and Health-Related Disasters

The 2003 severe acute respiratory syndrome (SARS) outbreak in Canada and the 2009 H1N1 influenza epidemic in the US let researchers look at the psycho-emotional effects of pandemics. During the SARS outbreak public health officials asked over 15,000 Toronto residents, who were exposed to the infection, to remain in voluntary quarantine. They were supposed to remain in their homes, not have visitors, and follow several other measures: wear masks when in the same room as other household members; wash their hands frequently; not share towels, drinking cups, or other personal items; sleep in a separate room; and take their temperature twice a day. In at attempt to evaluate the psychological effects of this program, Laura Hawryluck and colleagues invited the quarantined, via media and posters, to take part in a survey. Only 129 people completed the 152-item survey, composed of multiple choice, short-answer questions, the validated Impact of Event Scale-Revised and the Center for Epidemiologic Studies Depression Scale. Despite the small sample size and other limitations (self-selection, and requirement for online access), the study documented some interesting trends. First, the lower the combined annual household income of the respondent, the more symptoms of post-traumatic stress disorder (PTSD), including anxiety, avoidance of stimuli associated with the event, and increased vigilance. Longer quarantine was significantly related to increased PTSD symptoms and sense of isolation. The authors say, "Infection control measures imposed not only the physical discomfort of having to wear a mask but also significantly contributed to the sense of isolation."

In the H1N1 study by Sprang and Silman, 398 parents completed a survey composed of multiple choice questions, a rating scale, questions about their pandemic experiences as well as a parent-report version of the UCLA Posttraumatic Stress Disorder Reaction Index and the PTSD Check List – Civilian Version. The authors say, "…pandemics have much in common with other disasters: community impact, unpredictability, fatalities, and persistent effects." But unlike other disasters, in which people come together and help one another, pandemics often require separation and isolation. Their data showed that quarantine and isolation were associated with PTSD symptoms in 30% of children (from parent reports) and 25% of the parents (self-report). This incidence of PTSD was similar to the incidence after natural disasters and terrorism. The

authors say, “While such disease-containment measures may quell the outbreak, they have the unintended consequence of inhibiting family rituals, norms, and values, which regulate and protect family functioning in times of crisis….the inhibition or interruption of such functioning may both diminish individual and family resilience and increase the potential for adverse reactions.”

Although both of these studies used validated PTSD scales to assess psychological effects, Paula J. Caplan, PhD, a clinical and research psychologist, activist, and associate at Harvard University’s DuBois Institute, warns against pathologizing the mental and emotional suffering that is stemming from the COVID-19 pandemic. The unknowns, the continual change in recommendations, fear of becoming seriously ill or dying, physical isolation, loss of connections with others, job loss, and financial crises are all totally understandable reasons for mental suffering. She writes, “…as soon as a person is diagnosed as ‘mentally ill,’ their own focus and that of professionals tend to veer sharply away from non-pathologizing, low-risk and no-risk approaches that are known to be effective.” Creative or artistic projects, physical exercise, meditation, having a companion animal, doing volunteer work, and having a listener are just some of the ways to mitigate the stress. “People who are suffering emotionally from the effects of COVID-19 deserve help,” says Caplan, “but it must be real help, such as lifting their economic burdens, protecting them from violence, and increasing community support, including all of us showing we are willing to listen to what they are going through and acknowledging how common these struggles are.” Community mental health programs that promote self-care, such as the one in Ashland, Ohio, could prevent a surge in crisis calls as the pandemic drags on and when the crisis finally ends. The Ashland Mental Health and Recovery Board offers breathing and mindful exercises online and also provides opportunities for social connection and peer support, such as a sewing group that has made masks for the community and a writing group that sends cards and letters to patients in state hospitals.

Toning – vocalizing sounds (usually vowel sounds) on a full exhalation of breath – might also be helpful. A 2019 study, conducted by Erik Peper and colleagues, found that three minutes of toning UO (pronounced as you) reduced mind wandering and intrusive thoughts more effectively than mindfulness practice (MP). The authors report, “There was a decrease in respiration rate during TP [toning] (4.6 breaths/min) as compared to MP (11.6 breaths/min; p<.001) and an increase in heart rate variability during TP (SDNN = 103.7 msec; SD = 11.6) as compared to MP (SDNN = 61.9 msec; SD = 6.4).” The study was conducted on college students in a group setting and may not be as effective for other populations and settings. But, for people who find themselves overcome with fearful thoughts, this is a no-cost tactic that is worth a try.

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Sprang G, Silman M. Posttraumatic Stress Disorder in Parents and Youth After Health-Related Disasters. *Disaster Medicine and Public Health Preparedness.* 2013; 7(1):105-110.

Thomas T. Mental health services in Ohio prepare for their own surge of new clients. April 27, 2020. www.richlandsource.com

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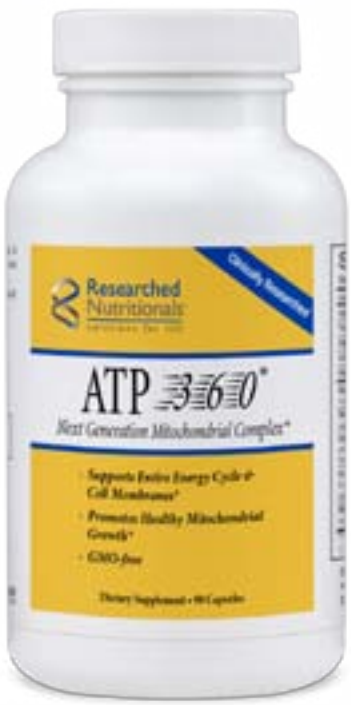

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ONLINE ONLY

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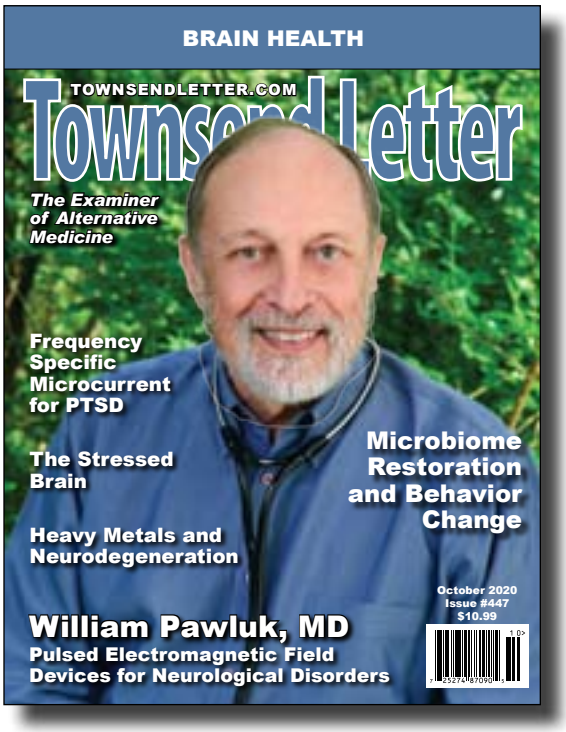

On the cover

PEMF (Pulsed Electromagnetic Field) Devices in the Care and Management of Neurological Disorders

by Dr. William Pawluk, MD
with Nancy Faass, MSW, MPH and Jerry Stine, CNC

PEMF (pulsed electromagnetic field) devices reflect an emerging approach to cost-effective therapy for difficult-to-manage neurological and pain disorders. The primary focus of this review is the use of devices of low electromagnetic intensity and frequency, designed for self-care by consumers, with assessment, guidance, and follow-up by their clinicians.

Indications. PEMF has been shown to support progress in neuro-chemical conditions that include addiction, ADHD, anxiety, autism spectrum disorders, depression, PTSD, and sleep issues. Research also indicates benefit for neuro-pathologies such as concussion, memory loss, mild cognitive impairment (MCI), mild traumatic brain injury (TBI), multiple sclerosis, Parkinson's disease, stroke, and tremor. A variation of PEMF, high-powered rTMS (repetitive transcranial magnetic stimulation) devices used in hospitals, are FDA-approved to treat conditions ranging from bone healing to resistant major depressive disorders (MDD).

Mechanisms of action. As a magnetic field passes through the body, that field can stimulate a range of potential processes and activities, depending on the frequency and power:

- Reducing inflammation and edema
- Increasing ATP production
- Rebalancing circadian rhythm
- Improving circulation and blood supply
- Increased oxygen to the body's tissues
- Healthy levels of growth factors and nitric oxide
- Increasing cell metabolism
- Production of endogenous antioxidants
- Enhanced detoxification
- Wound healing and tissue regeneration
- Stimulating and rebalancing the immune system
- Improved nerve conductivity and regeneration
- Promotion of growth gene factors
- Support of neural stem cell production
- Reduced pain
- Autophagy
- Enhanced neurotransmitter levels
- Effect on monoamine function
- Reduced reaction time
- Charge displacement from neuronal membranes of cortical

Overview of the Devices

Pulsed electromagnetic devices encompass a broad range of appliances with corresponding capabilities.

High-intensity, high-frequency devices utilized in health systems. In terms of high-end devices, a robust literature is available, documenting approximately 2,400 clinical trials using rTMS to date. rTMS devices operate at an intensity of 8,000 gauss. By way of context, an MRI machine produces a high-intensity magnetic field ranging from 20,000 to 60,000 gauss (2 to 6 Tesla) depending on the machine and application. rTMS devices have been approved since 2008 and are reimbursable for the treatment of medication-resistant depression. These units cost approximately $50,000, so they are typically found in hospital medical centers and large mental health group practices. rTMS enhances neuroplasticity, entrains and resets brain cell oscillators between the thalamus and the cortex, normalizes regulation, facilitates reemergence of natural cerebral rhythms, and through these mechanisms restores normal brain function. TMS can be administered broadly at a lower magnetic field strength to treat multiple brain areas simultaneously.

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Low-intensity, low-frequency devices for self care. At the other end of the spectrum are devices designed for home use and self-care, currently in the range of $500 to $13,500. The frequency of these devices is typically 1 to 100 Hz, with intensity ranging from 70-7,000 gauss, and typically used to support sleep, pain management, or numerous other aspects of healing. Electronic appliances for consumer use are typically in the range of extremely low frequency (ELF) at or below 100 Hz.

The therapeutic process focuses on given brainwave states (gamma, beta, alpha, theta, and delta) that are scientifically well known and frequency specific. Ninety years of brain imaging have made it clear that the brain is an organ that responds to frequency. Alpha and beta brainwaves, produced by the brain itself, were first reported in the medical literature in 1929 by the German neurologist, Hans Berger, the inventor of the EEG. Since that time, frequency states of the brain have been measured and associated with a range of mental states and activities – alpha through gamma. In meditation, for example, a great deal of research exists for use of magnetic field frequencies to achieve the same brainwave entrainment using the theta frequency (4 to 7.5 Hz), or the more recently discovered 40 Hz gamma. In contrast, a PEMF signal within the beta range (from 13 to 25 Hz) provides an overall effect of increased alertness by typically causing a greater portion of the brain to synchronize with that frequency.

In essence, brainwave entrainment is the practice of causing brainwaves to fall into step with an external stimulus. Neurofeedback, for example, seeks to entrain the brain by promoting these frequencies. However, as a clinician, I personally stopped providing neurofeedback because I found that magnetic field therapy could do the job faster than neurofeedback, which tends to be slow and costly to patients.

More powerful PEMF systems, designed for use in the clinical environment by providers, for therapeutic interventions such as pain management rely primarily on higher density pulses rather than frequency. A PEMF will pass completely through the body whether it is of high or low intensity. The difference is the amount of charge the field will stimulate in the tissues as it moves through the body. This is based on Faraday's law. The magnetic fields produced by the body itself are well under 1 thousandth of a gauss (nano- or pico-Tesla), or less than one tenth of a microTesla. Human biology is affected by and responds to a vast range of magnetic field intensities.

Differentiating PEMF from EMFs. The EMFs produced by cell phones, microwave appliances, cell towers, Wi-Fi, and now 5G transmission, all involve extremely high frequencies, with very short wavelengths, so they are absorbed by the body, with the potential for damage. In contrast, ELF (extremely low frequency) PEMF devices are extremely low frequency with extremely long wavelengths; therefore, they penetrate through the body completely and do not stop in the body. The body is transparent to extremely low-frequency magnetic fields.

Table 1. The range of electromagnetic frequencies in medicine, manufacturing, and communications.

Electromagnetic Frequencies	
Static electric or magnetic fields	**0 Hz**
Extremely low frequency (ELF) electric or magnetic fields	**1 – <300 Hz**
Delta, theta, alpha, beta, gamma frequencies	0.1–100 Hz (200 gauss or greater)
rTMS treatment	1-20 Hz (8,000 gauss)
Intermediate frequency electromagnetic fields	**300 Hz – 100 kHz**
High Frequencies	**100 kHz – 10/30 THz**
Radiofrequency electromagnetic fields (radiowaves),	100 kHz – <300 MHz
Microwave ovens	100 MHz
Microwave radiation	300 MHz – <30 GHz
Wi-fi communications	900 MHz – 60 GHz
5G Millimeter waves (MMW)	30 GHz – <300 GHz
Terahertz waves	300 GHz – 10/30 THz

Contraindications and Safety

Patients with a pacemaker or other type of implanted electrical device. The only absolute contraindication for use of a PEMF device is the caution not to place an active applicator over an implanted electrical device such as a pacemaker, cochlear implant, intrathecal pump, etc., because the magnetic field can shut off the device or otherwise interfere with its function. There are increasingly available MRI-safe implanted electronic devices such as pacemakers that pose no risk when using a PEMF device.

Organ transplant patients. PEMF therapy is also contraindicated for these patients, since they take immune suppression medications to prevent organ rejection. There is a chance that PEMFs may actually stimulate or activate a more aggressive rejection process by stimulating the immune system. PEMF interactions with immunosuppressive medications can be unpredictable.

Implanted metals. Extremely high intensity PEMFs should be used with caution or with professional guidance for people with implanted metals, such as joint replacements, dental implants, mechanical heart valves, metal stents, or metal staples because extremely high intensity PEMFs may stimulate the nerves in the area of the metal, causing sharp pain.

Pregnancy. Although there is no evidence of harm, the safety of PEMFs has not been proven in pregnancy.

Active bleeding or Grave's disease. PEMFs should be used with caution in Grave's disease or in the case of active bleeding.

Seizures. There is now a fairly large body of research regarding the use of PEMFs of different intensities and the potential association with seizures. Evidence indicates that PEMFs are very unlikely to cause seizures, based on research

studies using high-intensity PEMF systems, which have the highest concern for generating seizure activity. In fact, studies have suggested that PEMFs are capable of providing benefit to epileptic patients. In a study of 58 patients with partial or generalized epilepsy, TMS did not provoke seizures or EEG changes in any patient. Long-term follow-up found that their epileptic conditions were not made worse by TMS. Subsequently, a review of 20 studies involving 859 subjects found that 45% of patients responded favorably to treatment and 28% experienced seizure cessation.

Adverse effects. Most adverse reactions are mild and temporary and can be managed by simply continuing the therapy. Sudden increases in circulation, especially in ischemic tissues (areas with restricted or reduced blood flow), may lead to uncomfortable increases in circulation for a short time after the magnetic field has been applied. The increase in circulation, while usually a desirable effect, can lead to a surge of oxidative stress. It is desirable to have adequate antioxidant support in the body before beginning treatment. Sudden improvements in circulation may also lead to aggravations of existing extensive or severe inflammatory processes, typically in the skin or soft tissues. Aggravation of hives is likewise possible and should be considered before starting treatment. In individuals with electrical hypersensitivity and electromagnetic hypersensitivity, these reactions are more common and more uncomfortable. However, rarely does magnetic therapy have to be discontinued as a result. The recommendation is to use lower intensities and shorter treatment times.

If the adverse reactions are intolerable, they can be lessened by making small changes to the protocol. These changes include lowering intensity and decreasing treatment times. These changes should only be necessary on a short-term basis since reactions tend to diminish relatively quickly with continued treatment and as healing progresses.

PEMF in Clinical Practice

The problem with being prescriptive or instructive on the use of a PEMF device is that it depends on the specific PEMF system. PEMF in clinical practice means first, understanding the instrument you are utilizing, and how PEMFs work. You must also understand the pathophysiology that you're dealing with, and the therapeutic potential of the device for that disorder. Essentially this is a process of putting two and two together. There are many PEMF systems and every system is going to have a particular aspect of how you would use it.

Begin with low intensity and brief treatment times. Observe the patient's response. If they are progressing in the desired direction, increase the time and intensity in increments. Once the patient is stable in their response, it becomes a matter of healing the brain, to the extent that it can be healed, and then moving into maintenance mode to maintain the benefit.

Frequency. Frequency is literally a measure of the frequency of the pulsing of a particular magnetic field. Typically, a single frequency is selected and with the continued stimulation, an increasing number of cells will vibrate to that frequency. Consequently. the brain will perform in that mode, such as ➤

beta which promotes alertness. Gamma frequencies are often deficient in Alzheimer's patients, and there is animal research indicating that promoting gamma frequencies will slow the onset of AD and tau deposits.

Intensity. Magnetic field intensity (flux density) is basically a measurement of the strength of a magnetic field. The intensity of a magnetic field is responsible for how much charge is induced in the stimulated tissues. The intensity is often defined by the power of the device. By varying degrees, that determines the strength of the magnetic field, and therefore the amount of stimulation that accompanies it.

Medical PEMF. Devices are available that are more powerful than the ELF PEMF designed for home use. Higher-intensity devices are available for use by clinicians that range from 200 gauss to 8,000 gauss. A device that I would use in clinical practice for brain treatment would be 2,000 gauss ($4,600), or a 4,000 gauss unit ($6,000 – $7,000). A device that enables treatment of two areas of the body at the same time runs approximately $13,000. As the intensity deliverable by the machine increases, the price typically increases. Often practitioners can manage to provide substantial treatment using moderate intensity devices.

Duration. For low intensity systems, duration is not a concern. If I am treating a patient with exquisite sensitivity, I go "low and slow." In clinical practice, the provider becomes highly familiar with their device of choice, so treatment involves getting to know the patient and their specific response to that particular device.

Treatment Outcomes

Bone and wound healing. The FDA approved pulsed EMFs (biphasic low frequency signal) for the treatment of fractures with delayed bone healing in 1979. Beneficial effects on wound healing have been documented for three decades. A study published in 1992, for example, reported: "Wound surface area, ulcer depth, and pain intensity were assessed at weeks 0, 4 and 8. At week 8 the active group had a 47.7% decrease in wound surface area vs. a 42.3% increase for placebo ($P < 0.0002$)."

Our Upcoming November Issue Focuses on Fibromyalgia, Chemical Sensitivity, and Pain Management

Contributing Authors:

JACOB TEITELBAUM, MD
Fibromyalgia and Chemical Sensitivity

WILLIAM PAWLUK, MD
Non-Pharmaceutical Treatment for Chronic Pain

JONATHAN PROUSKY, ND
Stress and Brain Health

Nerve regeneration. Animal research conducted over the past three decades has found evidence that PEMF can support regeneration of nerve tissue. We see this in patients with neuropathic numbness who begin to experience paresthesias once the nerves begin to recover.

Sleep issues. In a double-blind study using a 4 Hz (5 gauss) unit, researchers found that this intensity and frequency were effective in reducing sleep disturbances within six weeks in 83% of the treatment group, compared with 57% in the controls. Another study, a four-week double-blind, placebo-controlled clinical trial, involved 100 people with insomnia (sleep latency, interrupted sleep, or nightmares). Some 90% of the treatment group experienced clear or substantial improvement, whereas approximately 50% of the placebo group benefitted.

Neurochemical Disorders

Bipolar disorder and depression. PEMF therapy was found to produce rapid mood elevation in depressed patients with bipolar disorder, as well as other depressive disorders. Another study found that after five weeks of active treatment with low-intensity transcranial PEMF (T-PEMF), there was a 62% reduction on a Depression Rating Scale among those in the active T-PEMF group. This study also found that improvement was noticeable within the first weeks of therapy. A home study of patients with depression using the T-PEMF reported that progress was gradual but sustained. At five weeks, only 27% had obtained relief. However, after eight weeks of daily treatment, 73% of subjects were relieved of their depression. Two years after the treatment, 52% of subjects were still in remission, and those who were not in remission, found relief after another course of treatment.

Neurological Pathology

Mechanisms of action. In PEMF studies conducted on nerve tissue cells in the laboratory setting, preliminary data suggest that exposure to a PEMF of short duration may have implications for the treatment of acute stroke. PEMF exposure counteracted low oxygen supply (hypoxia) damage significantly, reducing cell death by inhibiting activation of various tissue chemical processes involved in oxygen deprivation. PEMFs significantly decreased low oxygen-induced oxidative stress after 24 to 48 hours. Moreover, PEMFs were able to reduce some of the most well-known inflammatory cytokines, including TNF-α, IL-1ß, IL-6, and IL-8.

Memory loss. Studies have been performed over the past 25 years to investigate the effects of low-intensity PEMF stimulation on memory processing and attention. Researchers at Northwestern University studied the use of PEMFs to stimulate the brain in older adults to determine whether they could improve memory by stimulating the parietal lobe. Stimulation was performed using a 100 gauss PEMF at 10 pulses per second for 20 minutes in each session, over five consecutive daily sessions. Full intensity stimulation was compared to low intensity "sham" stimulation. The researchers also used functional MRI (fMRI) to check brain function and neurological activity between the parietal

lobe and the hippocampus. By the completion of the study, the memory tasks of the older individuals had improved so much that they now appeared similar to the younger control group. In other words, memory loss was reverse-aged with active PEMF stimulation.

Multiple sclerosis. A study of relapsing remitting multiple sclerosis comorbid with TBI reported that no patient showed evidence of relapse during the follow-up of at least 8 months. The authors concluded that magnetic brain stimulation was easy to perform, painless, and safe.

Inflammation. The longer inflammation persists after even a mild neurological injury, the more damage is done to nerve tissues, which may become ultimately unrecoverable. In this context, an animal study of TBI explored whether PEMF signals could alter the course of inflammation. Cytokine IL-1ß levels in cerebrospinal fluid (CSF) were proportional to injury severity in brain bruise injury. PEMF treatment applied continuously reduced IL-1ß levels by up to ten-fold in CSF within six hours after injury, and significantly suppressed IL-1ß within 17 to 24 hours after injury.

Ischemia, ten minutes post-blockage. In an animal study involving blockage of the carotid artery, PEMF treatment at 75 Hz was initiated ten minutes after onset of ischemia and continued throughout the recovery of the blood supply. The PEMF did not reduce the total area of injury evident on MRI. However, PEMF reduced brain edema by 65%. Subsequently, the PEMF-treated group evidenced double the recovery, 55% vs. 28%.

Post-stroke, three to four weeks previous. Forty-eight patients (average age 45-48 years old) three to four weeks post-stroke were divided into two groups and provided with the same rehabilitation program. Patients in the treatment group were additionally exposed to a standard series of 10 ELF-EMF treatments, for 15 mins at 5 mT (50 gauss), 40 Hz. The PEMF group had double the amount of BDNF (brain-derived neurotrophic factor), 50% increases in VEGF (vascular endothelial growth factor), and 2.5 times more gene expression. Clinically, the PEMF group averaged 35% better cognitive functioning and 45% better depression scores. In the non-PEMF group, stroke scale severity and function measures were typically 65% and 50% worse, respectively.

Post-stroke, six months previous. A study of higher-intensity, low-frequency TMS PEMF for stroke investigated the long-term behavioral and neurophysiologic effects of combined HI-PEMF and physical therapy (PT) in chronic stroke patients with mild motor disabilities more than six months post stroke. In this study, 30 patients were enrolled in a double blind, randomized, single-center clinical trial. Patients received two PEMF sessions per day, 25 minutes each, 1 Hz high-intensity PEMF over the intact (not affected) motor cortex with standard task-oriented upper limb exercise PT. The real treatment group had greater behavioral and neurophysiologic improvements, particularly in the group receiving treatment before PT, with robust and stable improvements.

Post-stroke, two to twenty-four months previous. In a lower intensity PEMF study, 95 patients with spastic paralysis received treatment three times per day using 40 Hz, 10 mT (100 gauss), for 12 minutes, over 21 days, and repeated every six to eight weeks. Patients also received medication and physical therapy. Improvements in actively treated patients included significant decrease of muscle tension (88%), increased muscle contraction force (76%), increased active motion of paralyzed limbs (74%), improved gait (71%), and decreased aphasia (64%).

As a magnetic field passes through the body, that field can stimulate a range of potential processes and activities.

Post-stroke, one to ten years previous. Another study of stroke rehabilitation using low-frequency PEMF treated patients who had incurred a stroke years earlier (within the past 10 years). During a 15-day rehab hospitalization, each person received 22 treatment sessions of 20-minute low-frequency PEMF and 120-minute intensive occupational therapy (OT) daily. The PEMF of 1 Hz was applied to the side of the head opposite the area of the stroke, i.e., on the same side as the paralysis. Improvements were seen up to four weeks after discharge in 39% of patients. Researchers concluded that a 15-day inpatient PEMF treatment plus OT protocol is clinically useful neuro-rehabilitation for post-stroke patients with upper limb paralysis.

TBI Study. A pilot study was recently completed with a group of ten individuals with a history of concussion/TBI. In some cases, the injuries were more than ten years old. Using a medium intensity 10 Hz PEMF device applied to the head (front to back or side to side), patients were treated for two hours a day for three months. Everyone in the study noticed benefits. An objective measure of brain function was used to gather information before and after treatment and showed clear improvements over baseline scores, as well as important clinical improvements and better cognitive focus. Also note that research has reported improvement in post-concussion headaches treated with PEMF for as little as 30 minutes.

In situations involving neurological insult such as a stroke, treatment with a PEMF device for even short durations may have positive implications for recovery and for the treatment of acute stroke. While it is difficult for the medical system to be able to treat people in the acute phase of brain injury, whether it is stroke or trauma, if an individual owns a PEMF system, it can be used immediately and continually to reduce the extent of the damage. This is critical in improving the prognosis and in reducing the length of time for more complete recovery.

Full references are available at www.DrPawluk.com.

PEMF

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Resources

PEMF Devices. Dr. Pawluk has purchased, tested, and validated approximately 100 different PEMF devices over the 30 years he has worked in this field. Based on his experience, a number of devices are recommended and available for purchase on his website, drpawluk.com. They include local and whole-body systems, both battery and AC-powered devices. Intensities of devices range from very low intensity (<1 Gauss), medium intensity (10 – 1000) Gauss, to high intensity (2000-8000 Gauss), including devices that are able to cause muscle contractions.

Consulting services. Dr. Pawluk provides consulting services to both consumers and practitioners. Consultations are recommended for practitioners to be able to have more certainty about the most appropriate tools for the professional setting. Many professionals spend much more than they need to for PEMF systems. On the other hand, many professionals also acquire PEMF systems that are not likely to provide much benefit quickly in the practice setting.

Website. drpawluk.com is an extensive resource for patients, with basic educational information, more than 40 blogs with references, numerous videos, and a virtual store from which devices may be purchased. Once devices are purchased Dr. Pawluk and his staff provide significant ongoing support for both initial use and subsequent informational needs.

Book. *Power Tools for Health* is a readable, highly referenced work on basic concepts in the application of PEMFs, mechanisms of action, an extensive clinical section on 50 different health conditions, and more than 500 references on laboratory findings and clinical trials.

William Pawluk, MD, MSc, is a holistic physician located in Townsend, Maryland, in the Baltimore metropolitan area, with past appointments at Johns Hopkins University and the University of Maryland. With a background in family medicine, he has additional training in functional medicine, nutrition, acupuncture, homeopathy, hypnotherapy, bodywork, and energy medicine. He is the foremost authority on pulsed electromagnetic field therapy in North America, with 30 years of experience in clinical applications of PEMF for healing, regeneration, and holistic pain management. Author of *Power Tools for Health*, a comprehensive book on the research and usage of PEMFs in healing, he has also written professional book chapters and numerous journal articles. and provided more than 50 interviews for radio, podcasts, magazines, and TV. He has been cohost of a two-hour holistic health radio show for the past ten years and host of the Pain Solution Summit, www.painsolutionsummit.com. In 2019, Dr. Pawluk received the ACIM Lifetime Achievement Award for work with magnetic field therapy.

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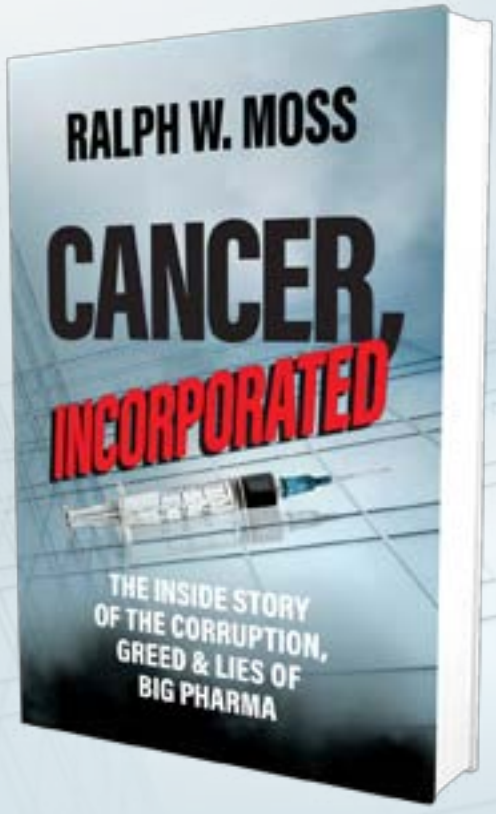

Dr. Jacob Schor Retires from the *Natural Medicine Journal*

For the past decade, the *Natural Medicine Journal* has enjoyed the deft expertise and intense commitment of Jacob Schor, ND, FABNO, as Abstracts & Commentary (A&C) Editor. Effective August 1, 2020, he will be retiring and leaving his post that he has nurtured carefully over the years.

"It has been very rewarding for me to be a part of the extensive growth that *Natural Medicine Journal* has experienced over the past ten years," says Dr. Schor. "It has gone from being a small naturopathic journal in 2009 to a respected peer-review publication with an extensive readership that includes holistic medical doctors, chiropractors, nurses, nutritionists, and many other integrative medical professionals. We have been able to showcase the talent of the naturopathic community to a very broad audience and I am proud to have been a part of that mission."

"Dr. Schor has been key in shaping content at *Natural Medicine Journal*," says Editor-in-Chief Tina Kaczor, ND, FABNO. "His breadth and depth of knowledge, stemming from more than three decades as a clinician, is unparalleled."

"While the *Natural Medicine Journal* will continue to deliver valuable, clinically-relevant content, Dr. Schor's ability to put context to research findings will certainly be a tough act to follow but we feel we have an outstanding replacement that even Dr. Schor will approve of," says Dr. Kaczor.

Dr. Tina Kaczor on a hike with Dr. Jacob Schor

Dr. Schor and Karolyn

Dr. Schor, and his wife Dr. Rena Bloom

Dr. Schor's new found interest

Every month, the *Natural Medicine Journal* site has more than 160,000 unique visitors with nearly 900,000 total impressions. In addition to being the official journal of the American Association of Naturopathic Physicians, the *Natural Medicine Journal* is sent to 31 different non-profit trade organizations and universities. Dr. Schor has been instrumental in that growth.

Dr. Schor received his naturopathic doctorate from National College of Naturopathic Medicine and opened his practice in Denver, Colorado, in 1992 with his wife Dr. Rena Bloom, who is also a naturopathic physician. Both retired from clinical practice last year. Dr. Schor also has advanced training in oncology as a Fellow of the American Board of Naturopathic Oncology.

Dr. Schor's commitment to the naturopathic community has been extensive and includes a past presidency of the Oncology Association of Naturopathic Physicians (OncANP) and a past board seat with the American Association of Naturopathic Physicians (AANP). He is also a past recipient of the AANP's Vis Award, which acknowledges doctors who represent the healing power of nature in their work, personal life, and community.

Natural Medicine Journal is proud to announce that Lise Alschuler, ND, FABNO, long-time editorial board member and contributor to the journal, will take over the role of Abstracts & Commentary Editor.

"Dr. Alschuler brings much expertise and we are thrilled she will be joining our team," says publisher Karolyn Gazella. "In addition to her clinical knowledge and keen understanding of the scientific literature, her close working relationship with myself, Dr. Kaczor, and Dr. Schor will help ensure that Dr. Schor's legacy of A&C excellence will be carried on."

"Dr. Schor is a curator of ideas, especially in his editorial work for the *Natural Medicine Journal*. He has artfully, generously and insightfully shaped what we pay attention to through what he shares with us," says Dr. Alschuler. "His impact on integrative and naturopathic medicine practitioners is tremendous. I am deeply grateful for his contributions and I am inspired by his fine example as I begin my new editorial role at the *Natural Medicine Journal*."

"While we all understand that Dr. Schor's retirement is much deserved, he has become a close friend and we will miss our weekly interactions with him," says Karolyn.

"I had a patient once say to me that work is only considered work if you'd rather be doing something else. With that sentiment in mind, I realize that I've never really *worked* with Dr. Jacob Schor," says Dr. Kaczor.

Dr. Schor's retirement will be filled with what he loves most: time spent with family, in nature skiing or kayaking, and mastering his basket weaving craft.

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The Institute for Functional Medicine and the American Nutrition Association Release New Online Nutrition Course

The Institute for Functional Medicine (IFM) and the American Nutrition Association (ANA) have announced a new online course, *Therapeutic Food Plans: A Component of Personalized Nutrition*. The course, offered through IFM, with the first cohort scheduled for October 12–November 26, is designed to educate and support nutrition professionals and other clinicians on the functional medicine collaborative care team about how to personalize and apply IFM's suite of therapeutic food plans.

The goal of this course is to provide clinicians with the tools needed to empower patients to improve their health with personalized food plans and the skills to maintain positive, long-term lifestyle habits. Clinicians who have completed the *Therapeutic Food Plans* course will be listed in ANA's Practitioner Finder directory if they meet eligibility requirements.

"At ANA, we believe that nutrition belongs at the core of health care – and that *personalized* nutrition most powerfully taps our health potential," said ANA CEO Michael Stroka. "This new course helps nutrition professionals customize food plans to each individual, a critical element of personalized nutrition."

This interactive, online learning course provides tactical strategies for increasing point-of-care skills and tips for keeping patients engaged and accountable for their health. It also provides the necessary tools to address modifiable lifestyle factors that may be influencing dietary patterns. Upon completion of this course, clinicians will be able to design personalized, therapeutic food plans for their patients, taking into account their unique dietary needs and preferences.

"Nutrition professionals play an integral role in the collaborative care team model," said IFM CEO Amy R. Mack. "This course equips them with the clinical strategies and therapeutic tools to tailor nutrition plans that can support each patient on their journey to improved health."

This new course is intended for nutrition professionals and other clinicians who are applying IFM's food plans in practice. After completing *Therapeutic Food Plans: A Component of Personalized Nutrition*, functional nutrition–oriented RDs, CNSs, and nutritionists with advanced degrees will be prepared to contribute to collaborative care teams with other IFM-trained practitioners. Additionally, physicians and other healthcare practitioners who regularly provide nutrition advice to patients will find this course useful, as it works to personalize nutrition within the IFM framework.

Registration for this course is now available at IFM.org/food-plans.

About IFM

The Institute for Functional Medicine (IFM) is the global leader in functional medicine and a collaborator in the transformation of health care. IFM is a nonprofit organization that believes functional medicine can help every individual reach their full potential for health and well-being. Founded in 1991 and dedicated to the widespread adoption of functional medicine, IFM works to advance education and training, clinical patient care, research, and outcomes in functional medicine worldwide. To date, IFM has educated over 16,000 practitioners from 70 countries. For more information about IFM, please visit IFM.org.

About Functional Medicine

Functional medicine determines how and why illness occurs and restores health by addressing the root causes of disease for each individual. The functional medicine model is an individualized, patient-centered, science-based approach that empowers patients and practitioners to work together to address the underlying causes of disease and promote optimal wellness.

About the American Nutrition Association

The American Nutrition Association (ANA) is the professional association for the science and practice of personalized nutrition. A nonprofit, the ANA envisions a society of healthy people, powered by nutrition. The ANA educates, certifies, advocates, and connects to fulfill its mission to champion the science and practice of personalized nutrition. For more information, visit theANA.org, and continue the conversation on Facebook, Twitter, Instagram, and LinkedIn. ◆

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Pathways to Healing

by Elaine Zablocki

Metabolic Flexibility for Long-Term Health

Nowadays many people are using a diet that's high in healthy fats and low in carbs, called the ketogenic diet. Naturopathic physician Bonnie Nedrow, ND, has been working with her patients for 20 years to develop a version of this process that she calls Keto-Cleanse.

The Keto-Cleanse program is a medical tool to help people recover their health and stay healthy as they age. By following all the program components, people optimize their biomarkers and reduce their risk of chronic disease. "Many people think of Keto-Cleanse as a weight-loss program," Nedrow says. "I believe people are overweight because they are toxic, overstressed, have poor life-style habits and don't eat enough vegetables. While people often do lose weight on my programs, I consider that simply an objective measure of improved health, not the goal."

During the 8- to 12-week program a person eats a very low-carb diet. "Humans have evolved to survive, and indeed thrive, on a diet that oscillates between abundance and scarcity," Nedrow writes. "On feasting days what we can't readily use gets stored as body fat. These fat stores can then be used as fuel in times of food scarcity. This is metabolic flexibility: the ability to access fuel from either dietary intake or from bodily stores."

However, our bodies only burn fat after using up all the carbohydrates we've eaten recently. In our society we eat carbohydrates all the time, so we've lost our innate ability to burn fat. Keto-Cleanse is designed to re-establish the ability to burn fat for fuel. It is a strict diet, based on lots of healthy fat and very limited carbs.

Important note: you only do Keto-Cleanse once during your lifetime. At the end of the process, participants have developed metabolic flexibility. "As long as you follow some simple steps to maintain your ability to use fat as fuel, you will be keto-adapted forever," Nedrow writes. "I do not recommend staying on the ketogenic diet forever.... The goal of metabolic flexibility is to stay keto-adapted forever, while having the freedom to come and go from a state of ketosis."

New Book on Metabolic Flexibility Puts it All Together

Nedrow, who practices in Petaluma and Santa Rosa, California, decided to develop Keto-Cleanse as she came to understand the relationship between weight loss, fat mobilization and an increase in circulating toxic compounds.

Bonnie Nedrow, ND

She first realized how essential it is to cope with toxins during medical school, when she became ill from chemical exposure in the anatomy lab. She wrote her first two books, *The Cleanse Companion Cookbook* and *The Seasonal Cleanse Workbook* to support her patients in cleansing. "I was motivated not only to create something for my own practice, but also to write cleanse books other practitioners could use with their patients," she says. "I strongly believe we need to focus on detoxification with all our clients and I wanted a cookbook and a workbook to make cleansing more accessible."

Nedrow has developed a significant focus on preconception optimization. "We are seeing an epidemic of chronic disease, not only in the elderly but also in younger people and even children," Nedrow says. "When I sit with a 50-year-old suffering from chronic disease, I wish I could turn the clock back. The more I study the roots of chronic illness, the more I see that it all starts much earlier than expected, with the health of the parents. I use both my seasonal cleanse program and my Keto-Cleanse program to help people optimize their health prior to conception, to help their babies enter the world with maximum health.

Seven years ago, Nedrow keto-adapted. "This was a huge game changer for my health," she recalls. Now she has published a new book, called *Metabolic Flexibility: How to Use a Ketogenic Diet to Heal Your Metabolism*, which is a remarkable asset for anyone who wants to try the ketogenic diet.

"I didn't even pause before beginning the creation of *Metabolic Flexibility* from day one of using it as a tool in my practice," she says. "Once again, I wanted to create a manual, this time to make a ketogenic diet safer and more accessible to the public and practitioners alike."

Nedrow's vision isn't limited to food intake. In this book she discusses the challenges that may arise during Keto-Cleanse, and offers an array of tools to cope with those challenges. *Metabolic Flexibility* includes valuable information about detoxification, exercise, adrenal rhythms, and life after keto-adaptation.

Detoxification

We are all exposed to a multitude of chemicals, man-made compounds with harmful effects on human health. They are metabolized in the liver. "If your liver is overwhelmed by chemicals that you are regularly exposed to, or by toxicants stored in your body, you may have difficulty keto-adapting," Nedrow writes. She recommends avoiding common chemical exposures as much as possible, drinking lots of water, and using supplemental fiber and minerals.

During Keto-Cleanse when you start burning body fat, you will mobilize fat-soluble toxicants stored in that fat. When you keto-adapt, it is essential to eliminate those chemicals. Studies have demonstrated that overweight people store fat-soluble chemicals and have a higher body burden than thin people despite a similar exposure history. "Mobilizing chemicals without removing them from your body can make you sick and can make you gain weight or stall your weight-loss program," she writes. She recommends daily detox vitamins to support the body during keto adaptation. A four-day fat flush is a strategy to bind fat-soluble toxicants and move them out of your body. It can be used before the ketogenic diet, or as a one-day process during and after keto-adapting.

Adrenal Rhythms, Stress and Exercise

Adrenal hormones support our circadian rhythms and allow for rapid fight-or-flight response in times of danger. "Chronic stress-induced insomnia turns our rhythms upside down, stimulating the mind when we should be resting and leaving us tired when we should feel refreshed," Nedrow writes. "In every 24-hour cycle there is an optimal time for rest and an optimal time for activity." She recommends turning off the computer and TV in the evening and going to bed early.

Exercise is an essential part of the Keto-Cleanse program. It quickly burns carbohydrates which forces muscles to burn fat and stimulates the liver to make ketones. Regular exercise makes keto adaptation faster and easier. *Metabolic Flexibility* discusses various forms of exercise, exercise programs for different ages, and additional training in balance and proprioception.

Chronic stress is one of the key contributors to chronic disease. "Unless there is time and support to fully recharge on a daily basis this type of stress will build," Nedrow writes. "A healthy lifestyle is the foundation for adrenal recovery. While herbs and nutrients support the adrenal glands, there is no magic bullet that will compensate for 'burning the candle at both ends.'"

Life after Keto-Adaptation

After completing Keto-Cleanse, participants will "embark on a more personalized health journey that will involve intelligent experimentation… you will identify those foods that are most helpful for you…" Nedrow writes. Some people will reintroduce healthy carbs. "Foods like root vegetables, fruits, whole grains and legumes are packed with vitamins, minerals, phytonutrients and fiber, all very important for ongoing health promotion…. You have been extremely vigilant and disciplined during adaptation; now it is time to move to moderation and balance in food choices." Each person will need to be aware of their individual reactions as they reintroduce high-carb foods. Some will choose to fast one day every week in order to reduce inflammation and optimize body composition through fat burning. Some will choose to follow a rotation diet with complex carbohydrates on three days per week, and a ketogenic diet on the other four days.

These are individualized variations that fit each person's body type and lifestyle.

How Practitioners Can Use This Book

Metabolic Flexibility is written for lay people who want to safely and effectively heal their metabolism with lifestyle modifications and a nutritional program. However, many people suffer from chronic disease processes, such as diabetes and heart disease, and will require more support. These people will benefit from the guidance of a clinician who can personalize the program and monitor progress with lab tests and other subjective and objective measures. The book is designed to guide the patient through the program while the practitioner supports them in their healing process.

Resources

Go to keto-cleanse.com/ for copies of:

Metabolic Flexibility
The Seasonal Cleanse Workbook
The Cleanse Companion Cookbook

The website also features a blog, a one-hour video about keto-adaptation, and a page of "Frequently Asked Questions" about naturopathic medicine.

For practitioners

If you are interested in selling *Metabolic Flexibility* in your clinic, please contact Dr. Nedrow for wholesale prices. You can reach her at DrBonnie@Keto-Cleanse.com.

Elaine Zablocki is the former editor of CHRF News Files. ◆

Literature Review & Commentary

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

Does Pomegranate Juice Improve Cognitive Function?

Two hundred sixty-one non-demented subjects (aged 50-75 years) were randomly assigned to receive, in double-blind fashion, 8 ounces per day of pomegranate juice or placebo for 12 months. Compared with placebo, pomegranate juice significantly improved the ability to learn visual information, although it had no effect on other measures of cognitive function.

Comment: One possible conclusion from this study is that drinking pomegranate juice had a positive effect on a specific measure of cognitive function. However, it is also possible that pomegranate juice had no effect, and that the difference between pomegranate juice and placebo was due to an adverse effect of the placebo. The contents of the placebo were not mentioned in the paper, but a personal communication with the lead author revealed that it contained high-fructose corn syrup, FD&C Red #40, and FD&C Blue #1. High-fructose corn syrup has numerous adverse effects on metabolism, and FD&C Blue #1 is a potent inhibitor of mitochondrial respiration *in vitro*. FD&C Red #40 might adversely affect brain function, since it appears to exacerbate the symptoms of attention deficit hyperactivity disorder in children. These ingredients were included in the placebo in order to mimic the taste and appearance of pomegranate juice. Similar placebos have been used in previous studies in which the active treatment was blueberry juice. It might be better to use something like apple juice or orange juice as a "placebo," even though the study would no longer be blind.

Siddarth P, et al. Randomized placebo-controlled study of the memory effects of pomegranate juice in middle-aged and older adults. *Am J Clin Nutr*. 2020;111:170-177.

Case Report: Food Allergy and Depression

A 34-year-old woman with a long history of major depressive disorder experienced symptomatic improvement on an allergy elimination diet. Individual food challenges implicated dairy products as a cause of symptoms. Improvement was maintained over a two-year period as long as the patient avoided symptom-evoking foods, but symptoms recurred on five occasions when she went off the diet.

Comment: Nutrition-oriented practitioners have long known that hidden food allergy can cause depression, but mainstream medicine remains mostly unaware of this association. This case report should remind us that food allergy should be considered in the differential diagnosis of depression, particularly in patients who have other symptoms that are sometimes due to food allergy, such as migraines, irritable bowel syndrome, asthma, perennial rhinitis, or recurrent infections.

Aucoin M, Bhardwaj S. Major depressive disorder and food hypersensitivity: a case report. *Neuropsychobiology*. 2019;78:249-255.

Gluten-Free Diet for Schizophrenia

Sixteen patients with schizophrenia or schizoaffective disorder who had elevated IgG anti-gliadin antibodies but a negative test for celiac disease (tissue transglutaminase antibodies) were admitted to an inpatient unit. They were fed a gluten-free diet and were randomly assigned to receive, in double-blind fashion, a daily shake containing 10 g of gluten flour or 10 g of rice flour for five weeks. Fourteen patients completed the trial. Compared with the gluten-containing diet, the gluten-free diet resulted in improvement on the Clinical Global Impressions scale, which is a scale that measures the overall response to treatment. Improvement was also seen on the Scale for the Assessment of Negative Symptoms, which measures the so-called negative symptoms of schizophrenia such as apathy, blunting of the emotions, social withdrawal, and impairment of cognitive function. The gluten-free diet also improved gastrointestinal symptoms, as determined by the Gastrointestinal Symptom Rating Scale. No improvement was seen in positive symptoms.

Comment: About one-third of people with schizophrenia have elevated levels of IgG anti-gliadin antibodies, which is a higher rate than in the healthy population. An elevated anti-gliadin antibody level is not by itself diagnostic of gluten sensitivity, but it has been reported to correlate with a higher likelihood of a positive response to a gluten-free diet in patients with conditions such as psoriasis and recurrent aphthous ulcers. In the present study, the magnitude of the benefit was calculated by dividing the mean difference between the two diets by the standard deviation. This is known as the Cohen effect size, which is frequently used in clinical research. The Cohen effect size for the various improvements ranged from medium to large, which is considered good for most non-pharmaceutical interventions. Especially noteworthy is the improvement in the negative symptoms of schizophrenia, because these symptoms often fail to improve with antipsychotic medication. The study did not examine whether patients who do not have elevated anti-gliadin antibodies could also benefit from a gluten-free diet.

Kelly DL, et al. Randomized controlled trial of a gluten-free diet in patients with schizophrenia positive for antigliadin antibodies (AGA IgG): a pilot feasibility study. *J Psychiatry Neurosci*. 2019;44:269-276.

Nutrient "Cocktail" Not Effective for Depression

One hundred fifty-eight Australian patients (aged 18-70 years; mean age, 42 years) with major depressive disorder who continued to have moderate-to-severe depression on their current antidepressant medication were randomly assigned to receive, in double-blind fashion, a combination of nutrients or placebo for eight weeks. The nutrient combination provided daily 800 mg of S-adenosylmethionine, 500 µg of folinic acid, 200 µg of vitamin B12, 1,000 mg of eicosapentaenoic acid esters, 656 mg of docosahexaenoic acid esters, 200 mg of 5-hydroxytryptophan, 30 mg of zinc, 100 mg of vitamin B6, 60 mg of vitamin C, 40 mg of magnesium, and 40 IU of vitamin E. Placebo was nonsignificantly more effective than the nutrient combination, as measured by the Montgomery-Asberg Depression Rating Scale (MADRS) and the Beck Depression Inventory Rating Scale-II. The number of responders (defined as at least a 50% reduction in the MADRS score) was nonsignificantly higher in the placebo group than in the nutrient combination group (51% vs. 40%).

Comment: In this study, a "shotgun" approach using a combination of nutrients was not more effective than placebo in patients with major depressive disorder who had not responded adequately to antidepressant medication. To the contrary, there was a nonsignificant trend toward the nutrients being less effective than placebo. This finding is surprising, since many of the nutrients used in this study have demonstrated an antidepressant effect when administered individually. The authors suggested that the unusually high placebo response rate seen in this study may have masked a potential benefit of the nutrients. Another possibility is that some nutrients interfered with the positive effects of other nutrients. Over the years, a number of studies that investigated drugs, nutrients, or herbs as a treatment for depression produced surprisingly negative results that did not conform to the larger body of published research. We still have a lot to learn about how to use nutrients most effectively to treat depression. The results of the present study suggest that we should not begin with a "shotgun" approach.

Sarris J, et al. Nutraceuticals for major depressive disorder - more is not merrier: An 8-week double-blind, randomised, controlled trial. *J Affect Disord*. 2019;245:1007-1015.

Omega-3 Fatty Acids for Epilepsy

Ninety-nine Sudanese patients (85 children, 14 adults) with drug-resistant epilepsy were randomly assigned to receive, in double-blind fashion, two, four, or six capsules per day (based on age) of docosahexaenoic acid (DHA)-based fatty acids, eicosapentaenoic acid (EPA)-based fatty acids, or placebo for one year. Each DHA-based capsule contained 418 mg of DHA and 51 mg EPA. Each EPA-based capsule contained 386 mg of EPA and 81 mg of DHA. The placebo was high-oleic-acid sunflower oil. The mean number of seizures per month was 9.7 with EPA-based capsules, 11.7 with DHA-based capsules, and 16.6 with placebo. After adjustment for age, gender, and seizure type, compared with placebo, the mean number of seizures was significantly lower by 42% with EPA-based capsules ($p = 0.008$) and by 39% with DHA-based capsules ($p = 0.04$).

Comment: This study suggests that supplementation with the omega-3 fatty acids present in fish oil can reduce seizure frequency in patients with drug-resistant epilepsy. Previous studies that examined the effect of these fatty acids in the treatment of epilepsy have produced conflicting results. There is no clear explanation why the results differed in different studies, and the mechanism of action of omega-3 fatty acids is not known.

Ibrahim FA, et al. The differential effects of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) on seizure frequency in patients with drug-resistant epilepsy - A randomized, double-blind, placebo-controlled trial. *Epilepsy Behav*. 2018;87:32-38.

Is Coenzyme Q10 Effective for Sepsis, or Is This More Iranian Research Fraud?

Forty Iranian patients with sepsis received standard treatment for sepsis and were randomly assigned to receive or not to receive (control group) 100 mg of coenzyme Q10 (CoQ10) twice a day for seven days. The primary outcome measures were changes in markers of inflammation and oxidative stress (interleukin-6, tumor necrosis factor-alpha, glutathione peroxidase, and malondialdehyde). Secondary outcome measures included in-hospital mortality. Compared with placebo, significant decreases were seen in mean levels of tumor necrosis factor-alpha and malondialdehyde. In-hospital mortality was significantly lower in the CoQ10 group than in the control group (20% vs. 65%; $p = 0.01$).

Comment: Readers of the *Townsend Letter* know that I suspect a large proportion of the nutrition research coming from Iran is fraudulent. Numerous aspects of the present study cause me to question its validity.

1. Inclusion criteria: The patients had to have an APACHE (Acute Physiology and Chronic Health Evaluation) II score of at least 15 to be eligible for the study. However, in Table 1, the mean APACHE II score in the group that received CoQ10 was 15.3, with a standard deviation of 5.2. With that mean and standard deviation, it is mathematically highly implausible that all of the patients had an APACHE II score of at least 15.

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2. Sample size: The paper stated that the target sample size was 60, but the registration document on the Iranian Registry of Clinical Trials (IRCT) website stated that the target sample size was 40.
3. CoQ10 dosage: The paper stated that the patients received 100 mg of CoQ10 twice a day, but the IRCT document listed the dosage as 60 mg twice a day.
4. Blinding: The IRCT document stated that the study was double-blind and that the control group received a placebo. However, the paper did not state that the control group received a placebo; it only stated that the control group did not receive CoQ10.
5. Secondary outcome measures: The IRCT document listed length of hospital stay and duration of mechanical ventilation as secondary outcome measures, but neither of these outcome measures was discussed in the paper. In contrast, the paper listed length of ICU stay as an outcome measure, but length of ICU stay was not mentioned in the IRCT document.
6. Exclusion of patients: Twelve patients were excluded from the analysis because blood tests were not obtained at the proper time. However, the researchers presumably had data for these 12 patients regarding mortality and length of ICU stay. These 12 patients should therefore have been included in the analysis of mortality and length of ICU stay.
7. Discrepancies about recruitment dates: The paper stated that patients were recruited between September 2016 and March 2017. However, the IRCT document, which was registered on July 6, 2015, stated that recruitment was complete. The IRCT document also stated, "Registered while recruiting." It is not possible for both statements in the IRCT document to be true. Moreover, both of the statements in the IRCT document contradict the recruitment dates reported in the paper.
8. Implausibly large effect size: The reduction in mortality seen with CoQ10 was very large; much greater than what would be expected from a single nutrient. There is only one similar randomized controlled trial in the medical literature: a study that examined the effect of ubiquinol (reduced CoQ10; 200 mg per day) in patients with severe sepsis or septic shock. In that trial, the mortality rate was 21% in the ubiquinol group and 11% in the placebo group.[1]

Soltani R, et al. Coenzyme Q_{10} improves the survival and reduces inflammatory markers in septic patients. *Bratisl Lek Listy*. 2020;121:154-158.

High-Dose Vitamin D and Multiple Sclerosis

Mice were fed a vitamin D-deficient diet (less than 5 IU per kg of food), a diet with a standard amount of vitamin D (1,500 IU per kg of food), or a diet with a large amount of vitamin D (75,000 IU kg of food). The standard dose was designed to produce a serum 25-hydroxyvitamin D (25[OH]D) level of 40 ng/ml. The high dose was designed to produce a level of 100 ng/ml, and is roughly equivalent to 30,000 IU per day for a 70-kg human. After eight weeks on the diet, experimental autoimmune encephalomyelitis (EAE) was induced (EAE is an animal model of multiple sclerosis). EAE was the least severe in the animals given the standard dose of vitamin D. Vitamin D deficiency increased the severity of the disease, but the worst outcome was seen in the mice given the highest dose of vitamin D.

Comment: While animal models of multiple sclerosis are not necessarily relevant for humans, the findings from this study are consistent with an earlier double-blind trial in humans. In that study, 23 patients with relapsing-remitting multiple sclerosis received approximately 13,000 IU per day or 1,000 IU per day of vitamin D_2 for six months. The dose in the high-dose group was adjusted to maintain a serum 25(OH) D level of 52-70 ng/ml. After six months, the median amount of disability (as measured by the Expanded Disability Status Scale) was significantly worse in the high-dose group than in the low-dose group. In addition, 36% of the patients in the high-dose group suffered a relapse during the study, whereas no one in the low-dose group had a relapse.[2] These findings argue against the use of high-dose vitamin D to treat multiple sclerosis.

Hausler D, et al. High dose vitamin D exacerbates central nervous system autoimmunity by raising T-cell excitatory calcium. *Brain*. 2019;Jul 13:awz190.

Effective Treatment for Hidradenitis Suppurativa

In 2013, the authors described an effective new treatment for hidradenitis suppurativa. Twelve patients with axillary or perineal fistulas due to hidradenitis suppurativa underwent surgical excision or localized treatment. All patients were found to have IgG antibodies to brewer's yeast and were put on a brewer's yeast-free diet for 12 months. In every case the skin lesions regressed completely over the 12-month treatment period. All patients had an immediate recurrence of skin lesions after accidental or deliberate ingestion of beer or foods containing brewer's yeast.[3]

The present study was a follow-up of the original report and includes a total of 37 patients with hidradenitis suppurativa who followed a yeast-free diet. Twenty-six patients (70%) reported an improvement without any other treatment. In 21 of these 26 patients, improvement occurred in less than 6 months. Of the 37 patients who followed the diet, 32 (87%) had a recurrence after eating a restricted food, often within a few days after eating the food.

Comment: Hidradenitis suppurativa is a chronic painful acneiform condition affecting the axillae, groin, and other areas that contain apocrine glands. It is caused by occlusion of follicles, which leads to inflammation and infection of the surrounding tissue. Results of conventional therapy, which may include antibiotics, glucocorticoids, retinoids, or surgery, are frequently disappointing. The findings from the present study suggest that sensitivity to yeast is an important contributing factor in patients with hidradenitis suppurativa. Consumption of a yeast-free diet results in clinical improvement, and in many cases may obviate the need for surgery.

Aboud C, et al. Treatment of hidradenitis suppurativa: surgery and yeast (Saccharomyces cerevisiae)-exclusion diet. Results after 6 years. *Surgery*. 2020 Feb 22 [Epub ahead of print].

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3. Cannistra C, et al. New perspectives in the treatment of hidradenitis suppurativa: surgery and brewer's yeast-exclusion diet. *Surgery*. 2013;154:1126-1130.

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Vaccinations, Vitamin C, Politics, and the Law

by Thomas E. Levy, MD, JD
Orthomolecular Medicine News Service

The ability of properly dosed vitamin C before and after a vaccination to block any potential short- and long-term toxic side effects while enhancing the antibody-forming aim of the injection was covered in an earlier OMNS article.[1] The points made in that article, along with their validation in the scientific literature, remain pertinent eight years later. As emphasized in that article, the purpose of this article is not to praise or condemn the administration of vaccinations. Most who know me know my stance on this issue, coming from what I consider to be a purely scientific perspective. But that is not the purpose of this article.

Even the most ardent of vaccine supporters should be able to admit that vaccinations, however infrequently, sometimes result in an undesired outcome on the health status of the recipient. The "argument" then shifts as to whether the chance of such an outcome is so rare as to be reasonably and permissibly ignored. The pro-vaccine community maintains that population-wide vaccinations confer a high degree of protection against even more morbidity and mortality that would otherwise be inflicted by the diseases or conditions for which the vaccines are being given. However, here I emphasize the need for mitigation of the side effects of vaccination.

To fully appreciate the toxic origins of all disease, it is very important to understand the critical roles played by vitamin C and other major antioxidants in counteracting and minimizing the impact of new toxin exposures and old toxin damage. All toxins, whether originating from an infection, food or water intake, or environmental source, or even as an unintended consequence of a vaccination, inflict their damage on the body by directly or indirectly causing the oxidation of critical biomolecules in the body. When biomolecules are oxidized (depleted of electrons), they lose some or all of their natural chemical roles throughout the body and inside the cells. Essentially, oxidation inactivates the natural physiological role of a biomolecule.

Biomolecules include proteins, sugars, fats, enzymes, nucleic acid, or structural molecules.

Depending on the chemical nature of a given toxin, it will oxidize a unique array of biomolecules, varying in location, concentration, and degree. These are the primary factors that determine the resulting clinical medical condition. And the presence of sufficient antioxidants can either prevent this oxidation from taking place by directly donating electrons to the electron-seeking toxins, or it can repair the toxin-oxidized biomolecules by contributing electrons back to them (reduction). However, it is essential to realize that the oxidized state of the biomolecules **is** the disease, or toxicity. There is no additional ill-defined "disease" that is impacting the cells and tissues with the increased numbers of oxidized biomolecules. Once enough biomolecules are restored to the reduced state, "disease" no longer exists.[2]

With this concept of the etiology of all diseases in mind, it can then be appreciated that any potential toxic side effect of a vaccination (or any other toxin exposure) can literally always be blocked or rapidly repaired by counterbalancing it with sufficient levels of antioxidants (primarily vitamin C). When this is combined with the established concept that vitamin C is essential for a fully competent immune system capable of producing an optimal antibody response to an antigen presented by a vaccination, it is only logical that optimizing the vitamin C status of a baby, child, or adult (as with influenza vaccinations) should always be an essential clinical goal.

Numerous articles in the mainstream medical literature clearly indicate that the infectious diseases for which vaccinations are given are effectively prevented by vitamin C.[1-3] Furthermore, when vitamin C levels are low in the body, infectious diseases are effectively eradicated by vitamin C-centered protocols (that can include other anti-pathogen interventions).[1-5]

The pharmaceutical industry makes billions of dollars on selling vaccinations and continuing to develop new vaccines for different conditions in an ongoing basis. However, in the current environment of politics, big business, and the law, such billion-dollar businesses will arguably *never* lose. They also will never be minimized, and their profits will never be substantially decreased. Such businesses have many congressmen and senators owing them for campaign contributions, along with many judges as well. Furthermore, most of those politicians and members of the judicial system actually believe that opposing vaccinations is tantamount to opposing the most significant intervention available supporting optimal public health. Many of us understand how much this enrages those who are convinced that vaccinations are doing a great deal of harm.

With all of these issues in mind, the overriding concern is how to protect as many babies, children, and adults from any possibility of a negative vaccine side effect. Even if, say, 10 years from now the scientific community finds that some vaccines are doing more harm than good and largely eliminates them, how many more lives (and families) will have been devastated in the meantime? Whether autism ever results from vaccination is actually not the primary issue. The issue is how to protect the infants that will be vaccinated *today*. All old and new evidence of any vaccination-induced toxicity should continue to be revisited and given its due publicity. But protection needs to take place *now*.

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Also, while it will likely displease most of the anti-vaccination community, a successful vitamin C-centered vaccine-protection protocol will make the vaccine manufacturers look like the good guys. Very few individuals will sustain side effects, and the vaccine companies will ultimately be given credit for making "better and safer" vaccines, and they will ultimately make more money rather than less. However, and this cannot be overemphasized, the vaccine damage will drop, and even largely disappear. The immediate protection of everyone's health has to be the top priority.

My personal recommendations for an effective program of toxin protection with optimization of a vaccine antibody response are as follows.

Start the supplementation at least seven days before a planned vaccination (the longer, the better since everyone at any age should regularly supplement vitamin C). This regimen should be followed as well on the day of vaccination and continued for at least one week following the vaccination. However, it will be best to continue the recommended vitamin C dosing for life.[1-5]

For infants and very young children, 1,000 mg of liposome-encapsulated vitamin C. This can readily be mixed in a flavored yogurt or other favorite baby food. This higher dose is possible relative to the sodium ascorbate powder below since liposomes only rarely cause the loose bowel effect seen with higher doses of vitamin C. Liposomes also allow a much better intracellular uptake of vitamin C to occur.

When liposome-encapsulated vitamin C is not available, proceed with sodium ascorbate powder (this can also be done in addition to the liposome form for even better protection). Infants under 10 pounds can be given 500 mg daily in a favorite juice (just a salty taste). For infants between 10 and 20 pounds, this can be increased to as much as 1,000 mg daily, in divided doses. Very roughly, the daily amount of non-liposome-encapsulated vitamin C can be increased by 1,000 mg per year of life.

Magnesium chloride can significantly augment the anti-toxin and pro-immune effect of vitamin C.[6,7] Mix 25 grams in a quart of water. Depending on body size, give 1 TBSP to 1/2 cup (15 to 125 ml) of this solution at least once and preferably twice daily in the days leading up to vaccination. As with vitamin C, the solution is salty (and a little bitter) and to be palatable is best diluted further in juice.

Vitamin D3 and zinc supplementation can also afford additional benefits. Again, depending on body size, 1,000 to 25,000 units of D3 can be given daily. These doses should *not* be continued in small children beyond a week after the vaccination. However, D3 is a valuable supplement, and it is of value for everyone. Long-term dosing requires validation that the regularly administered dose is raising the blood level to the range of 50 to 100 ng/ml. Ten to 50 mg of zinc (as zinc gluconate or other well-absorbed form) daily can be given by pill or drops for the week before and the week after vaccination. Long-term supplementation with zinc (and D3) should be done in concert with the advice of your integrative physician.

Finally, if a vaccination simply can't wait, taking the recommended doses of vitamin C, magnesium chloride, vitamin D3, and zinc the same day or just following the vaccination, and continuing for several weeks can also offer enormous protection. The above regimen simply aims to help optimize the protection being provided.

An addendum regarding a practical treatment approach to coronavirus, currently at epidemic levels in China:

1. If the virus is actually as contagious as is being currently asserted, modern air travel and the purported time of incubation and asymptomatic status (about 2 weeks) means it can spread anywhere on the planet. As with nearly all other contagious viruses, spread is most commonly due to airborne virus in microdroplets from sneezing, coughing, and the exhalation of infected individuals. Similarly, when the virus gets on the hands in a sufficient amount, touching the nose, eyes, and mouth can initiate the process of transmission as well.
2. The measures that most readily inhibit transmission include regular hand washing or sanitizing, containment of the microdroplets with high virus concentration (protective masks), and avoidance of areas with multiple infected individuals. And even when these measures do not completely block the transmission of virus, they massively decrease the amount of viral exposure, and a strong immune system will often do the rest.
3. A strong immune system is really the only significant protection an individual has, unless, of course, an individual can completely eliminate the possibility of virus exposure, which is virtually impossible.
4. A great deal of immune system strength, possibly most of it, comes from the vitamin C content in the immune cells. When the levels of vitamin C in the body are low, the immune system can never function at full capacity. There are many measures that can strengthen and support the immune system, but regular supplementation of vitamin C with multi-gram doses (2,000 mg daily or more) is probably the single most important preventive measure. *Much larger doses* can be given if it is determined that the virus has already been contracted. If IV vitamin C is available, this is optimal. But always take as much as can be afforded and tolerated, in both liposome-encapsulated form and sodium ascorbate powder and by intravenous administration.
5. Follow the magnesium chloride regimen discussed above but take the recommended doses four times daily during a time of active infection.
6. The virus grows rapidly in the mucosa of the naso- and oropharynx. It is this quickly growing "reservoir" of virus that continues to feed the viral presence throughout the body and sustain the infected state. Nebulization with 3% hydrogen peroxide quickly destroys all or most of this source of virus, and the body, with the help of vitamin C and magnesium, can then "mop up" the rest of the virus and rapidly accelerate clinical resolution. This is arguably the *most important* intervention to rapidly eradicate any systemic viral infection. Also, if available, ozone treatments of the blood can further accelerate the clinical resolution of infections such as the coronavirus.

Note: By way of disclosure, I am a paid consultant to LivOn Labs. I am only comfortable recommending their liposome-encapsulated products, including vitamin C. Although "liposome" products are available from a variety of other vendors, many contain no liposomes at all. Also, contrary to popular belief, there are no liposomes in many homemade versions of "liposome" vitamin C. (See my article https://www.peakenergy.com/articles/nh20140411/Exposing-the-truth-about-liposomal-nutrients)

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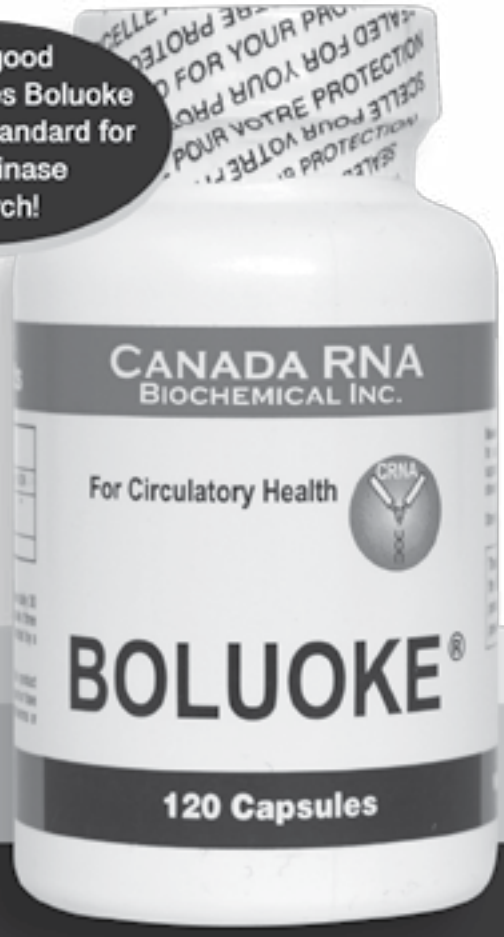

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CRP Screening Protocol to Help Control the Spread of COVID-19... and the Next Pandemic

by John Morgenthaler and Paul Taylor, PhD

We propose a protocol of testing for C-reactive protein (CRP) and other markers of early immune activation on all people determined to be at risk of exposure to COVID-19. The protocol could improve the efficiency and reduce the cost of mass testing for COVID-19 and may be useful as a screening pre- or post-travel through airports.

Incorporated into a contact tracing program,[1-3] all contacts of a person known to have COVID-19 could be screened for CRP, and perhaps also contacts of contacts. We will show that this protocol can add further precision, efficiency, and safety to contact tracing and can even catch probable asymptomatic cases.

CRP does not diagnose COVID-19 but, rather, signals early immune response. CRP can be elevated in a number of pathologies involving inflammation, including recent viral exposures. Therefore a single normal test result means the subject is unlikely to have COVID-19 and an elevated result means the subject might (but does not necessarily) have COVID-19. Finally, a history of normal results with a new-elevated result means something new is going on and it could be COVID-19. In this way the results of CRP testing can be used to stratify people more precisely by probability of being infected.

Suppose a patient is determined to have COVID-19 and the contact tracer begins to contact everybody who that patient has had contact with, and their contacts, branching down the tree as far as necessary. Each contact is mailed a CRP home test kit. The CRP test (for the purpose of detecting COVID-19 infection) will give false positives but not false negatives, and can be utilized to make contact tracing more efficient as follows:

- If, for a particular contact, the CRP is normal, it can be assumed that this contact's immune system is not highly activated. If it is low or normal, the probability that this contact has acquired COVID-19 is low, so nothing more needs to be done at the moment.
- If the contact does have elevated CRP then they should be asked to stay at home for 14 days or until further testing shows they do not have COVID-19.
- If a contact has a prior history of CRP tests, then a baseline has been established and any abrupt increase out of the ordinary for that contact signals possible viral infection and they should be asked to stay at home for 14 days or until further testing shows they do not have COVID-19.

There may be environments in which it would be prudent and cost effective to run weekly CRP tests on everybody. A work environment, such as a factory employing hundreds of people, could run CRP tests weekly and maintain a database of results. If any individual shows a breakout of CRP above their personal baseline, then they can be pulled off the job for quarantine and further testing.

The test is inexpensive (retail cost is $29 – $86), widely available, and relatively non-invasive (finger prick), allowing it to be employed on populations even where the risk of COVID-19 infection is much lower than would give cause for virus testing. Mailing CRP home-test kits to contacts, vs. taking a swab or blood for COVID virus testing, avoids unnecessary exposure for contact tracing personnel and increases safety for all parties.

CRP can alert the contact tracing worker to even probable asymptomatic cases, and at early stages when it might not otherwise seem that there is cause for a COVID-19 virus test.

Studies have shown that CRP goes up sharply in active symptomatic cases of COVID-19.[4-6] Higher levels were found to correlate with a worse illness later. One study reported patients with more severe disease symptoms had significantly higher CRP levels (average of 39 mg/L) compared with patients with mild symptoms (average CRP level of 19 mg/L).[4]

CRP is used along with other blood parameters in China as part of COVID-19 triage.[7,8] When CRP is high they consider it more probable that the patient has COVID-19, and they track these patients through the system differently.

CRP becomes elevated as early as day 2 and peaks within two to four days, after people become infected with other respiratory viruses such as influenza A, influenza B, and rhinovirus.[9] In contrast, testing for presence of virus with reverse transcriptase polymerase chain reaction (RT-PCR) yields 20% false negatives even at day eight making CRP screening useful even when done in conjunction with RT-PCR.[10,11]

CRP can also be elevated in asymptomatic carriers of COVID-19: Researchers in China documented a case of asymptomatic COVID-19 who, at day 14 after exposure, had no lab abnormalities except for elevated CRP (10.5 mg/L) and low lymphocyte counts. The patient was not brought into the

hospital for testing until that point in time so it was not known when his CRP began going up.[12]

CRP Screening for Travel

Health screening measures are sure to become a prominent part of the travel experience and, indeed, have already begun:

- Emirates became in April the first airline to test passengers for COVID-19 before check in. For travelers departing Dubai, Emirates' rapid blood test was designed to provide results in as little as 10 minutes.
- Etihad announced that it was evaluating in Abu Dhabi a new touchless, self-service technology[13] from Australia. Forming part of the check-in process, it would capture temperature, heart rate, and respiratory rate of passengers and reveal a current infection.
- In June the International Air Transport Association (IATA) released criteria for the use of COVID-19 testing in the travel process: Testing must be quick, scalable, accurate, and cost-effective.
- Hawaii's governor recently announced that, starting August 1, out-of-state travelers arriving in Hawaii must provide evidence of a negative PCR test result obtained from an entity approved by the Hawaii State Department of Health. Without this, passengers arriving from out-of-state will be subject to the 14-day quarantine.

But with the US still rationing COVID-19 tests in some communities, it's unlikely that the country's airlines will soon be in a position to safely implement PCR testing of passengers for this highly contagious disease. And a test prior to arriving at the airport does not ensure against the possibility of an infectious status developing after the test.

C Reactive protein testing, however, could provide a cheap and safer adjunct to a pre-travel COVID-19 PCR test, or a confirmation of status after arrival at the destination. If the C-reactive protein test is negative, it could affirm the continuation of a non-infectious status after a prior PCR test. If the C reactive protein test indicates an infection, it could require a further action at departure, or oblige the arriving traveller to submit for a PCR screening before being released from quarantine. There is a variety of equipment providing rapid CRP testing from a drop of blood in as little as 1 minute.[14-16]

We believe CRP and other immune marker screening will add further precision, efficiency, and safety to contact tracing and to workplace, convention, and travel screening. We hope that public health administrators engage to get this protocol incorporated into contact tracing and screening immediately.

What Was That About "... the Next Pandemic?"

Epidemiologists have been warning for decades that pandemics will come, and we can expect more. It is just a question of when. In a world with overpopulation, low nutritional status, and air travel it is certain to happen.

When the next pandemic first starts, there will not be a test for that infectious agent. These things take time, as we have now become painfully aware with COVID-19. Vaccines take even longer. But CRP and other immune markers can signal likely infection.

We can think of COVID-19 as a fire drill. It can help us get ready for the next pandemic. It has been said by historians that each time there is a war, the military fights as if it were just like the last war. When the next pandemic comes, a supply of COVID-19 tests will not be useful but an established contact tracing system with screening of general immune markers will be helpful.

We propose getting a system of emergency response in place now that incorporates this testing so that it can be deployed with no delay when the next pandemic first begins.

All Clear

We also envision a software "app" for people who get regular testing. As long as immune activity markers remain low, the app would provide an "All Clear" badge that displays on one's cell phone. An app such as this could be used to grant entry to a building, workplace, or a convention, or merely to reassure others on social occasions. This is similar to, and adds reliability to, the current method of testing body temperature before entry to some venues.

If this software is combined with or synced with a personal health record (PHR), it could provide much data to be mined for further medical findings. We'll leave a full exploration of this topic for a future publication.

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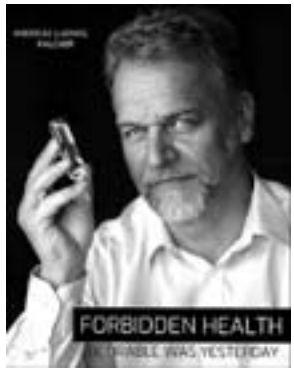

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Parkinson's Disease – The Vascular Connection and More

by L. Terry Chappell, MD

Parkinson's disease (PD) is the second most common central neurologic disorder after Alzheimer's disease. A million people have it in the United States. Six million carry the diagnosis worldwide. Primarily, PD is a movement disorder that results in tremors, stiffness, balance difficulties, and cognitive problems. Other organ systems in the body can be affected as well, including cardiovascular, peripheral vascular, respiratory, and urinary.

Levodopa is the most effective medication to control tremors and stiffness in the disease. Movement disorders are caused by decreased dopamine in the brain. Suspected underlying mechanisms are orthostatic hypotension, oxidative stress, and inflammation. L-dopa crosses the blood brain barrier and is converted into dopamine, which corrects a biochemical imbalance.

However, L-dopa has significant side effects, especially nausea and vomiting, which limits the tolerable dose. Cardiovascular side effects include aortic stiffness, elevated diastolic blood pressure, high LDL cholesterol, and increased homocysteine, especially the latter. Carbidopa has been added to the treatment specifically to prevent the side effects of L-dopa. It works by preventing the breakdown of L-dopa before it reaches the brain, thus requiring a lower dose for the L-dopa to be effective with fewer side effects. The carbidopa/L-dopa combination drug (Sinemet) is especially effective to reduce the cardiac side effects of L-dopa. However, the combo drug added its own set of side effects. See Table 1 for a partial list.

Table 1. Common Side Effects of Carbidopa/Levodopa (Sinemet)

- Repetitive movements of mouth, tongue, face, extremities
- Bladder pain, urgency, frequency, spasms
- Nausea, constipation, dyspepsia, belching
- Ischemic events, orthostatic hypotension, hypertension
- Headache, dyskinesia, dizziness, confusion
- Anxiety, depression, memory loss, hallucinations
- Rashes, wound infections
- Fatigue, weight gain or loss

Table 2. Heavy Metals, Vascular Disease, Other Neurologic Problems

- Sources of Lead and Cadmium: Water, air pollution, tobacco smoke, soil, old paint (lead), discarded batteries (cadmium)
- Mechanisms: Oxidative stress, decreased nitric oxide and glutathione, lower heart rate variability, induce epigenetic effects, raise blood pressure, cause kidney malfunction
- Consequences: Diabetes, atherosclerosis, heart attacks, strokes, peripheral vascular disease, Parkinson's disease, Alzheimer's disease, other neurologic problems

This article summarizes a number of treatments for Parkinson's disease that have beneficial cardiovascular effects, which in turn reduce the risk for heart failure, serious arrhythmias, myocardial infarction, and even death that Sinemet carries. If harmful effects are detected early and treated effectively, serious consequences might be avoided. PD patients should be carefully monitored for early signs of vascular disease. By paying attention to both neurologic and cardiovascular systems, better clinical outcomes are likely.

Physical Therapy and Boxing

Exercise is now the primary treatment for early PD. Currently, aggressive lifestyle programs, especially focused on exercise, have become the initial prescription. Thirty minutes of vigorous exercise is suggested daily. A skilled physical therapist teaches the patient a dozen or more exercises that counteract the stiffness and movement abnormalities characteristic of PD. A boxing regimen using a bag, with short bursts of energy is often included. Boxing is beneficial over a long period of time and also serves as an avenue for stress reduction. The PT/boxing program is more vigorous than the 150 minutes per week of aerobic exercise prescribed for cardiovascular fitness. This might be more effective. Boxing is particularly helpful and is often quite popular for PD patients. Of course, competitive boxing has just the opposite effect. Yoga, music therapy, dancing, and other activities that cross the midline also seem to have some benefits.

Metals Good and Bad

The CT scan for calcium score is a cost-effective screening test for coronary artery disease. An initial test is followed by a repeat test after one year. A high calcium level initially is suspicious, but a repeat test detecting a 15% increase from year to year is even more accurate for finding significant disease, which might require further testing and comprehensive treatment. A red cell magnesium level is a simple but important blood test. Less than optimal levels of magnesium are associated with many malfunctions in the cardiovascular tree. High levels of lead, mercury, and cadmium are showing up as major cardiovascular and cerebrovascular risk factors. The Trial to Assess Chelation Therapy (TACT) detected an impressive association between myocardial infarctions that was apparently due to toxic metals.[1] Unfortunately, blood lead testing is used for screening but is often unreliable. An exposure to lead stays in the bloodstream for

only two weeks or so. A normal blood test gives a false sense of security. An intravenous EDTA challenge test with urine collection is a much more sensitive tool to detect elevated body burdens of lead and other toxic metals. There exists an epidemic of toxic metals triggered by lifelong environmental exposures. These harmful metals are stored in the vascular system, bone, and brain. Once they are detected effective chelation treatments are available.

Previously, conventional medicine taught that PD was secondary to cardiovascular and cerebrovascular disease risk factors. The mechanism was thought to be conventional vascular risk factors, such as smoking, hypertension, inactivity, and high lipids. Recent evidence has shown that PD is actually associated with similar biochemical risk factors. The mechanisms are shared, but more likely to be poor glucose control, mitochondrial dysfunction, excessive reactive oxygen species (ROS), free radicals, and autonomic dysfunction. Toxic metals increase ROS and free radicals.

Exhausted Mitochondria

Mitochondria are organelles inside cells of the body that are most responsible for producing energy. As long as we have plentiful, healthy mitochondria, cells have a good enough supply of energy to function properly and age slowly. If mitochondria are exposed to excessive environmental risk factors and toxins, they cannot keep up with the energy required to do their job. Some of them die off and others become exhausted and function poorly. Over the years, various diseases develop, especially ones for which the patient is genetically susceptible. Common diseases such as Alzheimer's disease, PD, and heart diseases are particularly sensitive to mitochondrial dysfunction. Doctors skilled in complementary medicine utilize various tools and nutrient therapies that enhance mitochondrial function.

Until recently, it has been very difficult to know which patients have severe mitochondrial dysfunction and thus are in need of aggressive therapy. Patients with mitochondrial disease might not know they are headed toward serious illness. At an early stage they might not have any symptoms whatsoever. Frank Shallenberger developed a test to determine how well a patient's mitochondrial defense is working. He calls it "the Bio-Energy Test." For example, he has found that 46% of patients in their 30s have mitochondrial dysfunction that might raise their risk for eventual serious disease.[2] Fortunately, if those patients are treated, they will very likely be able to avoid expression of major illness, such as diabetes, Alzheimer's, Parkinson's, and heart disease. Clinics from around the United States now provide the Bio-Energy Test.

Other Treatments for Parkinson's Disease

Complementary medicine provides treatments that speak to the mechanisms that are linked to chronic vascular-related diseases. Some of them are chelation therapy, hyperbaric oxygen, ozone, UV blood irradiation, antioxidants, exercise with oxygen (EWOT), NanoVi, EECP, and IV glutathione. These therapies are given to patients for prevention or after symptoms have developed. Empirically, they usually are helpful for patients who receive them. Large clinical trials have not yet been funded. Other diagnostic and screening tests are available and are being utilized by alternative physicians. Two of them are the calcium score by CT scan and the CardioRisk carotid ultrasound test. As noted above, the TACT trial showed a nice reduction of repeat MI's in patients treated with chelation therapy. The mechanism of action in TACT appeared to be a reduction of toxic metals. However, other mechanisms are possible.

Jonathan Wright has reintroduced the work of Benjamin S. Frank, who used RNA nucleic acid supplements to get results for patients who suffered from various diseases including PD. A nucleic acid rich diet was also employed by Frank. His work was completed in the 1970s but continues by supplement companies. Rubidium drops and low-dose lithium have also been utilized by Wright. A rigorous protocol of a natural form of L-dopa, has been been a successful treatment of PD by Marty Hinz.[3] High doses of IV pushes of glutathione have brought PD under control, as directed by David Perlmutter. He is well known for the use of a ketogenic diet with his PD treatment. A number of neurologists have introduced neurofeedback treatments to improve brain waves with some success. Severe cases of PD have responded to surgically implanted, deep brain stimulators, as a last resort. These stimulators can be remarkably effective if needed.[4]

Conclusion

The mechanisms and risk factors for chronic neurodegenerative diseases such as PD frequently overlap with silent or overt vascular problems. The treatments mentioned above are under-utilized. We live in a toxic, stress-filled world. We need to do a lot more to identify the cardiovascular and cerebrovascular stresses we face, so we can live longer, avoid the silent killers, enjoy life, and function at a high level of wellness. I recommend guided exercise, boxing, and stress reduction programs for all patients. Home BP monitoring, a healthy diet with limited sugar, the calcium score, a CardioRisk test, a challenge test for toxic metals, the Bio-Energy test, and some form of bio-oxidative treatment. One must be very careful about potential side effects from medications prescribed by the treating neurologist. As briefly described in this article, many nutritional, vascular, and other complementary therapies for Parkinson's disease are available. Individualized treatment protocols are required for individual patients. With aggressive therapy, a PD patient might live a normal lifespan, with functions at a reasonable level.

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Evidence-Based Nutritional Interventions for Pediatric Brain Health

by Carrie Decker, ND

In the outfall of secondary events related to the pandemic, anyone who is a parent has likely had substantially more one-on-one time with their children. In addition to the typical activities of momming and dadding, suddenly there was the job of teaching as well. The difficulties of getting kids to do their homework and stay on task that seemed like a struggle back in 2019 really took on a whole new meaning in 2020.

Because of this, many parents have an increased interest in things that can be done to improve their child's cognitive function and attention span, and also have increased concerns about how the new life restrictions have impacted their child's mood as well. Herein, we look at key nutrients that support pediatric brain health and a healthy attention span, with additional considerations for mood.

Pediatric Nutritional Deficiencies

Nutrient deficiencies are prevalent in children worldwide, with iron, vitamin B6, vitamin D, folate, iodine, and zinc being among the most common in developed regions.[1-3] A 2017 review of nutritional deficiencies in US children reported that in addition to these deficiencies, vitamins A, C, and E, calcium, and magnesium are also under-consumed.[4] Though all children are at risk, the incidence of nutritional deficiency is especially high in children from low-income regions. Sub-optimal nutrition also predisposes children to neurocognitive deficits like attention deficit hyperactivity disorder (ADHD).[5,6]

Iron deficiency is particularly common in children with ADHD. In one study, done in Paris, France, serum ferritin levels were shown to be abnormally low in 84% of children with ADHD compared to only 18% of controls.[7] Additionally, low ferritin levels were correlated with more severe ADHD symptoms and greater cognitive deficits. A 2017 meta-analysis further demonstrated an association between low ferritin levels and ADHD.[8]

Iodine is another nutrient that is important for normal cognitive development. With many individuals opting for more natural salts (rather than iodized table salt) or to reduce the intake of salt altogether, iodine deficiency is resurging in developed countries. Unfortunately, many of the problems related to iodine deficiency in utero and infancy affect brain development.[9] Because of this, it is very important to ensure maternal iodine adequacy during pregnancy and breastfeeding.[10]

Inadequate iodine intake in childhood and adolescence can also lead to impaired cognitive function.[11] A 2016 study found 71.9% of 89 children with ADHD to be iodine deficient, and further revealed a significant association between low iodine levels and hyperactivity.[12] An increased incidence of ADHD and lower intelligence quotient scores also has been shown in the offspring of mothers at a risk of iodine deficiency during pregnancy.[13]

Lower levels of vitamin D have been shown in children with ADHD as well.[14] Again, a recent meta-analysis also points towards this association, finding that a lower vitamin D status is associated with an increased likelihood of ADHD.[15]

Magnesium and zinc deficiencies also have been shown in children with ADHD and are correlated with hyperactivity, inattention, and impulsivity.[16-18] Meta-analysis show a relationship between low levels of these minerals and ADHD as well.[19-21]

In addition to these many vitamins and minerals, numerous studies have shown lower blood levels of omega-3 fatty acids in individuals with ADHD.[22,23] Healthy children who have low levels of docosahexaenoic (DHA) have been shown to have below average reading ability, diminished working memory performance, and increased oppositional behavior and emotional lability.[24]

Nutrients with Therapeutic Evidence

Although a nutritional deficiency state should be remedied for a multitude of reasons, correcting it does not necessarily mean it will resolve the characteristic behaviors of ADHD or improve cognitive function in general. Thus, we must also consider what research supports each of these as a therapeutic.

Iron. In children, the Recommended Daily Allowance (RDA) of iron varies by age, ranging from 7 to 11 mg per day. Higher doses are often used to resolve iron deficiency; but before this course of therapy is embarked upon, testing must be done to assess for deficiency.

In children with ADHD and low ferritin levels (25.9±9.2 ng/mL), supplementation with 5 mg/kg/day of an iron preparation for 30 days improved ferritin status to 44.6±18 ng/mL and significantly improved Conners Rating Scale (CRS) scores, given by parents, of behavioral symptoms; however CRS scores given by the teacher were not improved.[25] In another study, non-anemic children with ADHD and serum ferritin levels lower than 30 ng/mL were treated with 80 mg/day of ferrous sulfate or placebo for 12 weeks.[26] Although there were improvements in both the parent and teacher CRS scores, improvements did not reach significance (P=0.055 and 0.076, respectively). The small size of this study (N=23) likely was a factor in the lack of significant findings.

In non-anemic adolescent girls with iron deficiency, supplementation of 650 mg of ferrous sulfate twice weekly for eight weeks significantly improved test scores related to verbal learning and memory.[27] Other studies also point to some improvements in cognitive function or psychomotor development with iron supplementation.[28-30]

Iodine. The majority of studies investigating the impact of iodine supplementation on pediatric cognitive function look at iodine-repletion programs in iodine-deficient areas as opposed to routine, daily intake of a specifically prescribed amount of iodine.[31] That said, there are some studies looking at iodine as a monotherapy or in combination with other nutrients that are common deficiencies.

In one such study of iodine-deficient youth, repletion of iodine and increased urinary iodine levels were associated with significant improvements in mental performance after roughly a one-year period.[32] In moderately iodine-deficient children between 10 and 12 years of age, a single 400 mg bolus of oral iodine significantly improved performance scores at 24 weeks (versus placebo) related to information processing, fine motor skills, and visual problem solving.[33] In another, similar setting, moderately iodine deficient children (6 to 8 years in age) given a single 490 mg dose of oral iodized oil were observed to have improvements one year later in fluid intelligence, perceptual skills, and hand-eye coordination.[34] Finally, in a study where schoolchildren were provided with a seasoning powder containing 5 mg of iron, 5 mg of zinc, 50 µg of iodine and 270 µg vitamin A with their school day lunches, there was an improvement in visual recall along with a reduction in symptoms of upper respiratory tract infection and diarrhea at 31 weeks.[35]

Typically, the best way to ensure nutritional needs are being met in children is with a quality multivitamin and mineral supplement.

Vitamin D. Multiple studies have shown a positive impact of regular vitamin D supplementation on behavioral symptoms and cognitive function in children with ADHD. Vitamin D, of course, also plays an important role in the mental health of children and adults.[36,37] Although four of every 10 toddlers and children are deficient in vitamin D,[38] a deficiency state should be documented prior to supplementation of vitamin D exceeding the RDA, which is 600 IU/day for children ranging from 1 to 17 years of age.[39]

In vitamin D deficient children with ADHD, supplementation of 3000 IU of vitamin D3 daily, in addition to methylphenidate treatment, for 12 weeks significantly improved cognitive function scores on the domains of conceptual level, inattention, hyperactivity, and impulsivity compared to baseline, while scores in these categories did *not* significantly improve in children receiving placebo with methylphenidate.[40] Additional studies have also shown improvements with vitamin D supplementation as an adjunctive to treatment with methylphenidate.[41-43]

Magnesium and Zinc. Magnesium is probably the mineral we look to most often for "turning the circuits down" – e.g., it has therapeutic usefulness in adults for reducing anxiety, blood pressure, headaches, and muscle twitches, and promotes relaxation and healthy sleep.[44,45] We see similar effects in children: daily supplementation of magnesium in pediatrics decreases migraine-related disability, also reducing anxiety and depressive symptoms and improving psychosocial well-being.[46]

Multiple studies have shown that adding 200 mg of magnesium daily to the therapeutic regime for children with ADHD improves outcomes, decreases hyperactivity and impulsivity, and improves cognitive function.[47,48] The combination of magnesium with vitamin B6 (6 mg/kg/d of Mg with 0.6 mg/kg/d

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of vitamin B6) has also been shown to be effective, improving hyperactivity, aggressiveness, and attention at school.[49]

In addition to its impact on cognitive function and development,[50] zinc deficiency also can be a factor in mood disorders such as anxiety and depression.[51] One consideration with zinc as a therapy is that if higher doses are taken for a prolonged period, it should be balanced at roughly a 10:1 ratio with copper.[52]

In one study, unmedicated children with ADHD were randomized to receive 150 mg of zinc sulfate (providing 40 mg of elemental zinc) or placebo for a period of 12-weeks.[53] At the end of the study period, children receiving zinc had significant improvements in hyperactive, impulsive, and impaired socialization symptoms compared to the placebo group. Improvements were greatest in children that were older, had a higher body mass index, and/or lower pretreatment zinc or free fatty acid levels. A shorter six-week study considered a lower, 15 mg daily dose of elemental zinc (55 mg of zinc sulfate) or placebo as an adjunctive to methylphenidate.[54] Although both groups saw improvements as they were initially treatment (including medication) naïve, significantly greater improvements in both parent and teacher scores of ADHD severity were reported in the children receiving zinc compared to placebo.

Omega-3 Fatty Acids. In addition to these essential vitamins and minerals, which have systemic implications when there is deficiency, the omega-3 fatty acids DHA and eicosapentaenoic acid (EPA) are essentially important to the health of every cell in the body. They have particular importance for the fatty tissues of the brain where they are found at high levels and impact not only inflammation and cellular membrane function but also neurotransmission.[55-57] Omega-3 supplements, typically sourced from fish such as salmon, sardines, anchovies, or mackerel, have a high safety profile in individuals of all ages.[58,59]

Numerous systemic reviews and meta-analysis have looked at the impact of omega-3's on the cognitive and behavioral performance of children with ADHD. A 2018 publication reviewing randomized, controlled trials found that omega-3 supplementation significantly improved clinical symptom scores and cognitive measures associated with attention in youths with ADHD.[60] A larger, 2015 review that also included healthy, typically-developing youth found that omega-3 supplementation improved short-term memory in those with low omega-3 status.[61]

A three-arm study compared treatment with a blend of omega-3 and omega-6 fatty acids versus methylphenidate or methylphenidate plus omega-3/-6 fatty acids, finding that over the course of the one-year study all groups had similar improvements in mean ADHD Rating Scale total scores and multiple subscores. However, improvements were typically seen more rapidly and were greatest in the groups receiving methylphenidate.[62] Additionally, in the group receiving both methylphenidate and omega-3/-6 fatty acids, a lower dose of the medication was needed to achieve the same level of clinical improvement as those using the medication alone. Adverse events were less frequent in the groups receiving omega-3 and -6 fatty acids.

Studies have also shown that symptoms of depression and anxiety in children also may improve with supplementation of omega-3 fatty acids.[63,64,65] Given their high level of tolerability and safety, omega-3 fatty acids are important to consider, particularly in children and adolescents with low levels of dietary intake.

In Closing

Typically, the best way to ensure nutritional needs are being met in children, especially picky eaters, is with a quality multivitamin and mineral supplement. A 2017 meta-analysis supports this, concluding, "Eight of ten trials assessing fluid intelligence reported significant positive effects of micronutrient supplementation among micronutrient-deficient children, especially those who were iron-deficient or iodine-deficient at baseline."[66] In times of additional life challenges, it also is important to note that increased intake of many of these nutrients has a positive effect on mental health, improving depression, anxiety, and related behaviors.[67-70] Beyond this, the additional nutrients with clinical research should be considered, often starting with omega-3 fatty acids due to the high level of evidence of their therapeutic value. ◆

Full article with references posted online at www.townsendletter.com

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Behavioral Changes Resulting from Microbiome Restoration: An Integrative Veterinarian's Perspective

by Margo R. Roman, DVM

Abridged: Full article with appendices and references are posted online at www.townsendletter.com.

Introduction

Modern medicine, both human and animal, acknowledges the need for a well-functioning microbiome to help protect the immune system and maintain health. Recent research indicates that a great deal of immune response derives from the gut. This research supports the claim that medical conditions may arise from damage to the microbiome. An individual may struggle with gastrointestinal (GI) issues, allergies, and a weakened immune system after exposure to antibiotics, other drugs and chemicals, environmental toxins, and a poor diet. Can we regenerate immune strength from that weakened condition? Evidence is now growing that such damage may be ameliorated by fecal transplantation, and this therapy, while still in its infancy, is being increasingly acknowledged in both human and animal medicine. In the near future, replenishing the GI tract with beneficial symbiotic gut flora that have supported the donor with good health may be the way we will treat many diseases.

In 2012, this author coined the term "microbiome restorative therapy" (MBRT), as opposed to the customary term "fecal microbiota transplantation" (or FMT), to better describe what the therapy actually does: restore the microbiome. This term was first published in articles by this author in 2014 and 2015. Some clarification may be in order regarding this change in terminology. The term "microbiome restorative therapy" can be considered a more palatable and accurate description of this restorative process, especially as defined and practiced by this author's clinic. Also, while fecal transplants have been used in humans and farm animals for years, until recently this therapy had not been used in small animal medical care. MBRT was developed at Main St. Animal Services of Hopkinton (MASH) for small animals and involves an *explicit, prescribed, holistic protocol not found in descriptions of FMT* (see Appendix 1). In fact, this therapy is now being followed in a growing number of holistic veterinary practices. The term "microbiome restorative therapy" and its acronym MBRT are increasingly referenced in veterinary information blog posts and YouTube presentations, and now at least two peer-reviewed veterinary publications. These terms are regularly included in veterinary and microbiome conference lectures and discussions and, in various veterinary contexts, seem to be accepted as standard nomenclature.

The novel and interesting observations we have made of the behaviors of many of the animals to whom we have administered MBRT are among the numerous positive possible outcomes of fecal transplants. What we have witnessed in our veterinary clinic, read in related credible reports, and have been informed about by our clients and others are intriguing and distinct behavioral or even personality changes in MBRT recipients. While we do not know why these behavioral changes occur, we posit that microbiome restoration appears to be at least in part responsible for this effect. This paper offers a viewpoint based on these clinical observations, some scientific reports, and the personal observations of others, and asserts the opinion that this therapy is worth developing further, not only for its physical healing qualities, but also for wider observation and documentation of accompanying behavior and personality changes. A better understanding of the role the gut plays in emotional and psychological well-being is timely and needed, and can inform the possibility of incorporating fecal transplants in behavioral therapeutic approaches for animals as well as humans. The "One Health" concept is a relevant paradigm from which to approach this issue. Recent research regarding the direct relationship between the gut and the brain sheds more light on this very topic. These kinds of studies seem to validate earlier clinical observations on the gut/brain connection, and more analysis can lead to continued illumination on this topic.

Holistic practitioners recognize that there are inherent limitations with double-blind, placebo-controlled studies, and that an integral part of gathering widely usable data is the inclusion of clinical reports, which often emerge first from informal clinical observations. From the perspective of an integrative veterinarian

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who has practiced for over 40 years, this author and her clinical colleagues have observed an added dimension of mental health and behavioral improvements within just a few hours or days of this novel treatment, and appreciate this as a significantly different consequence than the typical positive outcomes of other holistic treatments. This paper presents a unique viewpoint based on such novel clinical observations, a first step toward formulating a new hypothesis as well as formal case studies.

A better understanding of the role the gut plays in emotional and psychological well-being is needed.

Relevant Human Cases

Accounts in several published articles, both scientific and subjective, and at least one book describe effects in some organ transplant recipients in which they seem to suddenly take on emotions and characteristics unlike their own, but very similar to those of their donors. This effect has been documented in heart, lung, kidney, liver, and other organ and body part transplant patients (although the most dramatic and long-lasting effects seem to occur with heart transplants). This intriguing side-effect of organ transplants raises interesting questions regarding the nature and mechanisms of some kind of cellular memory that would generate such changes. Likewise, when we hear compelling subjective accounts of similar personality changes after fecal transplants in humans, the resulting questions become even more profound (see Conclusions below). It is not meant to be construed from this paper that all human cases of FMT will result in personality or behavioral changes; but these changes may be possible and should be inquired about by medical personnel shortly following FMT administration in humans.

It is this author's opinion that providing this information regarding behavioral effects in humans as a result of fecal transplantation is an important segue to any discussion of analogous responses in other mammals who cannot explain in words what they are experiencing. For the purposes of this paper, very brief summaries of these effects from human FMT are as follows, from patients who learned only minimal information about their donors at the time they were given the transplants, which, if any bias were present, does not seem to account for all of the responses each have described. In the case of humans, it is possible that placebo and "honest placebo" effects could have played a role in the improvements reported here. However, the effects reported by humans are similar to those observed in animals, in which placebo effects are not applicable. *Just as reported human effects are helpful in understanding the effects in animals, observed effects in animals can serve as a partial validation of reported human effects.*

Ms. M. A 35-year-old woman, a client of this author, had an autoimmune disease with a lot of gut issues. She became worse after expensive treatments with a conventional specialist. Alternative support was not improving her the way she had anticipated. She then read about fecal transplants and wanted to try this therapy. Because she did not have *Clostridium difficile*, she was not approved for this procedure as, in the United States, the requirement is for three episodes of *C. difficile* to qualify for insurance coverage, and doctors would therefore not perform the therapy for her. However, she eventually found a doctor who was doing a transplant research project. This doctor gave her one treatment in an office visit and subsequently mailed her two more capsules at different times for a total of three treatments.

The first transplant was from a screened and tested very happy, healthy, athletic 11-year-old girl who had been raised in a clean environment, was never on antibiotics, and ate only an organic whole food diet. In recounting her experience, Ms. M said she felt really light afterward, felt very happy and almost carefree. She attributed this significant change in subjective feelings to the addition of a young, healthy microbiome. After a while, this euphoric feeling faded, but she still felt much better than before the transplant. When she received the second transplant, it was from a 25-year-old woman, and while she did feel stronger physically, she had no significant change in emotions, positive or negative. When she later had the third transplant, she knew it was from a screened and tested healthy male but that is all she knew. That night and for the next few days she said she was sweating copiously, and her body odor was that of a man, which she intensely disliked. She was also up all night feeling much stress, anxiety, and upset. Ms. M contacted the doctor to find out if this was a normal reaction. The doctor had never seen this kind of reaction with this donor he had used many times but disclosed that the male donor was a body builder and he had just broken up with his girlfriend and was quite upset. Was the transplant responsible for these intense physical and emotional effects, as this patient thought was the case? This woman's health has improved but she has not been able to retain her microbiome balance for long periods of time. This is most likely due to the immune endocrine imbalance (atypical cortisol estrogen imbalance syndrome [ACEIS]) with which she was diagnosed (see Appendix II). Therefore, she is still seeking other ways to strengthen her microbiome as she realizes how it affects all of her.

Mr. G. Another client, a 43-year-old man, recently submitted a written testimonial regarding his own positive emotional and other effects as a result of fecal transplantation. This man described himself as suffering since childhood with "an undiagnosed mental/emotional condition" in which "my thoughts and emotions seem different from what is reality" and "my emotions become my new reality." After many years of seeking relief through alcohol, psychiatric treatments, and pharmaceuticals, all of which did not work well for him, he was able to start FMT treatments. He reported that he noticed an immediate change in his patterns of thinking, which allowed him to have better perspective and control over the destructive emotions. This change has also enabled him to seek other ways to further support his mental health as well as to follow a diet that will support a healthy microbiome. He said the effects of MBRT last about two weeks,

when some of the racing thoughts and other symptoms start to return. He stated in writing, "When I am consistent with doing the fecal transplants, my life, career, and relationships are AMAZING."

Mr. G offered a log of a recent transplant experience with a 12-year-old girl donor who is a gymnast and trains hard. He immediately felt a shift in his energy and felt very relaxed. He slept well that night and recalled dreaming of Barbie dolls. At the gym the next day, he noticed his reaction to some parts of his workout had shifted, and that some of his movements were different. After another transplant from the same donor, he reported feeling more comfortable with other youngsters with whom he had been working, while playing games with them. That evening, he mentioned to his wife how he feels like a different person after his transplants, and then "thanked God for showing me what my life can be like when I'm doing fecal transplants and when I am balanced in my brain."

Doris. Another case is that of a personal friend who was 86 when she endured two hospitalizations for *C. difficile* that almost killed her each time and left her with severe abdominal constipation pain, resulting in her inability to take her Parkinson's medication, which then resulted in an increase in Parkinson's symptoms and loss of mobility. She was on almost continuous antibiotics for urinary tract infections (UTIs) and was incontinent. Her family had determined that she needed to be in a nursing home as she had lost a lot of her mental capacity and appeared confused and demented.

When FMT was first suggested to this family, their reaction was disgust with the idea of this "gross procedure." After 15 months, the family finally decided to try FMT to see if this might correct her *C. difficile* overgrowth. Medicare will not allow FMT until there have been three separate hospitalizations for *C. difficile*. Doris was hospitalized only twice for *C. diff*, almost dying the second time, and stating she did not want this treatment again and would rather die. This author suggested the family pay out of pocket to get FMT because another hospitalization could weaken her more and could cost Medicare $30,000, based on the bills from her prior hospitalizations. Family members called around: Cedar Sinai in Los Angeles, California charged $9,000, Mayo Clinic in Scottsdale, Arizona charged $6,000, and Scripps Institute in San Diego, California charged $750. Doris went to Scripps where it took five minutes to complete the treatment with a nasal-gastric tube. The donor sample came from Open Biome in Medford, Massachusetts (www.openbiome.com). Open Biome screens their donors and at the time of this writing has sent out tens of thousands of transplant samples.

After five days, Doris could feel a big difference. Her stools were becoming more normal, and she could take her Parkinson's medication. D-Mannose was added to help her bladder infections and to keep her off antibiotics. She also said she actually could think a thought through. Five weeks later she was walking at the mall with her friends, with a ➤

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stronger bladder, feeling so much better, and with much greater mental acuity. By five months after the FMT she had regained all her brain capacity ("thinking like I was 50 years old again") and looked 15 years younger to the people in her life who love her. We do not know the age of the donor, but her GI problems stopped and her chronic UTI was gone. Her demeanor became happy and upbeat and her mental clarity returned. She had no need for a nursing home and was able to live independently. Doris has reported more than once that she is certain that she would have died had she received another round of conventional antibiotic treatment in the hospital, instead of receiving FMT. Now at 90 years old, she has fully maintained her physical and mental abilities, her Parkinson's symptoms have improved, and she has so much energy that she runs the activities center in her retirement community.

Animal Cases

Observations of behavior changes in animals usually occur within hours or 1-2 days of MBRT. The following are brief observational accounts of chief behavior and personality changes in some of our patients. Please note: In addition to MBRT treatments, an overall integrative therapy approach is provided to each animal, which includes several other holistic treatments. These combined treatment modalities appear to provide significant, healing synergistic effects, so are deemed crucial in our treatment protocols. Ozone insufflation, for example, is typically administered with MBRT in order to support the process, and we normally offer nutritional support, often nutraceuticals, acupuncture, and occasionally ultraviolet blood irradiation therapy and other treatments as needed (see more on this in Appendix I). *But it is clearly only after receiving MBRT in particular, that animals show a marked change in their behaviors.* This brief listing primarily reports these notable changes.

Mojo, a seven-year-old neutered male Maine Coon cat presented with a mangy coat from years of atopic dermatitis that had been treated with multiple courses of antibiotics, cyclosporine, antihistamines and steroids. Mojo received rectal MBRT from a one-year-old Siamese male kitten who had *just* been neutered. Within two days Mojo stopped scratching his face and was able to be relieved of his Elizabethan collar. In two weeks, his hair started growing back. Mojo showed a much happier affect than he had prior. Six weeks later the owner requested a second MBRT. Mojo continued to improve after the second fecal transplant, so his owner requested a third one six weeks after the second. This owner was a doctor and scientist and was curious about the process. She called two days after the third fecal transplant and said she thought Mojo had a problem with his anal glands as he was acting odd. Upon examination, the anal glands seemed normal. After discussion of his behavior, we realized that he was sexually mounting her arm. Had we given him the (still present) hormones and youthful vigor of a one-year-old Siamese cat who was the fecal donor? Mojo continues to improve and now has a beautiful coat. He has been consistently happier, along with much greater energy.

Archie, a seven-year-old neutered wire-haired fox terrier, presented with aggressive behavior and digestive issues. Pharmaceutical medications for his behavior failed, and he was kept on a very strict diet with lots of nutritional support for years and many other integrative approaches, but still was very aggressive with his sister. About 24 hours after his first MBRT, Archie was grooming and kissing his sister and could now eat foods he could never eat without them causing massive diarrhea. This remarkable behavior change stopped when he was given the heartworm preventive medication, *milbemycin oxime* (Interceptor). About 24 hours after this dosage, he attacked his sister, and his aggressive behavior returned. After another MBRT treatment, his improved behaviors resumed. It is logical that an anthelmintic could have the same side effect of destroying microbes in the gut as an antibiotic. This dog's case suggests that damaging the microbiome with any antimicrobial could create behavior and immune system issues. Because Archie showed symptoms of ACEIS (see Appendix II), he was maintained with multiple fecal transplants about every two weeks after that point and was taken off the heartworm preventative. Animals like this need to be maintained on microbiome support if there is any disruption to their gut. After eighteen fecal transplants, his chronic issues resolved completely after he was given the biome from a pregnant donor. What else is in the microbiome when the donor is pregnant? (See *Conclusions* below for further discussion on endocrine involvement.)

Rozzi, a six-year-old female golden retriever, had chronic urinary tract issues that were treated for years with antibiotics. This dog was so weak, the owner had decided to euthanize her. Rozzi was given an MBRT treatment and she regained her energy. As with others, we observed in Rozzi that when she was treated with fecal matter from a donor in estrus, she would mount everyone. She tested positive for ACEIS (see Appendix II), so she received MBRT treatments weekly to keep her strong for about a year, until she started taking Restore (www.ionbiome.com). When Rozzi started Restore (a human supplement, now called ION*Biome, that promotes an optimal gut environment), she seemed to hold onto the benefits of the MBRT longer than without the Restore.

Lola, a four-year-old corgi mix, was spayed as a six-week-old puppy at a shelter and never showed any signs of female traits. The owner requested that we give her MBRT to replenish a full range of microbiome, as she had been so depleted in her earlier life. She received MBRT orally from a dog who was in heat and the next day started sexually mounting and thrusting for the first time in her life. Lola was then happy and smiling for two years, until she was attacked by pit bulls, had to be dosed with antibiotics, and became fearful of many things. After another MBRT, Lola again became more loving and trusting.

Tober, an 11-year-old spayed calico cat, had suffered with constant diarrhea for over 18 months, with up to 10-20 stools a day, following years of on and off diarrhea. She endured extensive ultrasound, blood work, fecal exams, and years of intermittent *metronidazole*. This cat appeared very thin and fragile. Less than two days after MBRT, Tober's stools were normal and she, who used to hide in the basement and was afraid of the dog, was sitting confidently on the couch and playing with the dog. This behavior change and change of the stools shocked the owner, who even sent pictures of these

"perfect poops." Her owner said when she "picked her up she had always felt like a piece of paper and after just three days she has some body strength and seems so much happier." To have such a complete change in less than 48 hours in both her stool consistency and behavior after years of illness is very unusual, even if an animal is treated with standard holistic care for IBD. Tober continues with her contented affect, has gained weight, and has no diarrhea. She is being maintained on a high quality, protein-specific raw diet, and occasionally gets MBRT, as she has also tested positive for ACEIS (Plechner Syndrome).

Norton, an 18-month black Labrador, had been raised and trained as a dog assistant. Unfortunately, as a puppy and even with extensive training and good behavior, this dog was coprophagic and had some anxiety. His behavior as a dog assistant was fine but eating his stool as soon as it dropped to the ground was unacceptable to the organization, so they sold the dog to a caring family. For over six months this otherwise well-behaved dog still had some anxiety and his owners could not break his habit of eating his stools. One MBRT treatment and Norton never ate his stools again, and anxiety was no longer an issue. Four other cases of coprophagia also stopped right after receiving MBRT.

Dudley, a 10-year-old neutered male shih-tzu/poodle mix was diagnosed with multicentric, high-grade, T-cell lymphoma. He achieved two complete remissions after surgery, chemotherapy, autologous lymphoma vaccines, and integrative therapies. For most of the duration of his treatment period, Dudley received MBRT and other holistic therapies. The incidence of adverse effects during chemotherapy was low, and overall quality of life was excellent, with his survival of 2½ years post diagnosis far outlasting the prognosis of his oncology veterinarian and most dogs with T-cell lymphoma.The most impressive behavioral outcome his owners observed after each fecal transplant from a young donor was that he would play with a ball like a puppy for long periods of time, something he had not done for years. He was also the recipient of "microbiome mixology," in which he received the biome from our donor who survived cancer for 7½ years, as well as from her six-month-old grandson.

Kaylee, a seven-year-old beagle, was scheduled in 2017 for euthanasia by two veterinarians, including a board-certified veterinary oncologist, as she was dying from what he determined to be a hemangiosarcoma in her abdomen and leg (although she didn't have a positive biopsy, so this was never diagnostically confirmed). She received one MBRT along with other holistic therapies. In five days, she had a complete reversal of symptoms as well as a complete behavior change. Kaylee had always been a dog who was very insecure, afraid of water, and of leaving her owner's side. Nor did she exhibit other normal Beagle behaviors. Right after receiving MBRT, she started typical Beagle behaviors of sniffing and digging holes to chase rodents, chasing other wild animals, and jumping into water, and "smiling." The owner was shocked, as she never before could get Kaylee to go near water or even through a puddle, nor ever witnessed these other normal beagle behaviors. Kaylee's behavior became very similar to what the donor dog's behavior had been, including the smiling, which makes these observations even more curious. At last communication, now almost three years later, Kaylee was still thriving and acting like a puppy.

Baxter, a 14-year-old, 85-pound, lab/shepherd cross, was diagnosed with a liver tumor, (a suspected hemangiosarcoma) and subsequent complications, and presented with such weakness he had a hard time standing when he came to the clinic in August 2016, with euthanasia being recommended by the oncologist. We put him on an aggressive holistic program, and he progressed very positively as he gained more strength in his back legs and seemed to be feeling generally stronger. In the first week of November 2016 he had a massive bleed in his abdomen and was taken to an emergency clinic where they diagnosed that his mass had ruptured and he was hemorrhaging in the abdomen. With liver functions ten times normal, as well as his age, the recommendation was to euthanize him.

We decided to try intraperitoneal ozone gas in the upper flank area as well as other aggressive holistic therapies, and his lab values started to markedly improve towards normal. Baxter was still a little tired and showing his age. We gave him MBRT from a 2½ year-old female poodle in heat. The next day he was acting like a puppy, playing in the yard and sexually mounting on his bed, which he had not done since he was five years old. Each time he received a fecal transplant of youthful biome, he had youthful vigor and played like a young dog. Baxter passed away last year at 16½ years, several years after his expected demise.

Is the ability of the microbes to communicate with the body/brain affected by the hormones?

Rationale for MBRT

When any mammal comes through the birth canal, it gets what is essentially an inoculation of microbes. This action, called the "vaginal gulp," occurs because vaginal fluid excretes lactobacillus and other microbes as the fetus emerges. More exposures to microbes happen as the newborn nurses and later explores its environment. Chinese tradition recommends that women who had just given birth, not bathe for three months; this would allow the baby to get natural exposure to microbes while nursing and to naturally develop a strong immune system. In the wild, baby animals crawl around in a dirt den while the mother regurgitates the food she has eaten to feed her babies, or she brings recently killed prey for them. Recent research suggests that even prior to birth, fetuses ingest amniotic fluid which is the individual's first "meal" of microbes. All of this initial inoculation of the microbiome is the beginning of the individual's immune system. Any individuals not receiving these microbiome inoculations, as well as other environmental bacteria that would naturally occur in a healthy environment, may lose immune strength due to deficient exposure. In humans, full-term C-section babies, who do not get the initial inoculation from the birth canal, benefit from an oral transfusion of their mother's microbiome to allow this initial inoculation to occur. With the widespread use of hand sanitizers,

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bleaches, antimicrobials, and chemicals such as pesticides, preservatives, and herbicides within the home, many current newborns, infants, and pets have depleted numbers of microbes. In the case of animals, coprophagia may be a natural way for them to try to replenish their microbiomes. Giving them a healthy, balanced microbiome by transplantation can reset the gut flora and stop the coprophagia and eating of other undesirable things. Animals in the wild often eat particular plants, dirt, and other substances that provide certain nutrients as well as diverse bacteria as an additional way to balance the microbiome.

Expanding Research

Research into the microbiome continues to expand into emerging concepts concerning the gut/brain connection. Recent papers hypothesize about the role of neurotransmitters in gut/brain communication. These papers offer some explanation for some of our observations. For instance, in the case above of Kaylee, the beagle who had no interest in normal beagle behaviors until after receiving MBRT, this may have been enabled by stimulation of neural sensory (olfactory) circuits after the donor's microbiome restored some normalcy to the beagle's. New research also finds more evidence of the microbiome's role regarding inflammation in the body and effects on the brain. Certain food substances, such as gluten and casein, can cause inflammation and are now seen as probable culprits in some cases of depression, among other ailments in humans. Conversely, improved diets are found to directly and indirectly lead to better mental health and affect. We know that antibiotics and other drugs that are integral to conventional health care cause major disruption of the microbiome balance. Could this be causing, in large part, the epidemic of mental and physical health issues now plaguing our society?

Caveats About Donors

While science has much to learn about identifying the approximately 500 species and 1,000 subspecies or organisms in a healthy human GI tract, enough is now known about the microbiome and predictable clinical treatment outcomes, to determine **the vital importance of having pre-screened, healthy, happy, uncontaminated, and varied human and animal fecal donors**. It is also important that donors be clear of some of the chronic medical conditions we often see in individuals. Therefore, a standard screening of any animal donor should be undertaken for all different types of intestinal parasites such as hookworms, roundworms, whipworms, and tapeworms, and consider checking a PCR test for *Giardia, C. difficile, C. perfringens, E. coli,* and *Salmonella*. (Even these tests do not detect most of the 500 microbial species present in canines.) Additionally, it is important to ensure that exposure of the fecal donors to undeclared gut-microbe-killing chemicals or herbicides in their food does not occur.

The chemical glyphosate, found in the herbicide RoundUp, is ubiquitous in many processed foods, including commercial pet food, especially in non-organic food made with genetically modified products, and is a probable carcinogen. Additionally, this herbicide damages mitochondria, chelates minerals, and disrupts the detoxification pathways of the body. It also becomes a synergistic agent, making combinations of low-level toxins more damaging. This and all other pesticides and chemicals that kill plants and bacteria and other microbes must be eliminated from the fecal donor's diet. Making sure our pets eat a raw or other balanced diet with fresh, organically raised food without preservatives can increase the health of the gut flora. Modern society has been feeding companion animals depleted, contaminated, and in many cases inappropriate food (e.g., corn and wheat for cats, who are obligate carnivores). Generation after generation of loss of microbes is most probably a major part of the cause of so many deficiencies and chronic diseases now commonly found in animals.

How Long Does One MBRT Last?

In some transplants, after only one treatment, we have seen animals completely changed and healed. But there are others that need more than one, and one patient needed 19 transplants. The criterion for re-inoculation is a recurrence of symptoms. However, some owners of formerly very ill animals elect to give their pets MBRT orally on a weekly, bi-weekly, or monthly basis, as they see that their healthy state is better maintained. The addition of nutraceuticals allows the microbes to thrive and maintain the health status of the animal (see Appendix I). It is imperative that we remain aware of what chemicals the animal might be exposed to, in order to avoid disruption of the microbiome. Foods and chemicals that we should and should not give or expose to our patients, who have such delicate microbiomes, is another area to be explored and developed.

We have found that animals with ACEIS, or Plechner Syndrome (www.drplechner.com), seem to need repeated transfers. It

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is important to evaluate the IGA, IgG, IgM values, total estrogen, cortisol, as well as T3 and T4 to determine if an animal has this disorder so as to predict the success of MBRT treatments (see Appendix II for further discussion).

Conclusions

Study of the microbiome at this point in medical science creates more questions than conclusions. There are more than 100 trillion microbes in a healthy human being, microbes which had their origins about 3 trillion years ago. A large percentage of microbes is in the gut; however, there are microbes in every organ system. Medical science does not currently understand the full significance of each of the microbiomes throughout the body. Is it the organ that runs the system, or is it the microbiome within that organ that can affect good health? Through further observation as well as scientific research, we will uncover a better understanding of this primordial creation that is basic to all life.

What is vitality? What drives an individual to be healthy and have a positive mental outlook? Some call this the vital force, or life force. In a majority of our fecal transplant cases, the animals have become livelier, more positive, and seem happy, and it appears that their vital force has become stronger.

A consequence of better understanding the microbiome can be better healing and support for animals in health crises. The overuse of antibiotics, drugs, pesticides, herbicides, preservatives, and even chlorine and fluoride in water can affect the numbers and variation of the gut microbes. As the National Institutes of Health and other researchers continue to appreciate the value of the microbiome and how it can influence neurotransmitters, serotonin growth factors, and many other aspects of cellular communication and survival, we hope to be able to use that knowledge to help the body to repair itself.

Medical science is now looking at the brain/gut connection a lot closer than it had previously because researchers are finding that the precursors of many enzymes and neurotransmitters are within the microbiome, which can be a part of the answer to this brain/gut connection. An area that needs more science and exploration is how this brain/gut association is connected to actual behavior outcomes of MBRT. This would confer more validation on the consistent personal and clinical observations discussed in this paper, both subtle and dramatic.

Several questions have emerged from these observations of behavioral effects as a result of MBRT as well as other types of transplants: Is there a cellular memory that works within cells and stem cells, and possibly even in the microbial DNA, projecting not only just the functioning of those particular organs or microbes, but also that of the personality traits of the organ or fecal donor? What are the mechanisms that make this communication or projection possible? Could part of an explanation be a holographic framework within each cellular or microorganism DNA? Also, as each organ has its own unique microbiome in addition to the host cells, could each of these microbiome populations have inputs that effect responses in emotional or other ways? It is also imperative to rule out placebo and honest placebo effects.

In view of the consistent changes in behaviors of patients when receiving fecal donations from animals in estrus or pregnant, other related questions for further research include the following: How does the hormonal level of the animal in estrus or pregnant affect the microbiome? Does the activity of the microbes change in that case? Is the ability of the microbes to communicate with the body/brain affected by the hormones? And do the hormones affect the communications of the microbes with each other? Or, do excess hormones in fecal matter directly affect the behavior of the recipient? While more research is required to answer questions regarding the relationship between hormones and microbes, some of this research is addressed by Poutahidis *et al*.

In humans, can those with serious mental conditions and even those in prisons with certain personality disorders be helped by microbiome transplants? Can the loving behavior of a donor with a loving personality be passed through the microbiome? Can happiness and compassion be transmitted in this way?

There is much we do not know about ourselves, our world, and our universe. But we must appreciate the vast complexity of our world, and that science is always learning new facts, creating new theories, and tossing old ideas. It is the opinion of this author that we must respect what we do not know, and be open to new ideas, even if they are first based only on plausible clinical or subjective observations. Paying attention to the emerging science of the mind/gut connection may bring medical science into a new and fruitful paradigm of mental health care for animals as well as humans. ◆

Abridged: Full article with appendices and references are posted online at www.townsendletter.com.

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Multisensory Integration Often Overlooked for the Treatment of Brain Disorders

by Mary Ann Block, DO, PA

Brain disorders are increasing at an alarming rate. autism, attention deficit hyperactivity disorder (ADHD), dementia, Parkinson's, traumatic brain disorder (TBI), depression, anxiety, and post-traumatic stress disorder (PTSD) are all expected to increase by 8-18% in the next five years.

Autism, ADHD, dementia, depression, anxiety, and PTSD are classified as psychiatric disorders. They are all described and given codes in the Diagnostic and Statistical Manual of Mental Disorders (DSM). For most, there are no objective means to diagnose these disorders. They are defined by subjective symptoms.

The DSM states that psychiatric disorders are "…not due to the direct physiological effects of a substance or a general medical condition. This exclusion criterion is used to indicate that a substance-induced and general medical etiology must be considered and ruled out before the disorder can be diagnosed." Basically, it is saying that no psychiatric disorder should be diagnosed until all medical conditions are ruled out as causes.

Even though this is the criterion stated in the DSM, it is rarely used. In fact, the contrary is happening. Physicians are now being encouraged to give a psychiatric diagnosis without testing or ruling out an underlying medical cause. It appears to me that giving the diagnosis without evaluating someone for an underlying medical cause is a quicker and less expensive process.

In the journal, *Osteopathic Family Practice News* (2002),[1] Dr. Gregory James asserted that all adults should be "screened" for depression or anxiety during their periodic physical exams. He wrote that a nurse or medical assistant could do the screening. Such telltale signs as changes in hygiene, avoidance of eye contact, and responses to key questions were cited as potential indicators of depression.

In fact, the article said that neither a physician nor a nurse was required to make the diagnosis.[1] The patient himself could complete a questionnaire. The author referenced screening forms that could be used. These documents do not constitute a medical exam. They cannot be equated with lab tests or an MRI. They are only lists of symptoms to which the patient replies in the positive or negative.

The article identified risk factors for depression present in various diseases, conditions, situations, environments, and professions.[1] The author contended that genetics play a role because certain personality types are more prone to depression. Other contributing factors could include cancer, chronic pain, weight loss or gain, disability, sexual dysfunction, gastrointestinal problems, heart diseases, vitamin deficiencies, hormonal imbalances, and alcohol or drug abuse.

In addition, the author identified certain drugs such as blood pressure and anti-Parkinson medications, tranquilizers and others as increasing the risk of depression. He then described what he believed to be common symptoms of depression; fatigue, headaches, pain, sexual dysfunction and gastrointestinal problems were included. The author first wrote that these were physical symptoms that place one at risk for being depressed then turned around and named them again as actual symptoms of depression.[1]

This circular reasoning presents a real problem. If the symptoms of a medical condition cause depression, the physician would have to ignore that condition in opting to treat the depression with a psychiatric drug.

The standard treatment for most of these disorders is prescription medication. In no case, do these medications resolve the problems. The drugs might decrease the symptoms in some cases, but in every case, if you stop the drug, the symptoms will return. In some cases, such as Parkinson's, the medications will stop working and the symptoms return with no other options. The newer drugs for Parkinson's list dementia as a side effect, adding yet another brain dysfunction to the original one. The only medications approved for autism are antipsychotic drugs, which come with a host of serious side effects. Most prescriptions for ADHD are addictive, and those used to treat depression, anxiety, PTSD, and TBI have a risk of suicide attached to them. Are these drugs and the risks associated with them even necessary?

Anecdotal evidence, obtained from medical schools, indicates that up to 75% of medical students are taking a psychiatric medication. This is concerning because of the side effect risk of the drugs and because if medical students think these are appropriate treatments, are they not more likely to prescribe them to their own patients when they begin to practice?[2]

What are the underlying causes? Sensory dysfunction has been shown, in multiple studies, to be involved in each of the disorders.

Abnormal visual attention pathways were disrupted in those diagnosed with ADHD.[3] Dana Nicholls and Peggy Syvertson write:

> Learning and paying attention depend on the ability to integrate and organize information from our senses. Unorganized sensory input creates a traffic jam in our brain making it difficult to pay attention and learn. To be successful learners, our senses must work together in an organized manner. A person diagnosed with ADD or ADHD, due to their difficulty paying attention, may in fact have an immature nervous system causing dysfunction, making it difficult to filter out nonessential information, background noises or visual distraction and focus on what is essential.[4]

Tactile sensitivity is common in autism, and there are measurable differences in early auditory pathways, especially with increased stimuli. They often exhibit atypical visual behaviors and have an inability to filter simultaneous channels of auditory, visual, and tactile.[5] Children on the autism spectrum suffer with sensory issues, either too little input resulting in delayed development and disabilities or too much sensory input that overwhelms them and causes avoidance and withdrawal.[6]

According to Maria C. Carrillo, PhD, Alzheimer's Association chief science officer, new research finds that sensory impairments may also be associated with increased risk of dementia, especially when there are several of them at the same time and multisensory integration is beneficial to those with Alzheimer's.[7] Evidence suggests that hearing loss may predict or accelerate cognitive deterioration, and alterations of hearing may manifest as complex cognitive and behavioral symptoms relevant to the differential diagnosis of dementias.[8]

Parkinson's and other movement disorders have traditionally been considered to be disorders of impaired motor control. However, newer studies indicate the sensory aspects of movement disorders include intrinsic sensory abnormalities and acknowledge the effects of external sensory input on the underlying motor abnormality.[9]

Clinicians treating patients with traumatic brain injury (TBI) have noted that combat returnees frequently reported auditory, vestibular, and visual symptoms. These sensory issues are likely to negatively affect the ability of individuals with TBI to process cognitive information and perform daily tasks such as communication and ambulation. While researchers are still investigating the effects of sensory and communication disorders on rehabilitation outcome, preliminary data have shown that dual sensory impairment may adversely influence functional outcome in patients with TBI.[10]

If the sensory system is so involved in all of these disorders, it makes sense that we should be involving the sensory system in the treatment. While sensory treatment programs such as occupational therapy and sensory integration have been used for years for autism, they have not usually been widely employed for the other disorders. Studies have shown that using multiple sensory stimulation works significantly better and faster than using a single sensory input.[11] Kayser et al say:

> When sound and touch were activated simultaneously, the activation of the auditory cortex was strongest. Auditory information in conjunction with tactile input assists with making tactile decisions. Tactile and auditory stimulation simultaneously and individually may positively impact neuroplastic changes in individuals with neurological deficits or impairments. When both tactile and auditory stimuli were conveyed simultaneously, the response was more intense. Differences between sound and touch versus a combination of the two stimuli were significant. Again, the combined stimuli were most significant.[12]

This makes sense because that's how the brain works. It would not be very effective to try to learn to ride a bicycle blindfolded. A person uses their sense of sight, sound, touch and balance to learn and use this skill. The more of our senses we use and the more they function together, the more the learning process is enhanced. Training multi-senses at the same time can not only help to develop each sense but also train them to work together more optimally. A better integration of the senses can help lay down a fundamental foundation from which awareness, perception, reasoning, judgment, and knowledge can develop and grow.

While treating those on the autism spectrum and those diagnosed with ADHD, I saw a deficit in available treatments for the sensory issues they all had. When the research indicated that using input from two senses worked so much better than using only one sense, I wondered what would happen if I used five senses. From that question came Clarity Chair (formerly The SAVE Program). I developed Clarity Chair to improve those sensory deficits. Clarity Chair uses today's technology to target the underlying issue of most brain function problems, the sensory system, an often-overlooked issue that is at the core of many problems.

The more of our senses we use and the more they function together, the more the learning process is enhanced.

Clarity Chair not only combines and targets five important senses at one time, auditory (sound), visual (sight), tactile (touch), vestibular (movement and balance sense) and proprioception (perception of movement and spatial orientation), but it does it using an automated system that results in faster, more effective and consistent training that is passive, requiring no effort on the part of the client. It takes only five days to complete the program. In the new technical age of computers and digital programs, Clarity Chair takes sensory integration to a new level of training, which is firmly based on decades of solid published research and five years of clinical results on Clarity Chair itself.

Clarity Chair uses headphones to administer filtered music to the listener. Participants get visual input from watching a computer screen with colored lights and following those lights as the chair moves. The input for kinesthetic, vestibular and proprioception is through the motion of the chair itself. The chair moves in a figure-eight motion and also moves in a vertical (up and down) motion. This allows for the interaction of the semi-circular canals to have a positive effect on the vestibular system. Proprioception is engaged through the feedback between the nervous system and the sensory ➤

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receptors provided by not only the motion of the chair but also from the auditory and visual input. Kinesthetic input is provided from the body's engagement with the motion of the chair. Combining the five sensory inputs at one time appears to allow Clarity Chair to work faster and better, with positive changes occurring in only five days.

Clarity Chair was developed to help the sensory problems in the autism and ADHD populations and it has done that, with improved speech, decrease in self-stimulation, and improved focus, listening, and behaviors. Many have begun to speak for the first time while others are able to sit still and learn in the classroom. Clarity Chair soon showed promise in helping the symptoms of other disorders. A man with dementia had his memory return. Another with Parkinson's was able to discard his cane, walk six blocks, and climb to the top of a college football stadium without the usual effort it took from having Parkinson's. Years of PTSD symptoms, anxiousness, and sadness resolved in five days in several people when nothing else had helped. A study, to be published soon, found those with long-term traumatic brain disorder who had tried everything available without success, found much improvement in just five days.[13]

Isn't it time we stop using psychiatric medications on these brain disorders and start treating the real underlying cause, the sensory system?

Mary Ann Block, DO, went to medical school at age 39 to save her daughter from iatrogenic injury. In her medical practice, working with those diagnosed with ADHD and autism, she invented Clarity Chair, a medical device that helps improve cognitive symptoms. Dr. Block has appeared on *The Doctors, CBS' 48 Hours, Montel, CNN, Scarborough Country. and The Today Show* and many other television and radio shows. Dr. Block is the author of numerous articles and eight books: *No More ADHD*, *No More Ritalin*, *No More Antibiotics*; *Just Because You're Depressed Doesn't Mean You Have Depression*; *The ABC's of Raising Great Kids*; *Safe and Healthy Weight Loss; Find the Cause, Fix the Problem; Don't Just Cover Symptoms with Drugs*; and *Today I Will Not Die*, the story of her mother's recovery from terminal lung cancer.

Dr. Block was the recipient of the Wayne O. Stockseth Award for Osteopathic Excellence, has served on the boards of the Pan American Allergy Society, American Academy of Environmental Medicine, American Academy of Osteopathy.

Dr. Block is in private practice in the Dallas/Fort Worth area.

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The editors of the *Townsend Letter* recommend that all patients (and physicians) review further reports provided in the article's references and investigate the practitioner's techniques before undertaking an alternative diagnosis, examination, or treatment. Please discuss such treatments and examinations with a reputable health practitioner in your community. If you do use an alternative treatment discussed in the *Townsend Letter*, we would appreciate your report of the outcome, any side effects, and costs.

Using the Endocannabinoid System to Address Alzheimer's Disease

by Kellie Raydon, ND, MSOM

Whether you are a healthcare provider trying to prevent and treat Alzheimer's disease (AD) for your patients or have a family member struggling with this disease, most of us have been faced with people we care for who are suffering with this difficult and unfortunate neurodegenerative condition. While our knowledge of the pathophysiology of AD has become more advanced in recent years, treatment still remains elusive; and finding effective prevention and treatment strategies is becoming increasingly urgent with AD on the rise.

An annual report released by the Alzheimer's Association states that an estimated 5.8 million Americans 65 and over are living with AD in 2020, two-thirds of whom are women. Older African Americans are approximately twice as likely to suffer from the disease, and Hispanics are about one and a half times more likely than white Americans to suffer from it. AD is the fifth leading cause of death for Americans over the age of 65 and the projected annual cost for caring for people with AD in 2020 at $305 billion dollars.[1] With no existing cure in the conventional medical paradigm, (only medications that temporarily improve symptoms), it is easy to feel discouraged and hopeless with such limited and minimally effective treatments.

Perhaps the road to the cure is hidden in plain sight. Perhaps it is embodied within us already this very minute. Perhaps we need to look no further than the endocannabinoid system (ECS) that is already miraculously functioning in each of us before we are even born, maintaining homeostasis and regulating neurologic and immune functioning. As we continue to build our understanding of the neuroprotective and neurodegenerative effects innate to the endocannabinoid system and how it may be harnessed to prevent and treat neurodegenerative conditions such as AD, the fog seems to be lifting, literally and figuratively. As we continue to make headway in our understanding of cannabinoid receptors, their endogenous lipid ligands, and related enzymes, as well as phytocannabinoids, the endocannabinoid system continues to reveal its great potential as a therapeutic target for AD.

Overview of the Current Approach

The conventional treatment for mild to moderate AD is focused on the use of cholinesterase inhibitors. These medications block the breakdown of acetylcholine, which is thought to be important for memory and thinking. As AD progresses, the endogenous production of acetylcholine in our brain progressively decreases and eventually the cholinesterase inhibitors are no longer effective. For moderate to severe AD a medication called memantine is often prescribed, which is a NMDA receptor antagonist that regulates excitatory glutaminergic activity that leads to neuronal damage. These medications work to reduce symptoms for brief periods, sometimes only a few months before they are no longer effective. In addition to their limited efficacy, they are riddled with unpleasant side effects such as nausea, vomiting, headaches, fatigue, dizziness, diarrhea, and weight loss.

One of the tenants of naturopathic medicine is to identify and treat the root cause of any condition. While acetylcholine and glutamate activity certainly influence our neuropsychiatric functioning, the fleeting efficacy of these treatments, coupled by their dreadful side effects are some of the striking indications that these drugs are missing the mark and are not addressing the root cause.

Accessing the Root Cause Through the ECS

The endocannabinoid system is one of the most important discoveries in human physiology. In particular, it holds great promise as we continue to understand its role in neuroprotection and neuro-regeneration, and its potential is only beginning to be realized.

The system consists of endo-cannabinoids, primarily acid ethan-olamide anadamide (AEA) and 2-arachidonoylglycerol (2-AG), and their receptors CB1 (most abundant in the central nervous system) and CB2 (most abundant in peripheral nervous system and immune cells), as well as enzymes for their inactivation, such as fatty acid amide hydrolase (FAAH), and monoacylglycerol (MAGL). FAAH breaks down AEA, and MAGL breaks down 2-AG respectively, and are found throughout the body: in the brain, organs, connective tissues, glands, and immune cells. "With its complex actions in our immune system, nervous system, and virtually all of the body's organs, the endocannabinoids are literally a bridge between body and mind. By understanding this system, we begin to see a mechanism that could connect brain activity and states of physical health and disease."[2]

For decades now research has demonstrated various mechanisms through which the ECS contains regenerative and protective mechanisms. "Exogenous cannabinoids have been shown to exert neuroprotection in a variety of in vitro and in vivo models of

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neuronal injury via different mechanisms, such as prevention of excitotoxicity by CB1-mediated inhibition of glutamatergic transmission, reduction of calcium influx, and subsequent inhibition of deleterious cascades, TNF-alpha formation, and antioxidant activity. It has been suggested that the release of endogenous endocannabinoids during neuronal injury might be a protective response."[3]

Because of its mechanism in achieving homeostasis, the ECS is perhaps the most important innate adaptogenic system. It serves to calibrate or send help where it's needed in the body to maintain balance. Understanding the ECS is transforming the way we treat and prevent a multitude of neurological conditions, which is why it's being embraced across a diverse group of disciplines.

By working with this innate physiological system, our bodies can create balance and healing in a highly sophisticated and multidimensional way. Our bodies produce the endogenous cannabinoids, AEA and 2-AG, as a regulatory response to excitatory or inhibitory stimuli. There are at least 120 phytocannabinoids that also engage the ECS through the CB1 and CB2 receptors and related pathways. Advances in our understanding of the ECS have brought us to the dawning of a new age in medicine. Research propelling us towards preventing and treating AD through the lens of the ECS has stimulated new hope and excitement.

The more that we investigate the ECS, the more we are seeing how any dysfunction in this system could be a possible underlying root cause of AD. The concept of an endocannabinoid deficiency syndrome (ECDS) was outlined by Ethan Russo in 2001 as a possible unifying etiology for a multitude of treatment-resistant syndromes and neurodegenerative conditions. Studies of post-mortem AD brains showing low levels of AEA and 2-AG have corroborated Russo's theory. Also, an increase in CB2 receptors and other evidence of ECS homeostatic mechanism not performing properly has been shown in post-mortem AD brain analysis. The significant increase of CB2 levels found in AD brains mainly corresponds to receptors expressed on microglia surrounding senile plaques.

Non-psychoactive cannabinoids are potentially lead drug candidates for AD and other neurodegenerative diseases.

Neuroinflammation, which begins with microglial activation, is a distinguishing feature in AD that leads to progressive cell damage and neuronal loss. Also, both CB1 and CB2 cannabinoid receptors in the AD brain are nitrosylated, and this could contribute to the impaired coupling of these receptors to downstream effector signaling molecules.[4] Additionally, there are variations in the presence and activity of the enzymes related to endocannabinoid synthesis and degradation in AD brains. For example, endocannabinoid metabolizing enzyme FAAH has been shown to be upregulated in AD patients' peripheral blood mononuclear cells as well as plaque associated glia.[5] Another compelling study demonstrated altered 2-AG signaling during late stages of AD due to compromised MAGL recruitment as well as increased DAGL levels, which lead to synapse silencing in AD.[6] These variations in endocannabinoid levels and receptor sites, as well as discrepancies in the presence and activity of enzymes linked to endocannabinoid synthesis and degradation, are proof that the ECS is a lens that we can look through to gain a greater understanding of preventing and treating AD.

Understanding Pathophysiology and Etiology of AD and ECS Targets

There are several genetic and acquired risk factors that are underlying AD. Miraculously the endocannabinoid system provides underlying mechanisms that offer a potential therapeutic target for each one. Apolipoprotein E (ApoE) is a protein involved in lipid metabolism encoded by ApoE gene, located on chromosome 19. Apolipoprotein E is a carrier for brain lipids and is likely the most important genetic risk factor for Alzheimer's disease. ApoE binds the receptor sortilin, which mediates uptake of ApoE-bound contents into neurons. The significance of this uptake route for brain lipid homeostasis and AD risk is seen specifically with ApoE4.[7] There are three ApoE alleles present in the population at different frequencies. The ApoE4 allele is the main risk factor for late-onset AD. The presence of ApoE4 in heterozygosity increases three-fold the risk of AD developing, whereas in homozygous individuals, the risk is increased 12-fold. Conversely, the presence of ApoE2 allele reduces the risk of developing AD.[8] In the June 2020 issue of *Alzheimers and Dementia*, the journal of the American Alzheimer's Association, Assaro et al. published data highlighting how "Sortilin directs the uptake and conversion of polyunsaturated fatty acids into endocannabinoids, that act through nuclear receptors to sustain neuroprotective gene expression in the brain. Sortilin function requires ApoE3 but is disrupted by binding of ApoE4, compromising neuronal endocannabinoid metabolism and action." This is exciting new insight into the role that ECS provides in epigenetic influence of the development of AD. More specifically, how restoring endocannabinoid function, when compromised, may provide new specific preventative strategies.

Histopathogenesis of AD and ECS Targets

When we look closely at the brain of AD patients, the main pathophysiological mechanisms are deposition of beta amyloid in the brain and tau-related neurodegeneration. Nevertheless the exact mechanism by which the amyloidosis promotes neurofibrillary tangle (NFT) formation of hyperphosphorylated tau protein is not yet fully understood. Blurton-Jones and Laferla (2006)[9] suggest four basic mechanisms: 1. The Aβ peptide promotes the activation of specific kinases (eg, GSK3β) that catalyze the hyperphosphorylation of tau protein, leading to its conformation change and formation of NFT; 2. Neuroinflammation promoted by the deposition of Aβ peptide leads to the production of proinflammatory cytokines that stimulate the phosphorylation of tau protein; 3. Reduced capacity of degradation of tau protein by the proteasome, in a process

induced by Aβ peptide; 4. Defects in axonal transport promoted by Aβ peptide lead to inadequate localization of tau protein and its messenger RNA, which can lead to hyperphosphorylation and aggregation in NFT.

Once again, the endocannabinoid system provides us with therapeutic targets and new insights into each of these proposed mechanisms. It has long been known that the endocannabinoid 2-AG functions to provide neuroprotection from amyloidosis. The neuroprotective effects of 2-AG appear to be mediated via CB1 receptor-dependent suppression of extracellular signal-regulated kinases 1 and 2 (ERK1/2) and nuclear factor-κB (NF-κB) phosphorylation and cyclooxygenase-2 (COX-2) expression.[10] According to research published in *Neuroscience* by Chang et al, it is suggested that "elevation of endogenous 2-AG by inhibiting its hydrolysis has potential as a novel efficacious therapeutic approach for preventing, ameliorating or treating Alzheimer's disease"

The Entourage Effect

Hope really is on the horizon now that we are investigating the neuroprotective and neuroregenerative potential in targeting the ECS to treat AD. "Several in vitro and in vivo studies have demonstrated that cannabinoids can reduce oxidative stress, neuroinflammation, and the formation of amyloid plaques and neurofibrillary tangles, the key hallmarks of late onset AD. In addition, in population-based studies, cannabinoids reduced dementia-related symptoms (e.g., behavioral disturbances)."[11]

More human studies are needed so that we can continue to demonstrate efficacy and safety of phytocannabinoids and other medications targeting the ECS in the treatment of AD. Our scientific paradigm has long been predisposed to a reductionistic model focused on the possibility of finding the one enzyme, substrate, or substance that would be the silver bullet or golden key to target for a cure. However, those of us who use botanical medicine are constantly aware of the limitation of that model and are ever curious about the synergistic effects of compounds contained within one plant or combinations of plants, known as the entourage effect. We are seeing this play out as THC and CBD as isolates have often taken center stage in phytocannabinoid research. When looking at other various combinations of less popular and non-psychoactive cannabinoids, there appears to be a synergistic effect in targeting the ECS with various combinations for the treatment of AD patients, as research by Schubert et al show:

> Eleven cannabinoids were assayed for neuroprotection in assays that recapitulate proteotoxicity, loss of trophic support, oxidative stress, energy loss, and inflammation.

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These compounds were also assayed for their ability to remove intraneuronal amyloid and subjected to a structure-activity relationship analysis. Pairwise combinations were assayed for their ability to synergize to produce neuroprotective effects that were greater than additive. Nine of the 11 cannabinoids have the ability to protect cells in four distinct phenotypic neurodegeneration screening assays, including those using neurons that lack CB1 and CB2 receptors. They are able to remove intraneuronal Aβ, reduce oxidative damage, and protect from the loss of energy or trophic support. Structure-activity relationship (SAR) data show that functional antioxidant groups such as aromatic hydroxyls are necessary but not sufficient for neuroprotection. Therefore, there is a need to focus upon CB1 agonists that have these functionalities if neuroprotection is the goal. Pairwise combinations of THC and CBD lead to a synergistic neuroprotective interaction. Together, these results significantly extend the published data by showing that non-psychoactive cannabinoids are potentially lead drug candidates for AD and other neurodegenerative diseases.[12]

It is exciting to see this current research embracing the complexity of the entourage effect and its benefits that will ultimately serve our patients. While it is more complicated and time consuming to study the effects with all the nuance of various combinations of phytocannabinoids, I believe it will be well worth our while to continue to advance our knowledge of this vast array of combinations and possibilities in targeting the ECS. Mounting evidence continues to confirm that investigation of the endocannibinoidome in humans and other mammals holds a multitude of exciting possibilities for biomedical research, which will continue to be utilized to develop new therapies, medications, and diagnostic biomarkers.[13] For example this is seen in the presence of a complete and fully functional endocannabinoid system that has been revealed in human SH-5YSY cells, which serve as a widely used model for the study of neuronal cell death and damage, in addition to major human neurological disorders. Studies demonstrating the regulation of cell survival and death by endocannabinoids in stem-derived neuronal cells are moving us in a direction in their utilization as therapeutic targets. As a result, endocannabinoid-targeted drugs are being heavily researched at this time for the treatment of neurodegenerative diseases such as AD.[14] A thrust towards more human studies, including the use of human SH-5YSY cells, is bringing us ever closer to effective treatments and preventative measures for AD and other neurodegenerative conditions, where we have currently been at a loss.

The Future of ECS Targeted Therapies for AD

The science, research, and treatments targeting the ECS are unfolding at breakneck speed. While it is tempting in a reductionist scientific paradigm to try to single out endo/exogenous cannabinoids, or single enzyme pathways, I believe we will continue to find that the entourage effect will bring more success and sophisticated treatment approaches. We need to continue to keep up with the ongoing breakthroughs in the research in order to bring our patients the most cutting-edge treatments.

This is no small feat due to the complexity of ECS physiology, including retrograde signaling, "which makes it difficult to fully characterize how its various components may contribute to control genesis and functional integration of adult-born neurons within the post-developmental brain. Future research on the precise role played by distinct components of the ECS in neural stem cell biology will contribute to improving our mechanistic understanding of adult neurogenesis, and could provide the basis for new therapeutic applications of ECS based drugs."[15] These advances in our understanding of how to influence and support the ECS with therapies that may control neurogenesis and integration of neurons in adult brains have created a powerful shift in what may now be possible for patients diagnosed with AD.

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Mental Health, the Brain, and PTSD

by Carolyn McMakin, MA, DC

Abridged: Full article with references are posted online at www.townsendletter.com.

The most important things to keep in mind when considering mental health, the brain and PTSD is that in the brain:

1. Everything is connected to everything else;
2. The system is designed to help you survive trauma and threat in a primitive world; and,
3. Nothing in the brain reacts well to inflammation.[1,2]

Remember item 3. Nothing in the brain reacts well to inflammation. In general, inflammation slows conduction and interferes with neurotransmission. If the stress response and vagal function are not back to baseline in minutes, if the injury and infection are not repaired in eight weeks and the vagus remains suppressed, then the vagus is not suppressing the immune system or controlling the motility, the pH, and the microbiome in the gut; the gut leaks small peptides from ingested food, further activates the immune system, and inflammation increases. When inflammation increases, the nervous system becomes more dysregulated and slower. Corticotropin releasing factor (CRF) causes the cortex to focus on previous traumatic events to the exclusion of recent events that may be neutral or positive. If this goes on long enough, the amygdala, hippocampus, and prefrontal cortex eventually change in activity and even in size.

What does that mean for mental health? Remember that every mental health definition includes some reference to cognitive ability, social integration, emotional balance, stress coping skills and work productivity. Inflammation, CRF and alterations in the prefrontal cortex (PFC) impair cognitive function, executive function, and decision making. Social integration and emotional balance depend on the specific neurotransmitters serotonin, dopamine, and oxytocin all of which depend on branch chain amino acids for their structure. When the gut wall thins and branch chain amino acid transport is inhibited, synthesis of neurotransmitters that depend on branch chain amino acids is delayed or reduced. Without serotonin, dopamine, and oxytocin, mental health is elusive or impossible.

Stress coping skills depend on normal levels of stress hormones and the proper function of the prefrontal cortex and the limbic system (the amygdala and the hippocampus). In long-term stress, all of this delicate balance is disrupted. And the ability to do productive work will depend on the type of work that is considered productive for any specific individual. A concert pianist, an astrophysicist and a carpenter all have different demands on PFC, cortical function, and long- and short-term memory. The inevitable conclusion is that severe chronic stress is incompatible with mental health.

How does this lead to PTSD? A complete detailed description is beyond the scope of this article, and includes genetics, perinatal influences and nutrition as well as neurology and endocrinology. A broad-brush and incomplete answer takes us to the prefrontal cortex, the relationship between the cortex, the limbic system and the vagus, and inflammation. There are so many portions of the stress response that increase inflammation. Inflammation slows conductivity in neurons and impaired conductivity interferes with the delicate feedback system that keeps the brain, limbic system, vagus, and immune system balanced and supportive of mental health.

When your prefrontal cortex has to make the instantaneous discrimination between threat and not threat, cat and tiger, norepinephrine or serotonin, run or cuddle and communicates that to the limbic system on a moment to moment basis and inflammation slows the feedback, what can happen? Survival is the prime directive.

In chronic stress, limbic input to the cortex and PFC increases and biases the prefrontal cortex towards perceiving threat. The PFC in PTSD patients fails to extinguish conditioned fear, fails to inhibit neuroendocrine response to threat-related stimuli, fails to re-assess emotional responses and sees all stimuli as threats to the self. The amygdala in PTSD is more likely to perceive the environment and any stimuli as threatening. The hippocampus is more likely to remember only traumatic events and disregard context. The limbic memories are so strong that they spill over into the visual cortex and cause flashbacks and nightmares. The limbic system becomes sensitized to perceive any stimuli as threatening and turns down the vagus through its connections to the vagal nuclei in the medulla. The vagus stops suppressing the immune

continued on page 57 ➤

FREQUENCY SPECIFIC MICROCURRENT FOCUSING ON BRAIN HEALTH

The effects of frequency specific microcurrent on nervous system function have been documented for almost twenty years.

2000: blood samples analyzed at NIH showed log-rate reductions in all of the inflammatory cytokines by factors of 10 - 20 times in response to only one frequency combination. Only frequencies targeting the medulla and nervous system increased serotonin levels.

2003: Only one frequency combination (40/116) reduced lipoxygenase (LOX) mediated inflammation by 62% in four minutes in blinded animal research. COX mediated inflammation declined by 30% in four minutes. No other frequencies reduced inflammation.

2010: PTSD protocol reduced symptoms and scores after only four treatments in four weeks in 3-5-year chronic combat-induced PTSD. No improvement is expected when PTSD is more than two years chronic.

2013: significant and dramatic EEG changes were documented in TBI and autism patients treated with a combination of FSM and speech therapy.

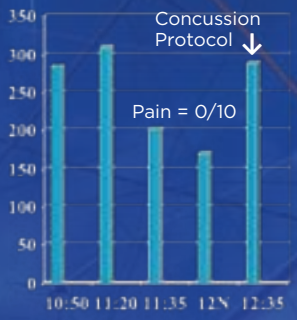
2013: only specific frequencies targeting the sympathetic and parasympathetic nervous systems changed autonomic function and heart rate variability dramatically and quickly.

The data suggests that combining FSM rapid effects with nutrition makes treating the brain more effective, more efficient and less expensive.

ONLY ONE FREQUENCY SEQUENCE INCREASED SEROTONIN

#1 Serotonin = 285.6
#2 Serotonin = 309.2
#3 Serotonin = 202.1
#4 Serotonin = 169.5
#5 Serotonin = 289.6

Serotonin
normal=100-300 ng/ml



- Serotonin dropped during pain treatment (40/10) as endorphins rose.
- Pain was 0/10 at 12N
- Only one protocol increased serotonin levels by as much as double in 35 minutes in every patient
- Serotonin was the only parameter that changed direction with that protocol

TREATING PTSD WITH FREQUENCIES ALONE

Case #1 Combat exposure score 28 (high)

4 years chronic (2006)
5 Treatments

	6/24/10	7/24/10
BDI	43	13
GAD 7	21/21	2/21
PTSD-M	77/85	60/85
PTSD-C	79/85	58/85

Case #2 Combat exposure score 35 (Heavy Exposure)

3 years chronic (2007)
4 Treatments

	3/17/10	3/30/10
BDI	47	32
GAD 7	19/21	2/21
PTSD-M	78/85	60/85
PTSD-C	79/85	61/85

Case #3 Combat exposure score 27 (high)

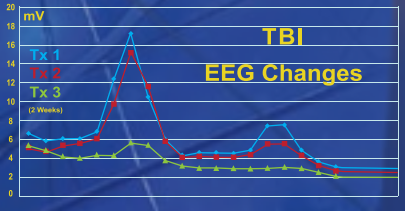
DOI 2005, Dx 2007
4 Treatments

	4/2/10	4/27/10
BDI	29	not done
GAD 7	8/21	4/21
PTSD-M	43/85	22/85
PTSD-C	44/85	22/85

THE PERFECT COMBINATION

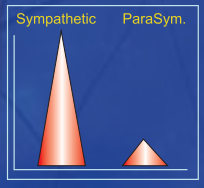
- **Specific frequencies change cell signaling to reduce inflammation**
- **Frequencies change neurotransmitters quickly and safely**
- **Specific frequencies change the brain**
- **Support those changes with nutrition and lifestyle**

Frequencies Change The Brain

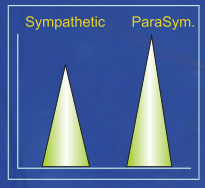


Change Autonomic Balance

Pre-Treatment

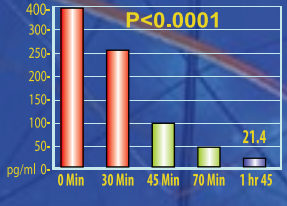


Post-Treatment

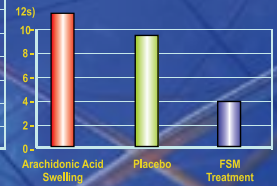


Reduce Inflammation

Reduce IL-1



Reduce LOX 62%



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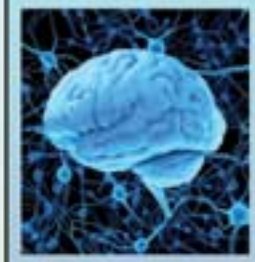
"An infrared sauna at 140 degrees for 30 minutes, that REALLY TAKES CARE of viruses in the body, including the Corona virus!" ~ Dr. Gary Null

Relax Far Infrared Sauna

"No Sweat? Know Sweat!" Author Recommends Relax Sauna

The Relax Sauna is Highly recommended by thousands of Medical professionals, doctors and therapists. Dr. Bill Akpinar, Best Cancer Doctor NYC, says. "The Relax Sauna creates more Chi than any other Sauna."



FAR INFRARED SAUNAS ARE GREAT FOR BRAIN HEALTH

In one shocking study on saunas, people that used a sauna 4-7 times per week had a 66% REDUCED RISK of dementia and Alzheimer's versus those who used the sauna only once a week! If only there were a pill that could do this! Additionally, heat stress, the good kind of stress, like working out, activates one of the genes associated with longevity called FOXO3, which makes cells resilient to age-related stresses. Evidence suggests that heat therapy, intensified by far infrared energy, can increase longevity & endurance, extend life and quality of life, create healthier genes and positive, lasting changes in the body. Surprisingly, those with depression who used a sauna ONLY ONCE reported great and lasting effects in their mental health! **Sweat is magic!**

Far infrared rays harmonize our nervous system into a more calm and relaxed state. This beneficial healing light also lowers cortisol, the stress hormone and puts us into peaceful and euphoric moods. So, the more often you activate your nervous system with far infrared light, this practice of enjoyable persistence trains the body to deal with stress on a more relaxed, easy going and less reactive way. Better for sleep, better for all relationships!

The autonomic nervous system has two legs that are supposed to balance each other out: the sympathetic and the parasympathetic. Many factors can disrupt the balance. Stress forces our reactionary fight-or-flight responses to extremes. This fight-or-flight mode puts the sympathetic (stress) nervous system in control and creates inflammatory responses in the body, which then lowers immune function. Far infrared light rays are very serene to the body, which helps us sleep better and deeper, and aids with insomnia and anxiety. Sleep is divine!

Far infrared and sweating helps our nervous system switch into a parasympathetic state that is more calming and relaxing which creates more tranquility. The parasympathetic (rest and digest) nervous system will send the blood flow back to the brain and you'll be able to think more clearly. It can also help reactivate your digestive system. A less chaotic digestion process sends signals to the brain that all is ok to rest peacefully. This gut brain connection is a crucial fundamental of overall immune health as all systems are connected and each must be content and in sync for symbiotic optimal conditions of the whole.

The best things in life make you sweaty!

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THE SUPERIOR SAUNA

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- Uses less electricity and takes less time with great results.
- Fast acting! Relief of symptoms happen quickly!
- You can breathe! Head outside the tent makes a huge difference in comfort and endurance.
- Effective even with clothes on for 5-10 minute treatments!
- Amazing, deep river sweats in 15-20 minutes or even less! (not 30-60 minutes or not at all!)
- It is safe, simple, strong, pure, portable, medical grade and very affordable!

DON'T THROW IN THE TOWEL, USE IT TO WIPE THE SWEAT!

The Relax Sauna is the highest quality & most effective portable sauna in the world! It is well-known & highly regarded for creating instant calmness, deep relaxation & pleasant, unbelievable dripping sweats, penetrating quickly & deeply into the tissues, skin & cells, pushing out toxins safely through the sweat. It is also a powerful lymphatic system activator and gets that vital system flowing! This is why Hippocrates has stated, ***"Find me a way to create sweat, and I WILL cure disease."*** ***SWEAT IS MAGIC!***

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➤ *continued from page 54*

system, inflammation increases and the split-second communication that would give the PFC time to discriminate cat from tiger is disrupted. Stress hormones go up and the brain becomes more sensitive to them.

Mental health as demonstrated by cognitive ability, social integration, emotional balance, and work productivity becomes secondary. Stress coping skills are overwhelmed. Survival is the prime directive.

PTSD is being treated as a mental health disorder instead of a neuro-endocrine-immune problem. It is a huge problem worldwide and affects 8% of the general population, worse in war zones and much worse for women than for men. It is more prevalent than cancer or schizophrenia. The statistic is staggering. There are pharmaceutical therapies aimed at raising serotonin by slowing the degradation of serotonin at the synapse and medications for anxiety. Cognitive behavioral therapy is a type of talk therapy aimed at teaching the cortex that it's really a cat not a tiger and trusting the cortex to prevail in communicating this to the PFC and limbic system. Eye movement and head tapping therapies attempt to recruit more primitive parts of the brain and distract them to reprogram the stress loop. These therapies seem to be able to interrupt the neuro-endocrine-immune cycle in enough parts of the loop to produce limited success. But the prime directive fights change, and PTSD that is two-years chronic is considered permanent.

But what if there was a single therapy that could reduce inflammation and quiet overactivity in specific parts of the brain, quiet inflammation in general, repair the intestinal wall to stop thinning and leaking and allow proper absorption of bulky branch chain amino acids to restore healthy neurotransmitter levels. If this therapy could also directly quiet the limbic system and increase secretions and function of the vagus, it would be ideal to restore mental health and repair PTSD. It appears as if frequency specific microcurrent (FSM) might have been doing exactly that since 2005.

FSM uses frequencies from a list found in an osteopathic office in 1946 that came with a machine manufactured in 1922. There is no evidence about how the frequencies were developed or verified that survived the medical purges of the 1930s.

In 1995, the list was discovered, and the frequencies were first applied with a two-channel microcurrent device approved for regulatory convenience by the FDA as a TENS device even though it delivers 1000 times less current than a TENS unit. The list has frequencies for tissues and for the conditions or pathologies that make those tissues dysfunctional. At first the frequencies were used only to treat muscle and nerve pain, but the list included frequencies for the immune system, the spinal cord, and specific parts of the brain. Clinical success was followed by limited neurochemical research.

Microcurrent electrical signaling can rapidly change the electro-neurochemical functions of the brain to change neurotransmitter levels.

In 2000, it was discovered that the frequencies from the list "to reduce inflammation" (40 hertz) in the "spinal cord" (10 hertz) could not only reduce pain in fibromyalgia associated with spine trauma but also reduce all of the inflammatory cytokines and substance P and increase endorphins (See Table 1). The neuroimmune data was produced by an NIH immunochemist and show rapid logarithmic changes in neuro-inflammation never seen with any medical therapy. The same data showed that serotonin decreased while endorphins skyrocketed but immediately reversed course and rapidly increased when a frequency protocol for the medulla, called "the concussion protocol" was applied. This finding suggests that microcurrent electrical signaling can rapidly change ➤

Table 1

Sample	DATE	IL-1	IL-6	IL-8	TNF-α	IFNγ	SP	CGRP	VIP	NY	β Endorph	Cortisol	Serotonin
MK1	5/11/00	392.8	204.3	59.9	299.1	97.2	132.6	100.8	8.5	18.1	5.2	15.5	285.6
MK2	5/11/00	288.5	200.8	47.6	265.7	99.8	127.5	97.6	10.2	13.7	7.1	12.6	309.2
MK3	5/11/00	103.2	121.7	21.3	96.5	73.7	82.4	61.3	32.9	7.2	21.4	33.7	202.1
MK4	5/11/00	52.6	33.9	11.4	43.4	32.6	38.2	22.4	48.4	5.1	69.1	78.3	169.5
MK5	5/11/00	21.4	15.6	4.8	20.6	11.4	10.5	8.6	69.9	6.6	88.3	169.9	289.6

All values are in pg/ml

IL-1, IL-6, IL-8, TNF-a, IFNg, CGRP are immuno peptides that mediate inflammation in the nervous system. Any intervention that reduces them is considered beneficial.

SP is Substance P, which is produced in the spinal cord and mediates pain signal transmission.

VIP is vasoactive intestinal peptide and has widespread complicated actions that causes vasodilation, modifies digestion and affects sleep. In general, VIP reduces inflammation.

NY is neuropeptide Y and follows the sympathetic stress response. When it goes down, it indicates that sympathetic tone and the stress response are being reduced.

b-Endorphin is an opiate-like neuropeptide produced in central and peripheral nervous system neurons. When endorphins go up, pain and stress go down.

Cortisol is a steroid hormone the body produces to reduce inflammation. In this case it is not increased due to stress or injury since neuropeptide Y decreases showing that the stress response is reduced. Cortisol apparently increases as a side effect of the increase in b-endorphins. To produce b-endorphins, the brain produces pro-opiomelanocortin, which splits into ACTH and b-lipotropin, which then splits into g-Lipotropin and b-Endorphin.

Serotonin rises at first, then drops while endorphins rise until measurement MK 4 when the treatment was switched to "the concussion protocol" that focuses on the frequencies for the medulla. The concussion protocol produces a rapid rise in serotonin between MK4 and MK5 a period of 30 minutes.

➤

the electro-neurochemical functions of the brain to change neurotransmitter levels based on specific frequencies.

Blinded animal research followed in 2003 showing that the frequencies to "reduce inflammation in the immune system" reduced lipoxygenase (LOX) mediated inflammation by 62% in four minutes and cyclooxygenase (COX) mediated inflammation by 30% in four minutes which was equivalent to injectable Toradol when it was tested in the same animal model by the same researcher. Clinical research followed demonstrating effectiveness in thalamic pain syndrome and phantom limb pain, both of which involve the thalamus and central sensitization.

In 2005, Barbara Harris, MD, combined her post-doctoral education in neuropsychology and her knowledge of FSM and began treating PTSD with FSM. When there were consistent successes and no adverse reactions, her protocol was taught at FSM advanced courses; and positive case reports were followed for the next fifteen years. Starting in 2015, the frequencies for the vagus were applied in atrial fibrillation, gastroparesis, vocal cord dysfunction, and small intestine bacterial overgrowth (SIBO) and appeared successful at restoring normal vagal stimulation and function.

PTSD is as hard to document as mental health and so FSM clinicians have limited data and no published case reports. Funding for non-pharmacologic treatment is hard to acquire in general and virtually impossible for unaffiliated clinicians, but the case report data is worth considering.

FSM in separate studies and case reports appears to address over activity and inflammation in the specific parts of the brain, raise serotonin levels, quiet central sensitization, quiet inflammation generally and specifically increases

Case Report

This case report is a compilation of similar cases to preserve patient confidentiality.

She was a 43-year-old woman with a precarious professional career in real estate sales. She could have been very successful, she said, but her stress levels were so high that she had trouble focusing on work and finishing the calls and the projects she needed to finish in order to close more deals.

"I sell condos in the most desirable area of Portland. This should be easy! My digestion is so weird. I bloat up and my stomach hurts after eating certain foods and lately it seems as if there are more of them that I react to. I don't sleep well. Maybe 4-5 hours a night I wake up more tired than when I went to bed. My body gets stiff and sore with any exercise and I used to love exercise. I'm achy most of the time – not pain exactly just achy. My boyfriend is helping to pay the bills, but he is so controlling and crabby, I find myself doing things, or not doing things, to keep from making him mad. He gets mad when I spend time doing work instead of sitting with him."

A hint to start: The answer is always in the history. Start with what the patient complains about but remember that this is hardly ever the real problem or the only problem.

- How long have you been with him?

"We've been together for 9 years, about one year after my divorce. I was a successful real estate agent in my twenties. And then we moved in together into this condo and I started getting tired all the time, stopped sleeping well, and my income fell off; and he started getting more controlling and seemed to enjoy the fact that he now made more money than I did. And he just loves to start arguments. It makes me so jumpy but now I need him to help financially."

- Does he ever hit you?

"No, just yells all the time about the most random things. I never know what to expect."

- What attracted you in the beginning? Why do you stay with him?

"He was cute and funny; we laughed a lot. He made good money, dressed well and drove a nice car. We were in the same business. He does property management.

Once we moved in together things changed as soon as I started to get tired and stopped sleeping. He got more controlling, and we fought more often. It's been really bad for the last three years. I can't focus well enough to do the kinds of complex deals I used to do so easily."

- Would you mind if I asked you what your childhood was like? Did your parents fight all the time?

"Yeah, towards the end when I was a teenager, they fought a lot."

- Did he ever hit your mom or you?

"No, but once they got divorced when I was 10, her next boyfriend yelled a lot at both of us. He hit her once I think but mostly, we had to be careful around him."

- Did you ever have any accidents, serious illnesses, or surgeries?

"When I was little, I had some problem with my intestines – maybe I had to have surgery when I was 4 or 5 – see I have this little scar on my abdomen. But I got better fast. I got rear ended when I was 25 and I rear-ended someone when I was 19. No serious injuries though just sore for a few days, I get headaches now and then, but I see a chiropractor and get a massage and they go away."

- Have you ever been molested, assaulted, or raped?

She squirmed a little at this matter-of-fact question, but since it was just in the same line as the other trauma questions, she avoided eye contact and answered it matter-of-factly: "The boyfriend used to come in my room at night when I was 11 or 12 and sit on my bed and talk to me like he was just

vagal tone. Reducing inflammation specifically in the small bowel could reasonably help repair a thin leaky gut. All of these effects would explain the success of the FSM protocol for treating PTSD.

It may not be possible to change the stress and threat in the world, but it appears possible to improve neurotransmitter levels, reduce inflammation, and improve mental health by restoring the healthy connection of everything to everything else in the brain. It's worth a try. ◆

Carolyn McMakin, MA, DC, developed Frequency Specific Microcurrent™ (FSM) in 1995 and began teaching it in 1997. She has a part-time practice treating chronic pain, does clinical research, and teaches FSM seminars in the US and abroad. She has lectured at the National Institutes of Health and at conferences on fibromyalgia and chronic pain in the US, Australia, England, Kuwait, Taiwan, and Germany. Her textbook *Frequency Specific Microcurrent in Pain Management* was published in 2010 (Elsevier). *The Resonance Effect; How Frequency Specific Microcurrent is changing medicine* was published in 2017 by North Atlantic Books. www.frequencyspecific.com

Abridged: Full article with references are posted online at www.townsendletter.com.

being friendly, but then there was touching and some other stuff that was not right. But when I told my mom she said he was just being nice and we couldn't afford to live without the money he brought in and I should figure out how to deal with it. So, I did. I started pretending I was already asleep and then I got a lock on my door."

The physical exam was pretty basic and close to normal. Sensation, reflexes are all normal (+2 and brisk bilaterally). Abdominal palpation showed no signs of acute abdomen, but her belly was tender over the small bowel and cecum.

The only test ordered was salivary hormone testing for estrogen, progesterone, and salivary cortisol levels at four times during the day.

I don't do relationship counseling, but it was clear that she had wandered into a relationship with a borderline personality, but the stress that causes wasn't the only problem. Many women would have had no trouble leaving him. Why did she stay? Why was she tired? Why didn't she sleep?

The diagnosis and treatment were determined by the history. Keys:

1. The vagus nerve is turned off by infection, stress and trauma.
2. Perimenopause is characterized by reductions in progesterone first. Estrogen falls 5 to 10 years later with menopause. Estrogen dominance results in fatigue, depression, and anxiety.
3. Diurnal adrenal secretion of cortisol keeps inflammation down, gets you up in the morning, and goes down at night so you can sleep

Treatment with frequency specific microcurrent (FSM) involved quieting the limbic system, increasing vagal tone by increasing secretions in the vagus. The frequency to reduce the activity of the thalamus reduced her pain better than anything else although the frequencies for allergy reaction (to reduce histamine) also helped. Histamine stimulates class C pain fibers, which are slow unmyelinated fibers that create diffuse aching. We used FSM to treat the small intestine to reduce IBS symptoms and quiet the macrophage mediated IgG reactions. Macrophages consume the IgG complexes and then basically explode releasing histamine. To stop the histamine, you have to heal the small intestine and repair the leaky gut that creates the IgG complexes.

The FSM protocol for "brain fog" seemed to help with concentration and mood. She purchased a FSM home unit so she could run the PTSD and sleep protocols at home.

Salivary hormone testing was done to determine whether she needed hormone replacement or adrenal quieting or adrenal support. Genetic testing was done to determine if she had a methylation problem for B-12 or folate and to determine if she could phosphorylate B6. She had problems with folate methylation and supplementation with 5 mg of methyl folate a day helped with that. I usually give patients 5-HTP and P-5-P and that seemed to help with her mood and digestion.

Urine mold testing was done to determine if she had a mold infection from mold exposure in the condo she moved into with her boyfriend. That test was negative, so we didn't have to deal with mold recovery.

Her homework involved seeing her ND for stool testing and treatment for parasites if necessary. There were no parasites but there was a need for butyrate for a month and probiotics.

Our clinic appointments were twice a week for four weeks. FSM produces rapid noticeable results which gave her hope after so many years of illness and anxiety. The hope was in itself helpful. Her symptoms changed within two weeks. As she recovered energy and was able to work more effectively, her income increased, and she moved out of the condo and separated from the difficult boyfriend. She had one follow up appointment at eight weeks and considered herself fully recovered. I don't have any experience with treating this kind of case without the use of FSM.

Estrogen Vindication, Part 2: Estrogen, Cancer Stem Cells, and Studies

by Devaki Lindsey Berkson, DC

Abridged: Full article with appendices and references are posted online at www.townsendletter.com.

Summary

As Part 1 of this article (*Townsend Letter,* August/September 2020) explained, estrogen was once an extremely popular hormone. Estrogen replacement was used as an anti-aging drug by multiple millions of women in the United States and all over the world. Estrogen therapy was even standard of care to treat certain breast cancers. However, once the Women's Health Initiative (WHI) hormone trials were prematurely stopped due to early negative conclusions (July 9, 2002), claiming prescribed estrogen caused cancer and heart disease, these scary headlines prompted many doctors and women to become *estrogen-phobic*.

Nonetheless, as crazy as it seems, within only months after the WHI first published negative estrogen news, re-analyses started to emerge from scientists and statisticians at prestigious institutions. These new "look-sees" of the WHI statistical data painted completely different stories.

But the emerging "good news" never made headlines like the initial "bad news." The bad news? Estrogen can "cause" breast cancer. This turned out to be "wrong news." The good news: Estrogen therapy in healthy women significantly "protects against" breast cancer.

This good news continued to accumulate over the past two decades, without influencing "standard of care," without entering the clinical trenches of most doctors or the understanding of most women. Until December 2019.

In December 2019, a 19-year re-analysis – looking back from an almost 20-year vantage point with new statistical in-depth collaborative investigation – was presented at the San Antonio yearly breast cancer symposium. Conclusions by a consortium of 12 highly respected cancer centers stated – once and for all – estrogen protects breasts. This re-analysis makes it finally undeniable that estrogen is not the enemy; rather, it *protects* breasts from cancer. Unfortunately, many doctors and women continue to not know about this news nor translate it into their practices or lives.

Even though no one knows exactly how breast cancer starts, it does not seem to be due to estrogen, but rather to *cancer stem cells* – a totally different kind of cell that has nothing to do with estrogen. Most older women, who naturally have less estrogen, have higher risks of being diagnosed with breast cancer than younger more estrogenized females. Pregnancy, which is the "highest estrogenic time" of life in any woman, is protective *against* breast cancer. In fact, there is a 70% decrease in breast cancer risk associated with a full-term pregnancy before the age of 18. It's also been shown that pregnancy is safe after treatment of breast cancer, even among estrogen receptor–positive women patients (ER+ means pathologists identify *estrogen receptors* in the tumor). Also, no benefits have been proven for abortion at the time of pregnancy in breast cancer patients so lowering the levels of estrogen didn't cause further improvement in outcomes.

Medical practitioners thought tamoxifen worked because it was an anti-estrogen. But tamoxifen works in a wide variety of anti-cancer mechanisms, not just by tamping down estrogen. In fact, tamoxifen can often raise estrogen levels. In the HABITS study (which concluded that hormones cause breast cancer), only the women on tamoxifen turned out to have higher risk of recurrences (though this was not easy to read within the inner depths of the study and was thus not noticed by many doctors). (Tamoxifen will be discussed in Part III of this article.)

The appreciation of estrogen as "foundational" in protecting many aspects of health is rapidly growing. A few examples are estrogen protects bones from fracture, blood vessels from hardening, brain from dementia, shields mitochondria (energy-producing cells) from damage, allows bodies to benefit from lifestyle changes as it promotes epigenetics, makes it easier to keep a smaller waistline, and maintains heart and kidney health.

Estrogen therapy is as close to an effective anti-aging tool that we have, even maintaining life-promoting telomere length. The longer and healthier our telomeres (the tips on our

DNA), the longer and healthier we live. In fact, estrogen reduces premature death from quite a large number of possible causes.

The Physiology of Estrogen

When you understand how estrogen works in the body, you can appreciate its role in women's health.

Older age, less estrogen, more breast cancer risk: As women get older and are in a less estrogenic state, they are more at risk of getting breast cancer even if they never took estrogen therapies. If estrogen were carcinogenic and the main cause of breast cancer, we would expect breast cancer rates to decline with menopause, but the opposite occurs.

Pregnancy is protective. Pregnancy is the highest estrogen exposure in any woman's life, with estrogen levels up to ten times more than at any other time. It is true that immediately and up to a year after pregnancy, a woman's breast cancer risk slightly rises, only to lower significantly across her lifetime. But it turns out that pregnancy – with all its huge hormonal exposure – protects *against* breast cancer in the long haul. In fact, women who are diagnosed with breast cancer during pregnancy have a similar prognosis as non-pregnant women at the same stage of breast cancer. Nuns, who never get pregnant, have a higher rate of breast cancer compared to those who aren't nuns.

Not ever having a baby increases a woman's risk of getting breast cancer by 30%. The younger you are at your first pregnancy, the more lifelong protection you'll have against breast cancer. Women who give birth before the age of 20 have the highest protection. When I worked as a hormone scholar at an estrogen think tank at Tulane University (Center for Bioenvironmental Research), it was often stated at conferences that there was no documented case of a woman getting breast cancer if she got pregnant before the age of 18. Yet the older you are when you give birth, the opposite is true. Women who have their first child after the age of 35 and have missed out on the surge of protective high levels of progesterone and estrogen (estriol) during pregnancy, have a 40% increased risk of breast cancer compared to women who have kids before the age of 20.

Pregnancy and BRCA genes. At the annual meeting of the American Society of Clinical Oncology in June 2019, an international team of investigators reported a retrospective, case-control study of 1,252 women who had been diagnosed with breast cancer, all of whom had BRCA gene mutations. Of this group, 16% (195) eventually became pregnant and were followed over the next decade. The women who became pregnant had a longer disease-free survival than women who did not become pregnant, although both groups were matched for age, tumor size, nodal status, hormone receptor status, type of surgery, and type of endocrine therapy. The two groups did not differ in overall survival either. Interestingly, but not surprisingly, hormone receptor status of the tumor did not affect disease-free survival or overall survival among the pregnant patients. This study provides further evidence that pregnancy, which elevates levels of estrogen tenfold, does not fuel its recurrence.

Estrogen reduces premature death from quite a large number of possible causes.

Estrogen as an anti-cancer agent. Estrogen was used for years to treat metastatic breast cancer. If estrogens were carcinogenic, this would not have worked. The use of high-dose estrogen, which began in the 1940s, was the first successful breast cancer therapy. Using oral estrogens to treat breast cancer continued all the way into the late 1970s, until tamoxifen (an anti-estrogen) was introduced. When tamoxifen became the standard of care in 1974, estrogen therapy pretty much stopped.

Breast cancer while on HRT. Women diagnosed with breast cancer while on hormonal or estrogen therapies have consistently been found to have better prognoses than women diagnosed without being on hormonal therapies.

Estrogen is not initiating breast cancer. Many doctors and women think estrogen receptor positive (ER+) breast cancer cells (having estrogen receptors on the tumors) means estrogen is "feeding' or "driving" the cancer. But a close look at the science shows this is most often not the case, although this is not widely understood even by many cancer doctors. Estrogen receptors are found on *all* normal breast cells. Estrogen receptors on tumor cells signifies that the tumor is growing so slowly that the breast cell still has some normal cellular characteristics. Estrogen receptors on tumor cells does not mean that estrogen is promoting tumor cell growth. Scientific biological studies are revealing cells that initiate tumor growth and recurrence, called cancer "stem" cells, or "cancer initiating cells," which do not have estrogen receptors nor proliferate (grow) in response to estrogen.

Estrogens Don't Fuel Breast Cancer, "Stem" Cells Do.

There appears to be a consensus that estrogen is a major cause of breast cancer. But when you take a scientific analysis of existing data, including findings from the Women's Health Initiative, you see that epidemiological strength and true scientific support are not met in the case of estrogen-causing breast cancer, raising serious questions about the validity of this widespread assumption.

The reality is that the exact mechanisms underlying how breast cancer starts are still not known, but evidence points to *cancer stem cells* rather than estrogen receptors on breast cells as being the responsible agent.

The human breast is made up of a number of cells. Basic breast cells are called epithelial cells, which are often guarded by myoepithelial cells. Breast cells live, function, and die, meaning they have a finite life span. Many of these healthy cells express estrogen receptors. The breast also contains a lot of fat cells that potentially contribute to milk production when and if the woman is breastfeeding, as well as giving shape and form to the human breast. ➤

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It had long been thought that most breast cancers arise from ductal cells, made of either epithelial or myoepithelial cells, and that this action was fueled by estrogen in *estrogen positive breast cancer*. Breast cancer tumor cells labeled as "estrogen positive" mean they have receptors that can receive signals from the hormone estrogen. The thought has been that estrogen signals fuel these cells to turn cancerous and to grow and become life threatening.

However, the cells that are estrogen positive are turning out to not necessarily be the root cause of cancers. In a report from the National Cancer Institute's Division of Cancer Etiology, published in 1991, analysis of existing data concluded that estrogens are neither direct *mitogens* nor direct *carcinogens* for mammary cells. These cells are distinct from cancer stem cells, which instigate cancer and are responsible for the recurrence of cancer.

Cancer stem cells make up about 5% of breast cells and are *not* fueled by estrogen. Cancer stem cells possess characteristics of both stem cells (which give rise to healthy breast cells) and cancer cells, in that they have the properties of self-renewal, asymmetric cell division, resistance to death (apoptosis – cancer cell immortality is a huge part of the nastiness of cancer), independent growth, tumorigenicity, and metastatic potential. These cancer stem cells are now thought to initiate cancer as well as drive recurrences of cancer.

Stem cancer cells are so regarded as cancer causative; they are also referred to as *tumor-initiating cells*. Tumor recurrence is the "leading" cause of breast cancer-related death. These recurrences arise from the residual cancer stem cells that survived initial therapeutic intervention. So breast cancer stem cells are at the "root cause" of recurrence. When I consult my breast cancer patients to help make their "remission" their "mission," they are taught many tools to tamp down cancer stem cells such as consuming foods high in anthocyanin pigments, which help eradicate cancer stem cells.

Triple-negative breast cancers are more aggressive due to lacking receptors (because they are furthest away from normalcy of typical breast cancer cells), and they maintain more breast cancer stem cell activity.

Cancer stem cells have been identified in the blood, brain, bone marrow, and the breast. They can literally hide from treatment. Europe has known this for many years; in some European cancer centers they test bone marrow for cancer stem cells when someone has a diagnosis of cancer. Just taking out a tumor in a breast doesn't mean there aren't cancer stem cells lurking elsewhere in the body.

Cancer stem cells can be enabled and stimulated by various elements such as pro-inflammatory molecules, dysfunctional immune cells, chronic inflammation, or various protein structures (made up of anillin) that in essence builds bridges to allow cancer stem cells to travel far and wide in the body, causing havoc wherever they go.

Avrum Bluming, MD

Avrum Bluming, MD, is the visionary cancer doctor who singlehandedly changed the way surgical standard-of-care was performed for breast cancer patients. Before Dr. Bluming a diagnosis of breast cancer meant disfiguring surgeries. Dr. Bluming helped stop resistance toward more conservative interventions. Now Dr. Bluming is trying to change the standard-of-care for estrogen therapies for both healthy and breast cancer patients, as laid out in his book *Estrogen Matters*.

Dr. Bluming has been in practice in Southern California for almost 50 years. Early in his practice, he started to sleuth out data proving that more conservative forms of surgery, such as lumpectomy and radiation without mastectomy, or mastectomy with less tissue removal and chemo, were as protective and successful as older severe surgical procedures, while maintaining efficacy and safety. He gathered together surgeons and cancer doctors and put on symposiums to demonstrate this data.

Dr. Bluming continues to be a visionary. He is the only oncologist who has created and continuously published a 14-year ongoing study tracking breast cancer patients given estrogen therapy (Premarin) over 14 years. He first did a pilot study. Then, after much elbow grease to get permission to do a study on breast cancer patients, he gave Premarin to 248 women with breast cancer, beginning in 1992. Dr. Bluming had 100% follow-up. Every year he published a study update. In 1997 he presented the five-year follow-up. "No" women prescribed estrogen had any increased incidence of recurrences compared to similar (matched) breast cancer patients not on estrogen therapies.

Dr. Bluming put together a review that highlights the history of research on HRT, including a timeline of studies that have or have not found a link between HRT and breast cancer.

Drs. Bluming and Tavris, his co-author on *Estrogen Matters*, write: "Breast cancer generates more anxiety than even heart disease, even though the number of US women who died of heart disease in 2010 is over seven-and-a-half times the number who fell victim to breast cancer."

A review of the statistics show that almost 90% of women with breast cancer at any stage will still be alive at five years after diagnosis. By 14 years, when Dr. Bluming's breast cancer estrogen therapy study ended, all of the breast cancer patients on estrogen therapy still did not have an increased incidence of recurrence of breast cancer compared to matched breast cancer patients not on HRT. They also reported higher quality of life. This is huge.

Studies Saying Estrogen Is Bad for Breasts

After a half-century, hormone therapy's influence on breast cancer still remains controversial, even though human studies, like Dr. Bluming's, showed estrogen protects from recurrence of breast cancer and death. Part of the controversy was due to a few studies that said estrogen did in fact cause breast cancer. Besides the WHI, these were the HABITS study,

The Million Women Study, and a more recent study published in the *Lancet* in 2019.

The Women's Health Initiative (WHI): From 2002 to 2008, reports from the WHI claimed that hormone replacement therapy (HRT) significantly increased the risks of breast cancer development, cardiac events, Alzheimer's disease, and stroke. These claims alarmed the public and health professionals alike, causing an almost immediate sharp decline in the numbers of women receiving HRT. However, the actual data in the published WHI articles revealed that the findings reported in press releases and interviews of the principal investigators were often distorted, oversimplified, or wrong.

Re-analyses that were begun within several years had opposite findings, and a 19-year follow-up WHI analysis found that estrogen protects against breast cancer while on it and even 10 years after going off it, while progestins do the opposite. Progestins, on the other hand, increase the risk of breast cancer while on them, and for up to a decade after going off them.

The group of women in the WHI study who did experience a higher risk of breast cancer were on the combination therapy: estrogen plus a synthetic progestin. The finger must be pointed at the synthetic progestins rather than at the equine estrogens. Remember that birth control pills are also made up of synthetic progestins, and they also have been linked in some studies to increased risk of breast cancer.

Depending on the study, *bioidentical progesterone therapy* has been found to have no stimulating effect on breast cancer or no effect at all. Large human studies have shown that natural progesterone does not increase the risk of breast cancer like the synthetic forms do. In some studies there is a protective effect. Progesterone has many beneficial actions on the body, nervous system, brain, and even breast tissue.

Ultimately, the WHI showed that estrogens do protect the breast against breast cancer.

HABITS (an acronym for Hormone Replacement Therapy After Breast Cancer – Is It Safe?). This is a widely referred-to study saying that estrogens are dangerous for breasts. Yet a number of experts felt that the conclusions from this study were not warranted. Dr. Rowan Chlebowski, the lead investigator on the 19-year re-analysis of the WHI, said this isn't the last word on hormones, breasts, and women.

The HABITS study, run in Sweden, proposed to randomize 1.300 breast cancer survivors on HRT or not and follow them for five years. The study, like the WHI, was prematurely stopped in 2003 as more women on HRT developed a recurrence of breast cancer. But these groups, in closer analysis, did not differ in risk of metastatic disease or risk of death. Furthermore, a follow-up analysis in 2008 revealed that recurrence of breast cancer in women on HRT only occurred in those taking tamoxifen – an estrogen blocker! Think about that, as this "screams" out loud something different than the warnings published about estrogen.

In 2019 a meta-analysis of 58 observational studies was published in the *Lancet*, in which estrogen plus progestin and also estrogen alone were both associated with a significantly increased risk of breast cancer. Also, in the Million Women Study, both estrogen plus progestin as well as estrogen alone were associated with a significantly increased risk of dying from breast cancer.

Dr. Avrum Bluming has written that these studies, once you evaluate their internal statistics or look at the research they cite to prove their point, did not reach the appropriate scientific conclusions. Thus, millions of women and doctors have been confused about the safety of hormonal therapies.

Diving into the Statistics of the WHI and Million Women Study

Samuel Shapiro, MD, and colleagues from the Department of Epidemiology, University of Cape Town, South Africa, took a deep statistical dive into the Collaborative Reanalysis, the Women's Health Initiative, and the Million Women Study, and concluded that the findings in these studies did *not* adequately satisfy the criteria of time, order, bias, confounding, statistical stability, strength of association, dose/duration-response, internal consistency, external consistency, or biological plausibility.

Their conclusions, after addressing the details, were that HRT may or may not increase the risk of breast cancer, but the statistics on these three studies did not establish that it does.

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Valerie Beral, the head of the cancer epidemiology unit at Oxford, the senior author of the paper, together with her widely respected colleagues, published the Million Women Study. This sounds very authoritative. A million women. Sounds like this should present proof since such a huge number of women are involved.

But get this. The Million Women Study consisted only of two questionnaires separated by about three years and sent to over a million women. In spite of the grandiose title, only 44% of the sample responded to both surveys.

The summary below of the negative critiques of that paper is taken from several critical analyses:

- The second questionnaire was mailed to only two-thirds of the participants, and only 65% responded (65% of 67% is 44%).
- The total incidence of breast cancer in this study was 15,759/1,129,025 or 1.4%.
- Of these, 7,107 or 45% developed in *current* hormone users and 8,652 or 55% developed in everybody else.
- The investigators estimated that for every 1,000 women taking combination estrogen/progestin for five years, there would be an extra six cases of diagnosed breast cancer, and for every 1,000 women taking estrogen alone for five years, there would be an extra 1.5 cases.
- *The authors never explain why current use is harmful and past use is not.* Of that 1.4%, the increased risk of breast cancer was identified only in current hormone users but not in past users, even if past use had exceeded 15 years. The authors never offer a biologic rationale. This criticism has been leveled as well against The Collaborative Reanalysis, The Nurses Health Study, and the WHI.
- The average time from beginning therapy to diagnosis of breast cancer was brief (1.2 years), suggesting to clinicians that, in many cases, cancer had been present before initiating treatment, and the women who filled out the second questionnaire may have been aware of a problem in the breast prompting their participation.
- The study appears to have been selecting this population with, not surprisingly, a high incidence of breast cancer.
- Just over 50% of invited women eventually had a mammogram, suggesting there could have been self-selection bias in the study population. Again, the women who were already worried there was a problem were the ones predisposed to get a mammogram and to follow up on the questionnaire.
- The study failed to take into account that a sizeable number of women switched treatments during the follow-up period – some ceased therapy (22%), others resumed their HRT (19%), and 11% initiated HRT during the study period.

In a paper published eight years after the original Million Women Study report, the same investigators reported that the admittedly small increased risk of breast cancer seen among women taking estrogen was found only among those who started it within five years of reaching menopause. For those starting it more than five years after a final period, the incidence of breast cancer was the same as that found among never users.

Dr. Avrum Bluming asks, how is this biologically plausible? The authors' reliance on questionably generated numbers to the exclusion of biologic plausibility raises serious questions about the reliability of the conclusions they present.

Nick Panay, Chairman of the British Menopause Society, Marlow, UK, in 2012 wrote the following about the Million Women Study: "I believe the use of statistics in this study is intimidating to most readers, and possibly to editors as well. I can't help but feel that these authors decide what conclusions they want to publish and use their data to construct the desired conclusion."

Having been reading the peer review hormone literature for decades, as well as writing and teaching on the science behind hormone therapies, I completely agree!

In an editorial, Joanne Katsopoulos of the Women's College Research Institute in Toronto, Canada, said: "The complexity of the study design makes it difficult to appraise the results and most of us will take the results on face value."

Dr. Avrum Bluming responds to Katsopoulos this way: "Read that statement again." When researchers dazzle readers with an avalanche of findings that require other professionals to "take the results on face value," something is very wrong. It is the

D. Lindsey Berkson, DC, has been a leader in functional medicine, with an emphasis on the gut, hormones, and the environment for several decades. Dr. Berkson has been teaching certification relicensing courses for MDs, pharmacists, NPs, NDs and chiropractors for decades – in the last few years focusing on the gastroenterology module for A4M and hormones and oxytocin for PCCA.

Dr. Berkson formulated Metagenic's first female nutrient line for physicians. Dr. Berkson was a scholar at an estrogen think tank at Tulane University where she worked with the top scientists that discovered "receptor physiology" and growing epidemic of competitive inhibitors found in endocrine disruptors.

Dr. Berkson has authored 21 books; several have been best sellers. She also hosts the Dr. Berkson's Best Health Radio, writes the Berkson Blog (@ DrLindseyBerkson.com), and is a research fellow with Health Sciences Collegium.

researchers' job to make their data available – and readable – so that the data can be assessed independently. And yet Katsopoulos, while admitting it was "difficult to appraise the results," apparently had no qualms titling her editorial "definitive evidence for breast cancer." Definitive?

Its inconclusive conclusions written with authority – based on too little statistics that fly in the face of the statistics that support estrogen – have made physicians and patients alike terrified of estrogen, a foundational hormone that could help our aging population age much slower.

Dr. Avrum Bluming continued:

> This huge (and complicated) endeavor presents an unbalanced picture of risks and benefits (no benefits are mentioned), and seems to value numerical results above context, ignoring data that does not fit with the easy and misleading conclusion that MHT (or HRT) is a direct cause of breast cancer.
>
> The authors fail to say that even if their finding of a small increased risk is valid, breast cancer is currently curable in approximately 90% of newly diagnosed patients. Additionally, they fail to provide a balanced discussion of HRT's benefits.
>
> We (Avrum and Tavris) regret that *Lancet* is facilitating a wide dissemination of this unbalanced and inaccurate reporting. This *Lancet* paper does not provide meaningful guidance to clinicians, and it sows confusion and fear among patients.
>
> The estimated incidence of breast cancer was 6.3% for never users of HRT versus 8.3% for five years of use of the continuous combination MHT – an absolute increase of 2%, or one extra breast cancer for every 50 users. [For estrogen alone, it was one in every 200 users. The statistics do not back up the scary headlines.]
>
> These reports alarm women, frightens them and many of their physicians away from the use of HRT, which will: [bullets put in by Berkson for easier viewing]
>
> • decrease the risk of cardiovascular disease (which kills seven times as many women as breast cancer),
>
> • decrease the risk of osteoporotic hip fracture, which is associated with almost as many deaths annually as breast cancer,
>
> • decrease the risk of Alzheimer's Disease, for which there is currently no available treatment,
>
> • and would improve their quality of life.

monitoring. Dr. Speroff reminded us that doctors had been using estrogen therapy for many decades and getting stellar results. Two randomized trials with dubious statistics should not fly in the face of years of clinical success!

When re-analyses data emerged vindicating estrogen, it wasn't headline news.

The Black Box Warning

Most of the findings linking HRT and breast cancer are weak or statistically insignificant. Still, the FDA has added a black box warning to the label of Prempro (Wyeth's commercial version of HRT and the combination therapy used in one of the arms of the WHI). A caution remains on all commercial preparations of estrogen: "If you have ever had breast cancer, do not take this medication." As you can see, this doesn't correlate with the human data. However, so many lawsuits were filed and won right after this FDA black label was added that many doctors today neither understand nor do they prescribe hormones. Many are understandably fearful of getting sued.

Many oncologists, gynecologists, and researchers have been frustrated with the way the media published big scary headlines and the FDA added the black box warning. The scary statistics on HRT continue to make front-page headlines. The uncovering that estrogen by itself carried no increased risk of incidence of breast cancer was placed in republished versions of the WHI in a tiny paragraph on page 18.

The father of gynecology, Dr. Leon Speroff, co-authored C*linical Gynecologic Endocrinology and Infertility*, the book that trains doctors who care for women. Dr. Speroff was aware of the bad press estrogen had been getting, but he was also aware of its benefits. Dr. Speroff published a flurry of professional articles criticizing hormone replacement being withheld from women and encouraged doctors to keep testing, prescribing, and But fear sells. When re-analyses data emerged vindicating estrogen, it wasn't headline news.

Right before the Women's Health Initiative statistical fiasco, in 2000, Henk Verheul, a medical oncologist and now scientific co-director of the Cancer Center of Amsterdam, and his research group wrote that none of the current treatments for breast cancer – surgery, radiation, chemotherapy – were negatively affected by estrogens, even estrogens that were prescribed at considerably higher dosages than typical estrogen replacement levels. These scientists concluded: "The available studies fail to demonstrate that once breast cancer has been diagnosed, estrogen worsens prognosis, accelerates the course of the disease, reduces survival or interferes with management of breast cancer. It may therefore be concluded that the prevalent opinion that estrogens and estrogen treatment are deleterious for breast cancer patients needs to be revisited."

Of the 20 studies between 1980 and 2008 that showed estrogen was not only safe for breast cancer patients but was also protective, only the HABITS study found an increased risk of recurrence in breast cancer patients on HRT. As previously stated, this risk only occurred if the women were on tamoxifen, which "blocked" the action of estrogen.

See Part III for information on tamoxifen and more on hormone therapy. ◆

Abridged: Full article with appendices and references are posted online at www.townsendletter.com.

Biochemical Observations on Women's Moods and Hormones

by Phyllis Bronson, Biochemical Research Foundation (Aspen, Colorado) and D. M. Smith, Department of Chemistry and Biochemistry, University of Denver (Denver, Colorado)

The Women's Health Initiative, a huge randomized study[1] on the impact of hormones on women's health, ignored the statistical relevance of the breakdown of the data; for many years the estrogen and synthetic progestin aspects were lumped together. It was only more recently that independent researchers began to tease apart the data and found that the women using just estrogen, even in a less than perfect form such as Premarin®, fared far better with the areas of gender health issues[2] than those on combination therapies utilizing synthetic progestins, not real progesterone. The standard bearer, Provera, which is in fact a progestin, has long replaced natural progesterone; it and indeed all the synthetic analogues block real endogenous or exogenous progesterone from reaching its receptor.[3] This same mechanism of action also may block testosterone from properly accessing its receptor – a concern as testosterone is being greatly researched as an anti-cancer molecule.

Figure 1: Infrared Spectroscopy of Synthetic Progestins (Provera©)

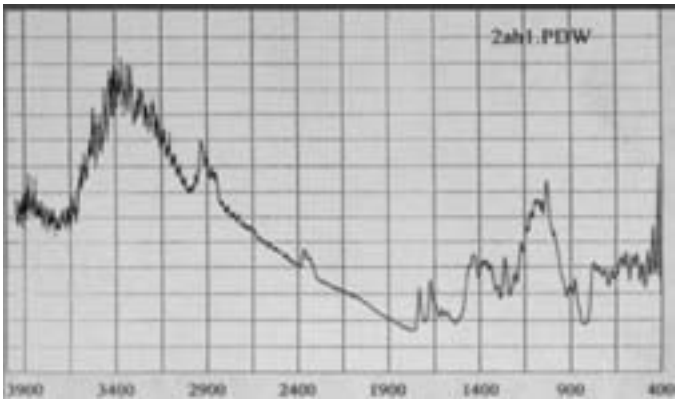
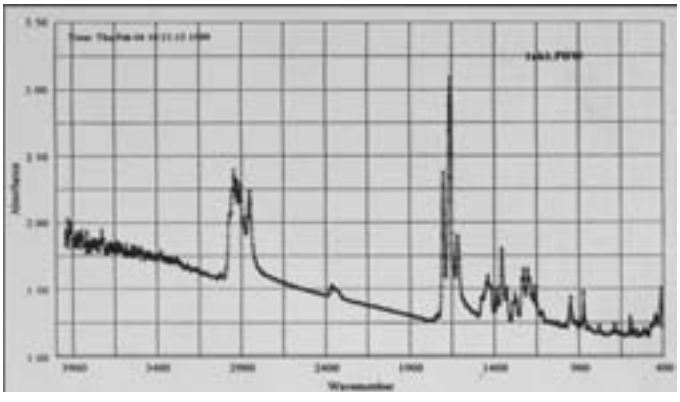


Figure 2: Infrared Spectroscopy of Natural Progesterone



There is increased concern in the demographic of young women, presenting with serious depression, who have been on birth control often for years. A large Danish study published recently is exemplary of the overarching issue here: the potential impact of birth control pills on major depression.[4,5] Women who use hormonal contraceptives are at increased risk for suicide attempt and suicide. The highest relative risk is seen in adolescent women, the Danish study indicates.

The reasons for mood disturbances are most plausibly linked to the fact that synthetic progestin blocks the receptor from accessing the real molecule, thereby inhibiting the calming effect at the GABA-AR, induced by either endogenous or exogenous (transdermal compounded cream) progesterone. Instead of properly opposing excess estradiol from becoming aggressive, synthetics such as MPA (medroxy-progesterone acetate) and all synthetic analogues may block estrogen too much and not allow its often-extraordinary benefits on mood.

There is an important distinction between actual progesterone (which is native and natural to female biology) and progestins, which are the synthetic analogues used pharmacologically in birth control pills, contraceptive devices, and most traditional hormone replacement. (HRT). This is in contrast to B-HRT, which utilizes real progesterone as well as other bioidentical molecules. Progesterone has a calming effect on the nervous system,[6] due to the interface of the progesterone molecule and the GABA-AR (receptor) that induces the opening of the chloride ionic channel; this impacts neuroinhibition and is the route to helping anxiety patterns. The way anti-anxiety drugs are developed (benzodiazepines) is predicated on this molecular aspect. Synthetic progestins (ie, medroxy progesterone acetate-MPA and their cousins and analogues) have a very different impact on these mechanisms of action and do not help anxiety.[3]

The correlation between the structural and clinical evidence-based interpretations from the women who have

been treated for anxiety using progesterone and GABA is evident: women using real progesterone report increased calmness and reduced impatience and irritability and an increased sense of general well-being. The latter is thought to be a result of reduced cerebral edema and other water retention due to the opposing effects on hydration between the less hydrophilic progesterone and the more hydrophilic synthetic progestins.

As shown by Figures 1 and 2, the structural differences between the molecules, an additional -CH3 on C-6 (B ring) and the acetate on C-17 (D ring) in the case of the progestin (Provera), are revealed by the infrared spectra[7] taken under identical experimental conditions. An intense broad band centered at about 3390 cm^{-1} is due to hydrogen bonding interactions of the water associated with the hydroxyprogesterone acetate. In contrast, the natural progesterone has virtually no water associated with the molecule. The overwhelming differences in the spectra are due primarily to the amount of water associated with each molecule under the spectroscopic conditions. The medroxyprogesterone acetate is clearly much more hydrophilic than the natural progesterone.

There has been a consistent push to create newer and more improved analogues of women's hormones. However, the perfect structures may already exist, which are identified as being exactly what the female body produces. Women do better when bioidentical structures are administered in proper balance.

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Dwight Morrell Smith, PhD, served the University of Denver (DU) from 1972-1989 and 1992-2017 as professor of chemistry, academic administrator, and chancellor of the university. He currently is Professor Emeritus and Chancellor Emeritus. His early career included appointments at the Texaco Research Center, Wesleyan University (CT), Hope College (MI), and Scripps Institution of Oceanography (CA). His research has included heterogeneous catalysis, infrared spectroscopy, structure and reactivity of black carbon, and animal-based inhalation studies.

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Anti-Gravity Suit Failure

by William Ferril, MD©2019

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Is Aging Really a Sulfur Deficiency Syndrome?

If you ignore sulfur biology and you become deficient, you will begin to look like you feel! You see, sulfur offers the healthful body six basic benefits. First, it provides molecular 'glue' that holds things like hair, hooves and your nails together. In fact, the glue it provides largely withstands acidic digestion. This is why, even when soaking a corpse in hydrochloric acid, the hair, and nails remain so resistant to breakdown; the double sulfur bond is the reason!

The second health benefit is beyond the view of the electron microscope, but it proves – as a major component of your body's water content – to resist the force of gravity from squeezing out precious cellular fluid. You see, life is compressional! Sulfur is one of your body's main antigravity support molecules. If you find yourself lacking in sulfur content, you compress. In other words, the shrinkage of old age begins!

Biologically active sulfur is acquired by consuming the sulfur-containing amino acids, methionine and cysteine. The third benefit involves sulfur's utilization at the rate of 1 billion times a second! If you lack this ability, you suffer from an increased likelihood of becoming depressed, acquiring heart disease, suffering from high blood pressure, and having impaired detoxification ability within your liver. These additional details are all discussed in my other writings. Here it is only initially important for you to realize that sulfur plays an important role in your lasting health! Two common markers can alert you to whether you enjoy the ability to utilize sulfur at the rate of 1 billion times a second! They are your homocysteine level and your mean corpuscular volume. If either one of these elevates, suspect the sulfur metabolic pathway to be compromised!

The fourth pathway for how sulfur content benefits you involves important metabolic facilitators containing sulfur for activity. They are thiamine, biotin, lipoic acid, pantothenic acid, glutathione, heparin, and threonine. In each of the above important bioactive molecules, sulfur provides the active site! Note, glutathione is ideally present in every cell of your body! **It is the only way your body can store metabolically active sulfur**. The quantity of its presence determines much about your ability to prevent rust (oxidation) when toxins make their way into your cells! Recall, you can only acquire the sulfur within glutathione by consuming and absorbing either the amino acid methionine or cysteine. This is one way that low/incomplete protein diets can accelerate aging!

The fifth metabolic pathway for sulfur activities within the body involves the requirement for its critical presence within some of your hormones, for example, the hormone insulin. The sulfur-sulfur bond provides structural stability in the three-D world of molecules. In the molecular dimension, size and shape remain critically important.

As was introduced above, metabolically active sulfur also is the major player for detoxification pathways within your liver. *For example, every dose of acetaminophen consumes body sulfur and irreversibly damages your hemoglobin.* Obviously, the less sulfur available, the more damage. Glutathione contains sulfur. It gets compromised by things like acetaminophen because, to prevent this toxin from accumulating, biologically active sulfur (glutathione) gets used up.

So, let's say you take acetaminophen for joint pain. Emerging science suggests this may be a poorly thought out practice! *You see, acetaminophen consumes the very sulfur source that replenishes your joints!* Anticipate the A*nti-Gravity Suit System* introduced below.

Because tracer studies show that biologically active sulfur derives from either methionine or cysteine, diets higher in these amino acids confer health advantages. In fact, studies show high meat consumption confers up to four times the amount of these amino acids than does the vegan diet! Moving towards the lacto-ovo vegetarian diet increases these amino acids to half that of high meat intake consumers. Be careful with the idea of supplementing with methionine or cysteine, they can be toxic. Ideally, encounter them in your diet from eggs, meat, whey protein, and/or fish. Alternatively, if you want to remain vegan, pay close attention to ensuring adequate consumption of garlic, cilantro, onions, and Brussel sprouts.

The central role that sulfur plays in maintaining your anti-gravity systems deserves special emphasis. Recall that your life is compressional! Behind only phosphorus and calcium, sulfur is the third most abundant mineral within your body! I hope you are wondering where in your body it resides. Well, it is one of the major osmotic forces, throughout your extracellular and intracellular space opposing gravity's opposite force attempting to squeeze out precious body fluid through compression. In your cells it is intricately associated with the cytoskeleton. Outside of your cells, it lines all body surfaces and tubes in what is collectively called mucopolysaccharides or glycosaminoglycans (GAG's). Sulfur content seems to control the viscosity of the inside surface secretions like lung mucus and the protective barrier of the inside surface of the GI tract (its sufficiency here prevents auto-digestion!). It determines whether your cartilage is optimal or dry and cracking, and it determines much about the quality of the inside jelly (vitreous humor) within your eyeballs!

Bottom line concept: the amount of sulfur on the various forms of mucopolysaccharide or glycosaminoglycans (GAG's) determines the electrical potential that pulls water against the force of gravity. Diminished force within your anti-gravity systems becomes evidenced by progressive hunching, crunching, sagging and bagging, where sulfur becomes deficient. How could it be otherwise once you realize that sulfur when fully charged up to sulfate, powerfully pulls water towards it by simple osmosis! Likewise, dry mucous plugs plaguing many lung disease patients results largely from sulfur deficiency! Similarly, if you suffer from drying out, cracking and imploding spinal disc(s), it involve such a process as well. So do your joints. Your skin shrivels because less sulfur translates into more water escaping!

So now it's time to introduce yet another apparent scientific top secret; sulfation factor determines fully active amounts of sulfur placed on all your mucopolysaccharides or glycosaminoglycans (GAG's) throughout your body! What? You have never heard about sulfation factor? Well, it goes by several other alias names like IGF-1 (insulin-like growth factor type 1), somatomedin and the non-suppressible insulin-like activity of the blood stream! Multiplicities in naming confuse and isolate knowledge important for activating how the body heals!

In other treatment plans and books, I delve deeply into how you can enjoy ample amounts of sulfation factor again (remember all the other alias names described above). For now, it is only important to initially appreciate that its levels depend on the appropriate interplay of thyroid hormones, growth hormone, androgens, good night's sleep, appropriate fasting intervals, exercise, and adequate nutrition!

The metalloenzymes comprise the sixth metabolic pathway that sulfur benefits. Usually the metalloenzymes contain either selenium or iron. These will be discussed in the future because so many other variables make their discussion convoluted. For now, just be aware that some of your important enzymes contain selenium or iron and this cannot happen unless sulfur-containing amino acids occur in ample supply.

Finally, for now, it's scientifically emerging that although the literature focuses heavily on nitrogen balance (protein adequacy) the emerging literature is now focusing on sulfur balance (*anti-gravity systems* balance) as well! So, perhaps aging is sometimes really just the result of a worsening sulfur deficiency syndrome, which impairs the standard issue anti-gravity suit equipment!

This concludes the introductory discussion for one component of your anti-gravity system: sulfur. In other articles, I discuss other components like the American diet largely being composed of reversed mineral content, the internal war of your opposing steroid forces, the hormone cascade that charges up your cells, unstable proteins secondary to mineral imbalance, and the energies that heal contrasted with the energies that maim!

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William B. Ferril, MD, earned a bachelor of science degree in biochemistry and doctorate in medicine at UC Davis, California. He completed his postgraduate education at Sacred Heart Medical Center in Spokane, Washington in 1986. For more than 10 of the past 34 years, he practiced medicine on the Flathead Indian Reservation in western Montana. It was there; during this posting that he gained experience for the topics in his books. He has written three other books besides the 1st and 2nd editions of *The Body Heals; Glandular Failure Caused Obesity* and *Why is My Doctor so Dumb?* His latest work, *The Unseen Hand* is now available.

Dr. Ferril has lectured hundreds of doctors on the biochemistry of the steroids and the best testing methods. Since childhood he has remained fascinated with answering the mystery for why some people age gracefully while others do not. The biochemistry of the steroids provides part of the answer. Additionally, the appalling common malnutrition occurring in America provides more insight. He maintains a complementary medicine consulting practice in Whitefish, Montana.

His website, williambferrilmd.com, provides additional articles, references and over 30 Youtube healing summary videos. In addition, articles are arranged by diagnosis and cover Dr. Ferril's treatment strategies for achieving wellness again.

The Stressed Brain – A Clinician's Perspective Part 1

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The full article with references is posted online at www.townsendletter.com.

Abstract

This two-part series focuses on what stresses the brain and the different mechanisms that become triggered as a result. The stress-vulnerability model is highlighted to introduce the notion that with sufficient stress there will often be resultant mental health consequences. The concept of allostasis, allostatic load, and allostatic overload are highlighted to demonstrate the non-linear stress mechanisms (i.e., neural, neuroendocrine, and neuroendocrine-immune mechanisms) that help individuals to physiologically adapt to the stresses in their lives. The consequential effects on the body and brain (principally, the prefrontal cortex, amygdala, and hippocampus) that result from allostatic load and allostatic overload are further discussed. Many different sources of chronic stress are noted in reference to brain mechanisms and include the following: prenatal and postnatal early life experiences; social isolation, loneliness, and socioeconomic status; personality factors; medical diseases; and psychiatric illness (mental disorders), and suicide. Since the brain determines how the world is perceived and responded to, everything that chronically stresses this organ is positioned as being of paramount importance to an individual's current and future morbidity and mortality.

Introduction

Suffering is ubiquitous and inevitable. No living person escapes personal catastrophes, hardships, regrets, failures, loss, disease, and emotional pain. When a person is literally confronted with an overwhelming life situation, at some point – often described as a "breaking point," or "mental breakdown" – he will seek out assistance from a healthcare professional, such as a family physician, mental health professional, or a psychiatrist. The patient's psychological distress is usually ascertained to have passed some threshold diagnostically to meet criteria established for having a mental (psychiatric) disorder, or maybe even several mental disorders. No matter what the particular mental disorder or disorders the patient has been diagnosed with, the resultant signs and symptoms of psychological distress happened as a consequence of mediating factors arising from the patient's ***stress-vulnerability*** (or ***stress-diathesis***). The notion of stress-vulnerability was described decades ago in the context of schizophrenia and relapse.[1] In simple terms, with sufficient stress combined with significant vulnerability, there will always be some risk of having significant psychological distress resulting in clinically meaningful (and identifiable) psychological signs and symptoms. All patients (and therefore all humans), have their own intrinsic vulnerabilities (e.g., genetics), and when mediated by sufficient psychosocial stressors (e.g., a life crisis, relationship problems, and/or substance abuse), the net result is usually psychological distress often manifested as diagnosable psychiatric illness like major depressive disorder (MDD), generalized anxiety disorder (GAD), and post-traumatic stress disorder (PTSD).

Concepts of Allostasis, Homeostasis, Allostatic Load, and Allostatic Overload

When reviewing contemporary models to explain the aforementioned schemata, certain terms need to be defined before proceeding. ***Allostasis*** coined by Sterling and Eyer,[2] refers to biological adjustments that allow an individual to adapt to particular challenges that happen over the lifespan. Adapting to such challenges demands the synchronous though non-linear activation of many different physiological processes, such as neural, neuroendocrine, and neuroendocrine-immune mechanisms.[3] Allostasis begins with the brain and happens or is instigated by how an individual perceives and interprets any given situation. Conceptually, however, allostasis is different from ***homeostasis***, which it is often compared to because of related concepts. Homeostasis is about ensuring survival, and refers to "physiological parameters like blood oxygen and pH" that are "maintained within a narrow range" (p.37).[3] Allostasis, on the other hand, is about adaptation, but the physiological adaptations may not ensure survival because they can become deleterious over time and cause irreversible damage.

Allostatic systems become quickly activated when an individual is confronted by acute stress, which normally return to their baseline states rather quickly. It is more commonly chronic stress, however, that results in problematic sequelae. ***Chronic stress*** has been defined as "ongoing demands that threaten to exceed the resources of an individual in areas of life such as family, marriage, parenting, work, health, housing, and finances" (p.638).[4] In physiological terms, chronic stress refers to a "pathological state that is caused

by prolonged activation of the normal acute physiological stress response, which can wreak havoc on immune, metabolic, and cardiovascular systems" (p.56).[5] When an individual is faced with chronic stress, which is common among most psychologically distressed patients, it may seem enduring and without a clear ending. Chronic stress will eventually overwhelm allostasis, and cause ***allostatic load (AL) and allostatic overload*** **(AO).** AL represents body degradation that results from repeated allostatic responses during stressful situations.[6] This results when an allostatic system fails to habituate to the recurrence of the same stressor, fails to shut off following overwhelming stress, and/or whose response is deficient resulting in heightened activation of other, normal counter-regulatory systems.[3,7] AO is thus an extension of AL, which often results in irreversible damage to body organ systems, and/or mental illness. Thus, unmitigated chronic stress that results in AL and AO will typically cause all sorts of psychological distress signals, especially among individuals vulnerable to mental illness (Table 1).

McEwen[9] has described the common mechanisms involved in allostatic responses that work in a nonlinear but coordinated manner, such as the autonomic nervous system, ***hypothalamic-pituitary-adrenal (HPA) axis***, and the immune, metabolic, and cardiovascular systems. Activation results in the release of catecholamines from both nerves and the adrenal medulla (i.e., the ***sympathetic-adrenomedullary system; SAM***), and the secretion of ***corticotropin*** (a.k.a., ***adrenocorticotropic hormone; ACTH***) from the pituitary, which then results in the release of cortisol from the adrenal cortex.[5,9] By contrast, when allostatic responses are attenuated, the aforementioned systems bring cortisol and catecholamines to their baseline levels. This happens when the stressor, or the component that mediated these systems to act, have been contained. AL, on the other hand, happens when the inactivation is insufficient, and then the individual gets exposed to too many stress hormones, which can happen over "weeks, months, or years," leading to "pathophysiologic consequences" (p.172).[9]

What should be evident and perhaps obvious is that AL not only results in psychological distress, but also in consequences that harm both the brain and body over time. There are apparently four types of responses associated with AL (Table 2).[9] The first response involves frequent stress over time, such as surges in blood pressure resulting in myocardial infarction among vulnerable individuals. Or, in primates (which are genetically very similar to humans), repeated elevations in blood pressure over weeks and months can hasten atherosclerosis and increase the risk for myocardial infarction.

The second response involves being exposed to the same repeated stress, but adaptation is insufficient, which results in protracted exposure to stress hormones.[9] An example of this was evidenced in a study in which healthy male subjects were exposed to brief psychosocial stressors (i.e., mental arithmetic and public speaking) in front of an audience.[10] The group of men that were denoted as 'high responders' displayed large increases in salivary cortisol in response to each of the experimental treatments between days 1 and 5. These men were unable to show any habituation in their adrenocortical stress response despite being exposed to repeated and predictable psychosocial stressors.

The third response happens when the allostatic system cannot be inactivated once the stressful trigger has ended, which then prolongs the allostatic response.[9] An example of this has been documented among women with depressive illness, who experienced a long duration of "moderately elevated serum cortisol concentrations" that interfered with the formation of bone, resulting in decreased bone mineral density (p.173).[9]

In the fourth and final response, the allostatic system does not adequately respond (i.e., under responds), and this results in compensatory increases in other bodily systems.[9] For instance, if the secretion of cortisol does not increase when an individual is stressed, there could be a resultant increase in inflammatory cytokines since these substances are normally "counter-regulated by cortisol" (p.173).[9] This mechanism is purported to be responsible for the development of autoimmune and inflammatory conditions due to a hyporesponsive HPA axis.

Since both AL and AO operate on a continuum, AO represents a state when organic pathology emerges, which happens when the body breaks down over time due to unmitigated chronic stress and inadequate allostasis. Similarly, AO also emerges when the brain becomes exposed to chronic stress and inadequate allostasis, and its embodiment which is the mind, likewise breaks down and manifests sufficient psychological signs and symptoms characteristic of psychiatric illnesses, such as MDD or GAD.

Specific Brain Regions and Their Associated Stress-Response Mechanisms

Specific brain regions – i.e., the ***hippocampus***, ***amygdala***, and ***prefrontal cortex*** (PFC) – have been implicated in chronic stress and AL. The hippocampus is a "region in the medial temporal lobe that is instrumental for learning ➤

Table 1. Psychological Distress Signals of Allostatic Load and Overload (in no particular order of appearance; Adapted from: Prousky[8])

- Anxiety and/or persistent or intense episodic panic attacks
- Depression
- Despair
- Helplessness
- Insomnia
- Lack of optimism (absence of a positive outlook)
- Anger
- Denial
- Guilt
- Hostility
- Hyperactivity
- Isolation
- Restlessness
- Shame
- Addictive behaviors (e.g., cannabis and alcohol) to anesthetize feelings
- Inability to delay gratification
- Not eating, under-eating, or over-eating
- Habitual cutting or the desire to harm oneself
- Suicidal thoughts
- Homicidal thoughts
- Delusions
- Dissociation
- Depersonalization
- Grandiosity
- Hallucinations
- Hyper-religiosity or bizarre religious beliefs
- Mania
- Obsessions
- Paranoia
- Persecutory thoughts

➤

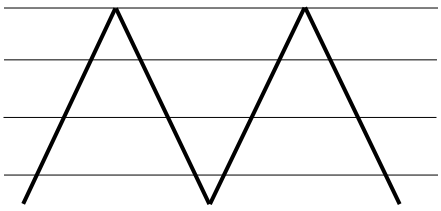
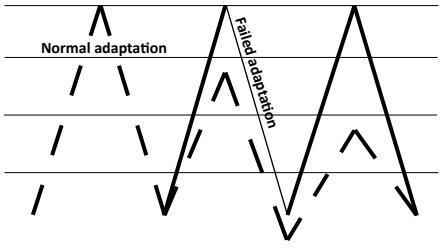
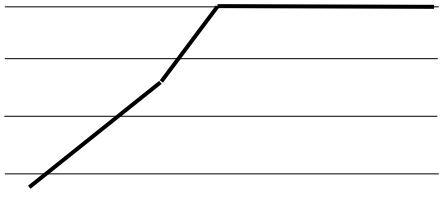
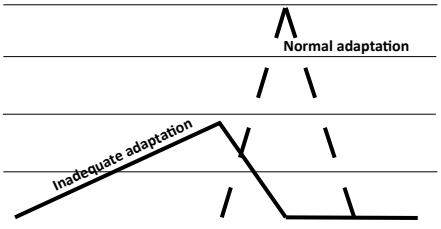
and remembering declarative and spatial information, processing the contextual aspects of emotional events, and regulating visceral functions, including the HPA axis" (p.435).[11] The amygdala is anatomically adjacent to the hippocampus, and "rapidly assigns emotional significance to environmental events, and it regulates physiological and behavioral responses to those events" (p.435).[11] The PFC is what makes us human and is a large brain region that occupies "the anterior portion of the frontal lobe" is "connected with the hippocampus," and is "broadly involved in higher cognitive functions (e.g., working memory and executive control), as well as the control of emotion, mood, stress functions, and impulsive actions" (p. 435).[11]

These brain areas work in a collaborative manner when something is perceived as stressful (i.e., threatening) or even meaningful. This circuitry involves the amygdala and hippocampus – i.e., limbic brain structures – "that process experiences by interfacing with lower vegetative brain areas (such as the hypothalamus and brainstem) and higher cortical areas, particularly the prefrontal cortex" (p.434).[11] So there are top-down mechanisms whereby the PFC attempts to exert some measure of control over limbic brain structures, and there are bottom-up mechanisms whereby limbic brain structures exert some measure of control over the PFC. Of particular importance is that the projections from the amygdala to the PFC are greater than those projections coming from the PFC to the amygdala.[12] As a result, our emotions can sometimes take over and control behavior because our amygdala, when triggered by fear, exerts more influence over how the PFC operates.[12] As one well respected researcher noted, "this hostile takeover of consciousness by emotion" happens because emotions "monopolize consciousness, at least in the domain of fear, when the amygdala comes to dominate working memory" (p.226).[13]

As we just noted, fear is an important mediator or perhaps mobilizer of activity, and yields physiological and behavioral consequences – such as fight, flight, or freeze – that have been experienced by all human beings. This brings us to another part of our brain's limbic system, called the ***thalamus***, which functions as a central relay station by sending motor and sensory signals to the cerebral cortex (i.e., houses the PFC) to be processed and interpreted.[12] So when distilling the pathways involved in strong emotions, such as fear, there are two pathways that ought to be reviewed further since they have important treatment implications, which will be addressed in future articles. The first pathway involves sensory information proceeding from the thalamus to the cerebral cortex (and PFC), and then to the amygdala, which then activates physiological responses that includes the ***sympathetic nervous system (SNS)*** and hormones such as cortisol and epinephrine (a.k.a., adrenaline) to assist our biology in getting mobilized for action.[12] The second pathway involves sensory information proceeding from the thalamus directly to the amygdala (i.e., as a result of being triggered by experiences that previously created fear), initially bypassing the cerebral cortex (and PFC), and directly activating the SNS as described above.[12]

With respect to the hippocampus and how it interfaces with the above-mentioned brain pathways, there is a lot of cross-talk that happens between the hippocampus and amygdala, and between the hippocampus and PFC. For example, should any of the aforementioned pathways become activated, the hippocampus (i.e., is rich in glucocorticoid receptors) can modulate the stress response by inhibiting or activating

Table 2. Four types of AL responses (**Adapted from:** McEwen[9])

Response #1 	Repeated "hits" result in frequent allostatic stress responses.	Causes accumulated damage over time (e.g., surges in blood pressure over weeks or months can hasten the development of atherosclerosis and increase the risk of myocardial infarction).
Response #2 	Repeated stress of the same type results in failed adaptation.	Causes protracted exposure to stress hormones and consequential damage over time (e.g., public speaking causes the same adrenocortical stress response each time).
Response #3 	Allostatic responses cannot deactivate once the stressful trigger has ended.	Causes prolonged allostatic responses and consequential damage over time (e.g., long-term depressive illness leading to moderately elevated serum cortisol levels, and decreased bone mineral density).
Response #4 	Inadequate allostatic response.	Causes compensatory increases in other bodily systems increasing vulnerability towards autoimmune and inflammatory conditions (i.e., associated with an increased amount of inflammatory cytokines).

corticotropin-releasing hormone (CRH) from the hypothalamus, which plays an integral role in the eventual release of cortisol from the adrenal cortex.[14] In situations of chronic stress, the hippocampus can become impaired or disrupted, and won't be able to terminate the stress response, leading to heightened HPA activity, and a consequential augmentation of damage-inducing adrenal steroids over the long-term.[11] Moreover, the hippocampus receives information from the PFC (just like the amygdala), and also influences the PFC regarding the importance of external stimuli, including threatening information (e.g., fear).[14] The hippocampal stress response is also linked to the amygdala's stress reactivity, but it differs in the way emotional events become managed. Whereas the amygdala tags emotional content, such as fear, the hippocampus further tags emotional content by forming episodic representations of emotional content in terms of its contextual meaning.[15]

All of this information has relevance to the pathways described earlier because when something is deemed chronically stressful by context-driven emotional experience, for which there was an initial strong reaction by the amygdala, the hippocampus undergoes specific neuroplastic changes that result in diminished coupling with the PFC. This results in increased stress-vulnerability to life experiences and consequently less top-down control.[15] Stated another way, diminished hippocampal functionality impairs the PFC's inhibitory control over the amygdala.[16] Diminished hippocampal functionality from chronic stress will further undermine an "individual's ability to process information in new situations and to make decisions about how to deal with new challenges or stressors" (p.435).[11]

To summarize the information presented above on brain structures and stress-related mechanisms, it can be ascertained that (1) the amygdala plays a prominent role in emotional processing; and (2) these three brain structures interface with each other and "unite aspects of cognition, memory, executive function with elements of emotional regulation" (p. 1170).[16]

Stress, Atrophy, and Damage to Specific Brain Structures

With chronic stress and AL, the brain undergoes plastic changes, which results in atrophy of the hippocampus, amygdala and PFC.[17] While chronic stress does lead to structural plastic changes, the human brain does possess "a life-long and clinically significant capacity for reversible, structural plasticity" (p. S22).[7] This is good news since the effects of chronic stress can at least, in part, be attenuated by appropriate measures taken by an individual over the course of his lifetime.

With chronic stress, dendrites in neurons in the hippocampus and PFC shrink, become shorter and less branched, and these changes result in diminished synaptic output.[7] These changes further compromise an individual's "capabilities for nuanced cognitive function, memory and self-regulation" (p.S22).[7] On the other hand, the same type of chronic stress causes an expansion of dendrites and increased synaptic input to an area of the amygdala known as the basolateral amygdala, which results in heightened anxiety, aggressiveness, and vigilance.[7]

One hypothesis that has been advanced is the ***glucocorticoid cascade hypothesis (GCH)*** of stress and aging, which refers to the chronic inability of the hippocampus to shut off the HPA axis, which leads to persistent damage to this brain structure and PFC over time.[3,7] Glucocorticoids also potentiate the release of damaging extracellular levels of excitotoxic amino acids (EAA) under stress, such as glutamate, and this happens within the hippocampus, and other brain regions.[17] Glial cell depletion (or alterations) have also been implicated in atrophy of brain regions like the hippocampus, amygdala and PFC.[17] The fact that all of these particular brain areas become targets of chronic stress suggests that a common mechanism may underlie the resultant atrophy and damage that has been noted.[17]

Of interest is the notion that glucose availability within the brain plays a role in mediating resilience or damage. A deficit or lack of available brain glucose can mechanistically lead to excitotoxic cell death within the hippocampus and likely other brain areas, whereas sufficient brain glucose may reduce excitotoxic cell damage.[17,18] A lack of available glucose within the brain has even been proposed as a limiting factor in an individual's free will because self-control demands available brain glucose, and with less available brain glucose, behavior (and therefore self-control) become more limited until brain glucose levels are repleted or restored to normal.[19] Though there are other mechanisms (i.e., such as an evolving number of genomic-mediated molecules implicated in stress-induced dendritic remodeling[20]) to account for neuronal damage, glucocorticoids, brain glucose debt or insufficiency, an unrestrained release of EAA, and glial cell depletion (or alterations) cause neuronal death and therefore neuronal loss in these particular brain areas.

Editor's Note: The second half of this article will be published in next month's issue.
The full article with references is posted online at www.townsendletter.com.

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Dr. Jonathan E. Prousky graduated from Bastyr University (Kenmore, Washington) in 1998 with a doctorate in naturopathic medicine. He furthered his clinical training by completing a family practice residency sponsored by the National College of Naturopathic Medicine (now the National University of Natural Medicine). In 2008 he obtained a master of science degree in international primary health care from the University of London, which focused on clinical epidemiology and evidence-based research. In 2016 he obtained a master of arts degree in counselling psychology from Yorkville University.

At the Canadian College of Naturopathic Medicine, Dr. Prousky's primary responsibility is the delivery of safe and effective naturopathic medical care in his role as the chief naturopathic medical officer. He was the first naturopathic doctor to receive the "Orthomolecular Doctor of the Year" award in 2010. In 2017 he was also the first naturopathic doctor to be recognized for his longstanding commitment to mental health by being inducted into the "Orthomolecular Hall of Fame." Dr. Prousky is the author of several texts, such as *Textbook of Integrative Clinical Nutrition*, and *Anxiety: Orthomolecular Diagnosis and Treatment.*

The Cost of Lost Connections

review by Jule Klotter

Lost Connections: Uncovering the Real Causes of Depression – and the Unexpected Solutions by Johann Hari
Hardback; ISBN 978-1-63286-830-5; 321 pp; $28.00 (US)

Johann Hari, journalist and author, has suffered with depression and anxiety from a very young age. As a teen he was relieved to learn that his depression was caused by low levels of serotonin in the brain and could be fixed with one of the new selective serotonin reuptake inhibitors (SSRIs) – or so he was told. He welcomed the treatment and held fast to the serotonin theory even though the darkness kept creeping back despite increased doses and adverse effects, like weight gain. Even at high doses, however, the depression remained. After years of gentle prodding by his therapist, Hari was weaned from the SSRI. He began a three-year worldwide investigation, conducting over 200 interviews with researchers, social scientists, doctors, therapists, people with depression, and those who recovered. The result is his 2018 book *Lost Connections: Uncovering the Real Causes of Depression – and the Unexpected Solutions.*

In the course of his research, Hari learned that depression and anxiety are no longer viewed as separate entities; they are "twinned": "Everything that causes an increase in depression also causes an increase in anxiety, and the other way around. They rise and fall together." He found that the depression/anxiety affecting some and the general unhappiness affecting many others have less to do with brain chemistry and more to do with our world and how we live. This is not to say that biology is not a factor. Pharmaceuticals do help some people; and, *Townsend Letter* has published numerous articles on the use of orthomolecular and nutrition therapies for addressing behavior and psychiatric disorders. But equally important (or in some cases more) are social factors and life circumstances.

Hari learned that the symptoms of grief match the symptoms for depression: "If you simply use the checklist, virtually anyone who has lost someone should be diagnosed as having a clear mental illness." These symptoms are considered reasonable and acceptable after the death of a loved one. Hari began to wonder: "What if depression is, in fact, a form of grief – for our own lives not being as they should? What if it is a form of grief for the connections we have lost, yet still need?"

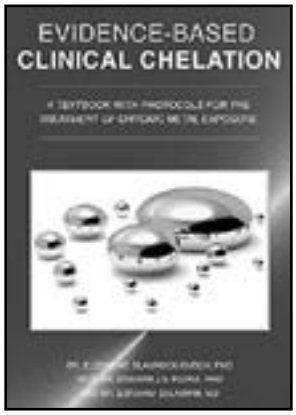
From interviews with clinicians and social scientists, Hari learned that lost connections and disconnections can take many forms: disconnection from meaningful work, from other people, from meaningful values, from childhood trauma, from status and respect, from the natural world, from a hopeful or secure future. Do you engage in work that touches you and has purpose for you, or are you trapped in a deadening cog-in-a-wheel job? Do you have people with whom you share "mutual aid and protection" and meaningful exchanges? Do you follow goals that arise from your core being and heart, or do extrinsic values (eg, money, possessions, outside approval), "sold" through media and culture, rule? Have you received aid and support to face traumatic events and their effects in your life? Are you treated with respect? Can you welcome the sense of vastness that comes with being in nature, which is an antidote to self-absorption and ego's prison? Questions like these can help uncover sources of depression and anxiety.

Just as disconnection contributes to depression/anxiety, reconnection can ease, even resolve, it. Hari writes about seven forms of reconnection "that early evidence suggests can begin to heal depression and anxiety." One example occurred in East London where patients with depression "were prescribed" to take part in a volunteer group. One group was assigned to transform "an ugly scratch of scrub and concrete" into a garden of flowers and vegetables. In the process they found connection. They found similarities in life experience despite differences in backgrounds, gender, ethnicity, and religions. They began to form a tribe of mutual support. Their project required that they learn about horticulture and connect with nature. And they gained respect and appreciation from those living in the community as they transformed the ugly area into a place of beauty. Sam Evenington, a doctor who took part in this social prescribing program, "learned, especially with depression and anxiety, to shift from asking 'What's the matter with you?' to "What matters to you?' If you want to find a solution, you need to listen to what's missing in the depressed or anxious person's life – and help them to find a way toward that."

As Hari points out in this book, long-term stresses and insecurity, severe negative events, and poverty greatly increase the risk of depression and anxiety. Because of our public health response to the COVID-19 pandemic – consisting primarily of business restrictions, school and church closures, social distancing, and wearing masks – communities have experienced months of economic uncertainties and social disconnections. As the social-psychological effects of the shutdowns come to fore, *Lost Connections* may offer a much-needed perspective. Hari is an excellent writer, and this book is thoughtful, well referenced, and an enjoyable, easy read. He has excerpts from the recorded interviews, used as sources, on his website (https://thelostconnections.com/). ◆

Treating Chronic Metal Exposure

Evidence-Based Clinical Chelation: A Textbook with Protocols for the Treatment of Chronic Metal Exposure
by Dr. E. Blaurock-Busch PhD with Dr. Swaran J.S. Flora, PhD, Director of the National Institute of Pharmaceutical Education and Research, Raebareli, Lucknow U.P., India, and Dr. Ebrahim Sulaiman, MD, Kuala Lumpur, Malaysia
Introductions by Prof. Abdulkareem Almomen, MD, King Saud University, Saudi Arabia, and Prof. Ulf Lindt, Biology Center, Uppsala University, Sweden



Softcover DIN A4, 120 pages
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This is an easy-to-read manual about chelation therapy, compiled by eminent researchers in the field of metal toxicology. It contains specific protocols for the treatment of chronic metal overexposure, all of which are evidence-based, aiding the inexperienced and the experienced therapist in selecting the optimal chelating agent for the metal(s) in question. Expert chemists and chelation therapists have contributed invaluable knowledge and experience to this book.

Most importantly, this is not a book promoting personal views or preferences. In fact, the information presents and explains different approaches, and allows the reader to draw his own conclusion. All the protocols listed have been modified for the treatment of chronic metal intoxication, included are oral and intravenous treatment plans. The authors explain in simple terms how a chelating agent's bioavailability affects metal binding and how various protocols such as the Cutler Protocol affect the detoxification process, why DMPS binds mercury but not gadolinium, and which chelators are useful for the detoxification of certain organ systems. Also discussed is the importance and effect of treatment pauses.

Diagnostic tests are discussed, including which are useful under certain conditions. Shown are tables comparing the average metal binding ability of the various EDTAs, DMPS, DMSA and other chelating agents. Detailed information explains which chelator binds which metal and why, all of which aids the therapist in finding optimal treatment schedules.

Novel chelating agents are presented such as MIADMSA, a new oral chelator specifically designed for arsenic intoxication, or how natural pectin may be used as an alternative for gadolinium intoxication, which metals are bound by lipoic acid, and why a slightly alkaline environment supports metal binding, preventing the often-feared metal redistribution.

All in all, this evidence-based chelation 'cookbook' should be in medical libraries and on the bookshelf of every physician treating environmental disease.

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Ask Dr. J

by Jim Cross, ND, LAc
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B Vitamin Urine

I am the lucky recipient of a Jesuit high school education in San Francisco. Fathers Becker/Clemo/Pallas/etc. taught me to think laterally and always try to find the root cause of an issue. This ideology is probably what led me into naturopathic medicine originally, but I digress. Father Becker especially taught me that seeking an answer to a vexing problem may also require learning to ask entirely new questions. He also made the point that you may have to see with new eyes or alter your view of the world a tad if you wish to actually understand what the answers to your new questions provide you.

An example of this was my article on gel capsules several months ago. When I realized some people are taking 20 – 40 capsules/day, my interest was piqued to discover if there was a clear-cut answer as to any health issues associated with consuming so many unnaturally "natural" substances. Unfortunately, there wasn't but I still empty my cap contents into a cup with my other powders every morning because it just feels right.

Today my mind stumbled onto another possible issue and the title of today's column: B vitamin urine. For some reason everyone, including those of us with exceptionally clean and diverse diets, require massive amounts of encapsulated B vitamins, sometimes hundreds of times the RDA. You take these exceptional amounts of B vitamins and, like magic, a strange yellow color to your urine appears. This occurs because urinary water-soluble vitamin levels reflect their relative dietary intakes and are suitable biomarkers for measuring those intakes, although vitamin B12 is an exception.[1]

Since B vitamins are water-soluble and are excreted through the urine, everyone thinks that they are harmless. Here is an example of the capsular contents of a typical B vitamin supplement:

B1	100 mg	8,333% of the Daily Value (DV)
B2	12.7 mg	977% DV
B3	108 mg	675% DV
B6	16.7 mg	982% DV
Folate	1007 mcg	167% DV
B12	400 mcg	16,667% DV
Biotin	400 mcg	1,333% DV
B5	100 mg	2,000% DV

No wonder our urine turns yellow! Maybe we shouldn't eat at all and just consume B vitamin capsules.

B vitamin urine should come as no surprise to anyone who has taken physiology or taught physiology, both of which I am guilty of. Basically, your kidneys filter on average 180 liters of blood/day or almost a 50-gallon drum. They accomplish this Herculean task rain or shine or snow, 24/7, and whether we ask them to or not. These large amounts of B vitamins are altering the color of our urine because they are floating around overabundantly in the plasma from excessive supplementation, which forces the kidneys to perform their main function: homeostatic control of blood plasma components. The kidneys then don't reabsorb the excess amount of B vitamins in the plasma and let them slide into the urine and eventually into the nearest urine receptacle. Now, my question today is: by taking such massive doses of B vitamins, are we unduly taxing our kidneys and ushering them into a non-functional form at a faster rate than if we didn't consume those enormous doses? Hopefully, I'll be able to answer this question, as I love my kidneys (as everyone should) and want them to work effectively for as long as possible.

First off, why do B's lead to this discernible yellow color of our urine? Most B-complex vitamins contain riboflavin or B2 which contains flavins or naturally occurring yellow pigments (For you Latin scholars, flavus means yellow).[2] As the kidneys filter out the excess B's, our urine will return to its normal straw yellow color once this housecleaning task is accomplished. Also, most B's are considered supposedly harmless, even when consumed in above dietary doses. The two exceptions are B6 and niacin, which can cause toxicity in moderate to large doses. For niacin, flushing is the major side effect. In addition, one may experience nausea, abdominal pain, dizziness, and liver damage. B6 in overzealous consumption primarily produces a peripheral neuropathy but also can initiate nausea and hypersensitivity to sunlight.[2]

Secondly, are there any studies demonstrating damage to the kidneys from B vitamins? One study from *JAMA* in 2010 was attempting to establish whether high doses of three B vitamins (folic acid, B12, B6) could lower hyperhomocysteinuria and improve diabetic nephropathy. Eligible participants had type 1 or 2 diabetes and a clinical diagnosis of diabetic nephropathy,

with at least 300 mg/day of urinary albumin excretion or ≥500 mg/day of proteinuria. Participants were either given placebo or 2.5 mg/day of folic acid, 25 mg/day of B6, and 1 mg/day of B12. At the end of the study high doses of these three B vitamins resulted in a greater decrease in glomerular filtration rate/GFR and oddly enough also an increase in vascular events in the B vitamin group than in the control group. Granted this is only three B's, but a lower GFR usually indicates some sort of renal deterioration.[3]

After scouring PubMed and various search engines, I could find no studies showing connections between low to moderate doses of vitamin B supplements and kidney damage. Almost every source said side effects of B's are not common unless consumed in very large amounts where the overdose could be signaled by dizziness, frequent urination, constipation, diarrhea, abdominal pain, nausea, vomiting, and/or redness of the skin and itching. Although extremely rare, a person could develop an allergic reaction and experience itching, a rash, swelling, wheezing, or hives. People can also develop side effects to individual B complex components, but none of them list damage to the kidneys as a side effect.[4]

So, what is a poor health care practitioner or Joe/Jane Q. Public to think? When I was teaching physiology, I always attempted to teach my refractory 18-22-year-olds that they needed to decide how healthy they wanted to be in the distant future and to take care of their bodies accordingly in the present. That way they might reach an older age and still be functional physically. My best example was the simple difference between heart rates of 60 and 70 beats/minute. Here is a list:

1 day..... 60 bpm around 86,000 beats
70 bpm around 101,000 beats
1 year..... 60 bpm around 31 million beats
70 bpm around 36 million beats

That is a 5 million difference in one year, which is a 150 million beat difference in 30 years, a 300 million difference in 60 years, etc.

Then I gave the example of identical twins who, until 18 years of age, wore the same clothes, ate the same food, received the same amount of parental love, etc. At 18, they went off to college. One twin started smoking cigarettes, not exercising, and being exposed to large amounts of stress. The other twin exercised daily, never smoked, and practiced Transcendental Meditation religiously. The second twin could conceivably have an average heart rate of 60 bpm and the other twin 70 bpm. Finally, given identical genetics but different lifestyles, I would ask my students whose heart is going to last longer?

I am going to pursue a similar train of thought here. If a person buys the train of thought that they need extremely large amounts of the various B vitamins and they daily consume a B capsule with the above values, are they slowly wearing their kidneys out? Unfortunately, I can't give you a scientific answer but will share what I choose to do.

I have a couple of genetic methylation SNPs that make it harder for me to keep my methylation cycle continuously running in a perfect circle. To help foster a smoothly running cycle, I take a supplement where the recommended dose is three capsules/day, which contain riboflavin at 90 mg (6,923% DV), vitamin B6 at 45 mg (2,647% DV), 5 mg of a mixture of L-5-methyltetrahydrofolate and L-5-methyltetrahydrofolic acid (a mere 1,250% DV), and vitamin B12 at 3 grams (a whopping 125,000% DV). I started out with one capsule/day and sure enough bingo: riboflavin urine. I put on Father Becker's thinking cap and started opening the capsule and taking just a little bit each day and made the capsule last six days. A win/win situation: no more riboflavin urine and a much-reduced supplement bill!

After reviewing this article before I submitted it, Alan McDaniel, MD, gave me a counter thought. He believes that simple and reliable tests to detect B-complex deficiency aren't available yet. So, he gives his patients, whom he thinks require a B multi, a low-dose B vitamin for two reasons. First, to establish if their intestinal tracts are functional enough to indeed absorb the B's which he will ascertain by the color of their urine. Secondly, if they are, then he will titrate the amount down over three doses per day to find a dose that minimally changes urine color. Thanks Alan, it's always awesome to hear other people's thoughts that go beyond my own.

To end, I don't need double blind studies to necessarily guide my clinical decisions. My common sense always seems to take me wherever I need to go. My recommendation: love your kidneys, eat real food, and try to take as low of a dose of any supplement, especially B's, as you can.

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Mercury – Neurotoxicity and Memory

Introduction

As we age, we expect some cognitive decline, but at what point is it considered problematic? For most people, a decline in short-term memory is mild and doesn't interfere with daily functioning. For others it can be an early sign of more serious cognitive changes such as dementia or Alzheimer's disease. When someone says they forget things easily or don't recall where they put their keys, it may or may not indicate neurocognitive decline. For most individuals, poor memory may be attributed to a lack of attention to detail, information overload, or stress. For some it may be due to a change in hormones, nutrient deficiencies, or sleep deprivation. Of course, it may be a sign of a more serious medical condition that should not be overlooked. Exposure to environmental toxicants can also play a role in poor short-term memory. Exposure to toxicants such as mercury can cause neurotoxicity and cognitive changes at any age. When evaluating someone for concerns about changes in short-term memory, there are many factors to explore and tests to run. This article will focus on the effects of mercury exposure as an often-overlooked link to cognitive decline.

Mercury

There are three forms of mercury that people are exposed to in their everyday life, often without knowing they are exposed. Elemental mercury is found in mercury-containing coal, thermometers, fluorescent bulbs, dental amalgam fillings, and latex paints. It eventually enters vapor state and is absorbed through the lungs. It is very lipophilic and accumulates in the brain and kidney. Organic mercury is another form of mercury and is present as methylmercury or ethylmercury. Most methylmercury exposure comes from dietary fish and poultry whereas ethylmercury used to be in vaccines and some antiseptics. Methylmercury, the most toxic form to humans, bioaccumulates in the tissues, crosses the placental and blood brain barrier, and is formed from the conversion of inorganic or elemental mercury in living organisms. It may be found in water and soil as the result of the methylation of elemental and inorganic mercury by microorganisms. It is lipophilic and accumulates in the brain and kidney. Inorganic mercury present as mercury salts is found in cosmetic products like skin-lightening creams and face creams, laxatives, teething powders, diuretics, and antiseptics. It also is formed from the metabolism of elemental mercury vapor or methylmercury. Inorganic mercury is water soluble and does not cross the blood brain barrier.[1,2]

Memory

Several studies show a link between mercury exposure, neurotoxicity, and memory. Methylmercury neurotoxicity was first discovered in adults with acute poisoning.[3] Later it was discovered that it can cross the placental barrier and affect the cognition and development of children exposed in-utero. It is known that eating fish is the main source of exposure of methylmercury, and studies show children in fishing populations experience developmental neurotoxicity.[4] The developing brain is more susceptible to the effects of methylmercury. Prenatal mercury exposure is a concern not only for the mother but also for the neurocognitive development of her offspring. It is well documented that in-utero methylmercury exposure from seafood consumption may lead to neurological alterations, including cognitive and motor dysfunction.[5]

Adults are affected by mercury exposure as well and can cause neurotoxicity signs and symptoms. High dose exposure can cause loss of neuronal cells in specific brain regions, such as the visual cortex and the cerebellum as seen on brain MRI.[5] As people age and memory declines, concerns of dementia and even Alzheimer's disease arise. Given the fact that mercury is a neurotoxin, studies have looked at its links to Alzheimer's disease, AD. Known changes in AD include plaques, beta amyloid protein, neurofibrillary tangles, phosphorylated tau protein, and memory loss; all of which can be caused by

mercury exposure.[6] Patients diagnosed with Alzheimer's disease exhibit higher levels of brain mercury and blood mercury. One study showed that in early onset AD patients blood mercury levels were almost three-fold higher as compared to controls.[7] A cross-sectional analysis of deceased patients in the Memory and Aging Project cohort study, conducted 2004–2013, showed seafood consumption was also correlated with higher brain levels of mercury; however, the higher brain concentrations of mercury were not significantly correlated with increased levels of brain neuropathology.[7] Fish consumption can provide essential nutrients that can be protective of cognitive decline such as essential fatty acids. It is advisable that adults continue the benefits of seafood consumption and at the same time avoid fish known to be high in mercury. A simple blood test for mercury can help determine if a patient's seafood consumption is creating elevated mercury levels in the body.

Other forms of mercury besides methylmercury from fish have been linked to changes in memory. A study of occupational exposure to elemental mercury showed a clear link to mild changes in short-term memory.[8] Changes in short-term memory was assessed through standardized cognitive tests in workers employed at mercury cell chlor-alkali plants. It was found that exposure to elemental mercury on the job was associated with higher urinary mercury levels and poor short-term memory. Most studies on elemental mercury and poor memory come from occupational sources of exposure.

In general practice, poor short-term memory is a common complaint amongst older adults. Although there is a link with mercury and Alzheimer's disease, most patients will be experiencing milder forms of cognitive decline such as poor short-term memory. A link to mercury can be a contributing factor. There are several studies linking mild memory impairment in adults to fish intake. One study used hair analysis to link elevated mercury from fish consumption in adults with memory decline.[9] Although hair analysis was used in this study, the preferred method for detecting methylmercury from fish in the body is blood test. Assessing for mercury exposure should be included in all patients with complaints of poor memory. Below is a case example.

Case

At the end of 2018, a 56-year-old woman who has been managed for surgical menopause with hormone replacement therapy returned with new complaints of fatigue and poor memory. Her neurological and physical exam were normal, and she had no other neurological symptoms. She said the memory concerns were poor short-term memory such as forgetting where she put something or walking into a room to get something and forgetting why. Her long-term memory was fine, and she had no changes in orientation, alertness, speech, thinking, comprehension, or word-finding. Her poor memory didn't interfere with her work or activities of daily living. Upon further questioning she described it as more of a brain fog and forgetfulness. She has

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no headaches or dizziness. She smokes cigarettes, a half pack daily for over 20 years. She eats fish two-to-three times a week, including tuna and swordfish. She is healthy other than she has osteoarthritis and joint pain in hands and knees. She is up-to-date on screening exams such as mammogram, pap smear, dexa scan, and colonoscopy; and all are normal. She sleeps well every night (7-8 hours), exercises regularly, and eats well. Her stress is low, and she feels emotionally balanced.

Her current supplements and medications include the following: vitamin D3 (5,000 IU a day), joint formula with glucosamine chondroitin, DHEA (5 mg once a day), HRT troche biest (2.5 mg/Prog 50 mg/test 3mg).

Labs and imaging were ordered, which included brain MRI without contrast (normal), CBC (normal), TSH (2.0), E2 (18), CMP (normal), lipid panel (normal), testosterone (25), B12, iron and vitamin-D (all normal), DHEA 285 (high), blood mercury 29 (high).

Patient was told to lower DHEA to 5 mg every other day and stop eating all fish and make appointment for a chelation/detoxification plan. Patient was also told to stop smoking and offered help and a referral to a smoking cessation program.

In January 2019, she decided to retest the mercury levels before making a follow-up appointment to begin chelation. The mercury lowered to 14, which is still elevated. Note: the mercury lowered from 29 to 14 without chelation and just by avoiding fish only. She said she had not stopped smoking yet. The chelation/detoxification plan included a detox diet, supplements, and chelation:

Detox Diet (organic and non-GMO)

1. 1 Tbsp ground flax seeds/meal a day on food
2. 1 teaspoon of psyllium husk powder in water
3. 3-4 cups of green tea
4. No red meat
5. Increase legumes, fruits, veggies
6. No sugar, no artificial sweeteners
7. 3-4 servings of cruciferous veggies a day
8. No cow milk products
9. Fish low in mercury is okay
10. Lean meats are okay, such as chicken, turkey, wild game
11. Protein - 20 grams three times a day= 60 grams a day
12. Nuts and seeds are good
13. Complex carbohydrates like quinoa and brown rice are okay
14. No simple carbohydrates; breads, pasta, bagels, tortilla

Supplements for Two Months

1. N-Acetyl Cysteine (NAC; 600 mg twice a day)
2. Cofactor support product (6 capsules/day, which contained vitamin A, vitamin D3, vitamin K1, vitamin B-1, vitamin B-2, vitamin B-3, vitamin B-5, vitamin B-6, vitamin B-12 (as methylcobalamine), vitamin C, vitamin E, biotin, folate (5-methyl-tetrahydrofolate), calcium, chromium, copper, iodine, magnesium, manganese, molybdenum, potassium, selenium, zinc, choline, inositol, boron, vanadium, green tea extract, turmeric)
3. Liver herbal support product, which consisted of milk thistle, beet root, dandelion, burdock and artichoke.

Chelation for Six Weeks

1. Oral DMSA (200 mg capsules). She took one capsule three times a day (6 hours apart) three days in a row then did not take it for 11 days. This equals one round, or two weeks. She repeated this process two times for a total of three rounds, which lasts six weeks.
2. Sauna Therapy. Start with 10 minutes in hot sauna, 30-second cold rinse. Repeat five times and end on cold. Do this one-to-two times a week for 10 sessions

Three months later the blood mercury dropped to 5. It started at 29, dropped to 14 by avoiding all fish, and then dropped to 5 with the chelation and detoxification plan. She is still smoking two cigarettes a day. Her memory is subjectively better, and she feels like she no longer has short-term memory concerns. Her joint pain and fatigue resolved completely. She was told to continue the NAC, cofactor support, avoid fish high in mercury, and stop smoking. She was sent to a local smoking cessation program.

Summary

Poor short-term memory or changes to memory are often a subjective complaint of patients as they age. There are numerous causes of cognitive decline that need to be evaluated for in patients presenting with this concern. Environmental toxicants such as exposure to mercury are often overlooked. It is important when working up a patient for changes in memory that all factors be explored. This includes testing for metals in the body, especially mercury as it is linked to both mild cognitive decline and Alzheimer's disease. Chelation along with detoxification support is an effective method of removing metals from the body once the source of exposure is identified and eliminated. By removing the mercury from the body, patients may have an improvement in memory and other health concerns.

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Dr. Marianne Marchese is the author of the bestselling book *8 Weeks to Women's Wellness* about the environmental links to women's health and how to detoxify from toxicants. She maintains private practice in Phoenix, Arizona, and is adjunct faculty at SCNM, teaching both environmental medicine and gynecology. She served on the State of Arizona Naturopathic Physicians Medical Board**,** National Association of Environmental Medicine, Arizona Naturopathic Medical Association, and Council on Naturopathic Medical Education. Dr. Marchese has helped formulate supplements for Priority One Vitamins. She lectures throughout the US and Canada on women's health, environmental, and integrative medicine topics.
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OCTOBER 10-11: FIELD CONTROL THERAPY (FCT) INTENSIVE TRAINING in White Plains, New York. CONTACT: 914-861-9161; https://www.yurkovsky.com/

OCTOBER 15-18: MISTLETOE & INTEGRATIVE ONCOLOGY COURSE in Denver, Colorado. CONTACT: https://anthroposophicmedicine.org/event-3678093

OCTOBER 16-18: FREQUENCY SPECIFIC MICROCURRENT CORE MODULE 1 – PAIN/INJURY MODULE in Anaheim, California. Also, **DECEMBER 6-8** in San Francisco, California. CONTACT: 360-695-7500; https://frequencyspecific.com/

OCTOBER 17: NEW YORK ASSOCIATION OF NATUROPATHIC PHYSICIANS ANNUAL CONFERENCE online. CONTACT: http://www.nyanp.org/conference/conference-registration/

OCTOBER 22-26: FREQUENCY SPECIFIC MICROCURRENT SEMINAR (CORE) in Taiwan. CONTACT: 360-695-7500; https://frequencyspecific.com/

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

OCTOBER 30-NOVEMBER 1: FREQUENCY SPECIFIC MICROCURRENT MASTER CLASS in Taiwan. CONTACT: 360-695-7500; https://frequencyspecific.com/

OCTOBER 31-NOVEMBER 1: ARIZONA NATUROPATHIC MEDICAL ASSOCIATION CONFERENCE – Neurological and Musculoskeletal Issues online. CONTACT: https://www.aznma.org/

NOVEMBER 6-7: NEW HAMPSHIRE ASSOCIATION FOR NATUROPATHIC DOCTORS (NHAND) 2020 ANNUAL CONFERENCE: Science, Spirit & Clinical Pearls. Virtual Online Event. CONTACT: conference@nhand.org; https://www.nhand.org/annual-conference/

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Pediatric Pearls

by Michelle Perro, MD

Autism: Neurodiverse or Neurodysfunction?

While lecturing at a recent conference on the role of pesticides and their relationship to autism spectrum disorder (ASD), I had the opportunity to have lunch with a young attendee with ASD. 'Ruby' attended a local college, where they (preferred pronoun) are receiving their master's degree in physics. They were involved in the neurodiversity community and had concerns regarding the viewpoint of ASD as a medical disorder and not as a neurovariant.[1] I have read about this line of thinking before, and noted experts in the field of autism, such as author and researcher Martha Herbert, MD, PhD, acknowledge both this position as well as describe ASD as a chronic, yet dynamic encephalopathy.[2]

Pause and reflect button activated: Honoring my New York-gone-California, old hippie vibe, I strive to embrace and support diverse and broad perspectives. But my inner editor and old observer of one's physical condition got me into a subconscious clinician mode. While they spoke, I noted the following characteristics: thin, sparse hair, thick lenses, pale facies, stooped shoulders, asymmetric skeletal structure, and low energetic state. Ruby described themselves as having "no health issues and needing no medications." (I've only too many times lamented that "Sick is the new normal," where kids with disorders such as asthma and allergies are so commonplace, they are viewed as customary.) The question pondered was not only regarding Ruby's state of health, but the overall health of children on the autism spectrum while still chewing on ASD as an adaptive common variant pathway of human functional brain development.

As ashamed as I was of my eyes drifting to her meal tray (I boldly claim I am not the food police even in my own family), I couldn't help notice Ruby's food choices; all carbohydrates, all processed. There was nothing remotely healthy on their plate. I inquired about their diet since they caught my gaze; and they felt that, as a scientist, GMOs were safe (quoting Cornell University's "Alliance for Science"),[3] and even though they were concerned about pesticides, they couldn't afford organic food on a student's budget. I censored myself from discussing anything about funding for the Alliance for Science and left my politics (briefly) at the door.

This was an excellent entry point into our conversation regarding glyphosate and autism, personalizing my just-delivered lecture to a virtual one-on-one experience. There have been several recent articles published that we discussed regarding the role of glyphosate, the main herbicide ingredient in Roundup and the development of neurocognitive disorders. While the many toxic effects of glyphosate have been written about extensively – such as its role as an antibiotic, metal chelator (both biologic and toxic metals), inhibitor of the cytochrome p450 detoxification pathway, role in vitamin A signaling, etc. – the direct brain effects by glyphosate have not been well elucidated, although epidemiological studies have linked glyphosate exposure in pregnancy and the development of ASD.[4]

In a recent combined Japanese/American university study, rats exposed to formulated glyphosate demonstrated behavioral abnormalities consistent with ASD. The pathway involved the metabolism of polyunsaturated fatty acids, which has been shown to be a factor via increased levels of soluble epoxide hydrolase.[5] (Soluble epoxide hydrolases and glutathione transferases are involved in the metabolism of xenobiotics.)[6] Additionally, alterations were demonstrated in the rat microbiota as well as changes in short chain fatty acids. The identification of an abnormal microbiota in children with ASD has been previously documented in the literature, for example, demonstrating increases in the firmicutes/bacteroidetes ratio, alterations in different microbial species, lowered levels of beneficial short chain fatty acids such as butyrate, as well as amplified levels of candida species and changes in the mycobiome.[7]

This conversation took an unexpected turn as Ruby began inquiring how can one improve brain function, which is where their own interest revolved. This idea of protecting the blood brain barrier integrity from chemical onslaught resonated with a young science-based physicist. In order to decrease brain barrier permeability, the first line of treatment is to decrease the toxic or allostatic load. When consuming a glyphosate-rich diet, phase I of liver detoxification is impaired since it is carried out by the cytochrome p450 enzyme system. Intermediaries

build up which can be more toxic than the original substance. Ruby did note but hadn't thought anything of the fact that they were highly sensitive to perfumes and odors and couldn't handle more than one cup of coffee: signs of an individual whose detoxification pathways might be imbalanced, also known as "pathological detoxifiers" (possibly linked to some individuals in politics).

Phase II consists of five main types of conjugation, including acetylation, acylation, glucuronidation, methylation and conjugation with sulfur. Glucuronidation is the step in the metabolism of many xenobiotics as well as bilirubin and food dyes. (Infants often have an underdeveloped glucuronidation pathway and hence can develop neonatal jaundice. Even more of a reason to feed babies organic only!) Getting into the scientific weeds with those with a left-sided brain dominance is a good strategy for attempting to lift "blocks to cure" (or miasms, for the homeopathically inclined). Ruby could see that just by an alteration in diet alone focusing on eating organic removes xenobiotics and decreases the allostatic load, hence improving liver detoxification and subsequent brain function. Cruciferous veggies can assist in both phases of liver detoxification and are number 1 in my prescription for health.

While ASD encompasses a complex and diverse array of physiologic disturbances and subsequent clinical manifestations, it is a treatable disorder (the younger the patient, ideally 6 years old and under, the better the outcome). According to the CDC, rates of ASD are reported to be at 1 in 54 children and 1 in 23 boys. Because of the shocking number of ASD children and now adults in the US, as well as individuals with developmental disabilities, it behooves all practitioners to understand the basics on how to treat them: "About 1 in 6 (17%) children aged 3–17 years were diagnosed with a developmental disability, as reported by parents, during a study period of 2009-2017. These included autism, attention-deficit/hyperactivity disorder, blindness, and cerebral palsy, among others."[8]

Pre-pregnancy clean-up for both parents is ideal six months to a year prior to conception. Prevention eclipses treatment and that mindset should now be part of our clinical practices. The following 'top ten' treatment overview recommendations (and by no means a complete list), are guidelines in how I approach children with ASD (or even showing early signs of autism without a "diagnosis"). There are a host of other treatments that are part of the treatment plan such as craniosacral osteopathy, occupational therapy (OT), physical therapy (PT), speech therapy, etc. In addition, there are other considerations to be factored in such as looking for further root causes when patients stall in their progress, such as chronic infections, PANDAS, EMF exposures, etc.

My Top Ten Steps in the Road to Reversing/Treating ASD

ORGANIC only. I spend time reviewing biosludge/biosolids, added as 'fertilizer' to crops (which may be even more toxic than the already present abundance of pesticides in conventionally grown food). Watching the movie '*Biosludged*' is a Saturday night must.[9]

Dietary review. Gluten and dairy are removed from my patients' diets, and they are prescribed a variety of paleo-type menus. Dietary changes are monitored and frequently adjusted. Families are encouraged to adopt the recommendations so that the child doesn't feel different, isolated, or the cause of family upset. Parents can hide goodies in their closets if they must. (My children always found my husband's stash.)

Water filters. Get the lead out! I recommend water filter systems based on families' wallets (https://www.ewg.org/tapwater/water-filter-guide.php).

Environmental over-haul. I refer patients to websites such as *MadeSafe* (https://www.madesafe.org/education/healthy-baby-guide/) for assistance. Decrease the allostatic toxic load!

Heal intestinal permeability. In the early days, I did a lot of food antibody testing. At this juncture, having seen so many children with leaky gut, I can make some

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educated guesses as to certain predictable findings such as elevated food antibodies and other markers of inflammation without testing. I can begin treatment empirically, saving the parents money on lab testing – which is not insignificant when we add in the costs of organics, supplements, and out-of-pocket treatments. (One of my families had 3 sons with ASD!) The child will appreciate and thank you for one less stick.

Rebalance the microbiome. There are general tendencies noted in the poop analysis (you know I love iPhone pics of their poop), which may include decreased microbial diversity, lower numbers of beneficial bacteria such as Lactobacilli and Bifidobacteria, and increased amounts of potential pathogens, such as clostridial species. I often will rotate probiotics to include targeted beneficial microbes (based on PCR analysis) and sporebiotics (https://microbiomelabs.com/home/products/megasporebiotic). Overgrowth of candida species is not uncommon. This is where we begin our education in fermentation and adding in probiotics.[10] Prebiotics should already be part of their fiber-enriched diets; but since so many ASD children are picky eaters, prebiotics (such as fructooligosaccharides) may need to be added. Low and slow is my motto to avoid occasional gassy tummies. No poop? No treatment. Pooping is the first priority! When gut function is solid (pun intended), I will go after candida with a combination of herbals such as *Mycoregen* (www.beyondbalanceinc.com) and a candida homeopathic series kit from Desbio if more intensive treatment is required (https://desbio.com).

Nutrient Replacement. Organics do not guarantee nutrient density, and vitamins and minerals are required for most children in general, particularly with neurocognitive dysfunction. Finding quality, tasty supplements can be a challenge! *Brainchild Nutritionals (*https://www.brainchildnutritionals.com*)* is one of the companies I use regularly. I start with vitamins and then add in the minerals, again, starting at a lower dose to assure their tummies can tolerate the supplement without digestive upset. I will get baseline nutrient levels when I can, including serum ferritin, RBC zinc and magnesium, B12, vitamin D and occasionally copper levels if I suspect an abnormal zinc/copper balance.[11] Insurance will often cover these lab tests.

Detoxification. There are many ways to detoxify, and we all have our favorites. I rarely begin this process until I get the gut working and rebalanced. My go-to's are homeopathics, and I usually prescribe *Pekana* liver, kidney and lymph drainage remedies that come as a kit. (https://www.bioresourceinc.com)

Neural pathway reconstruction. Fortification of neural function with improvements is hopefully occurring throughout the above process. By this point in time, the child is usually doing better, and we are noting significant health improvements. I like to bring in DHA and omega-3 supplements, as well as brain-crucial phosphatidylserine.[12] *Omega Focus Jr.* by *Nordic Naturals* contains DHA, omega 3s, phosphatidylserine plus DMAE. Giving combinations to a small child and making the parents' lives easier is a bonus.

Heavy metal detox. Big topic! I usually test for heavy metals both in a hair specimen from *Great Plains Lab* (www.greatplainslab.com) and occasionally with a heavy metal challenge and urine collection from *Doctor's Data* (www.doctorsdata.com). There are a variety of treatment methods using pharmaceuticals, herbals, homeopathics, and diet, and this is usually one of the last things I tackle in an ASD case. Because of the complexity of detoxification in children, I defer this treatment discussion until a future focus in the *Townsend Letter*!

Back to Ruby…

As we parted ways after lunch, we both agreed that our perspectives were broadened by amiable dialogue. While I support the fact that neurological differences in ASD should be acknowledged and respected, they are employed by self-advocates. My concerns reside with those individuals with impaired communication skills who are unable to become their own self-champions. Practitioners that care for children with ASD are still fighting conventional mainstream dogma that there is nothing to be done for ASD children. Indeed, despite many being 'neurodiverse,' a significant number of children with ASD have a host of medical issues that are treatable (and preventable). There is room to correct and prevent neurodysfunction in our neurodiverse population.

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Curmudgeon's Corner

by Jacob Schor, ND, FABNO
drjacobschor1@msn.com

Exposure to Fine Particulates Worsens Episodic Memory, A Marker of Alzheimer's Disease

In his 2015 novel, *The Buried Giant*, Kazuo Ishiguro tells the story of a civilization reminiscent of England during the Dark Ages. All the characters in the book suffer from worsening amnesia caused by mists, which we eventually come to learn are being generated by the breath of a dragon. Did that need a spoiler alert? Too late now.

I am reminded of this story by several recent papers on fine particulate air pollution, cognitive decline, and the risks of developing Alzheimer's disease. In recent years multiple studies have reported that fine particulate air pollution negatively impacts various aspects of health. By fine particulates, we mean the super tiny particles, those less than 2.5 micrograms in size. It turns out that these are the most harmful of all the tiny bits of stuff floating in the air. They are too small for the lung cilia to expel and so they end up absorbed into the bloodstream floating about and instigating inflammation and causing damage throughout the body.

The study that got me started on this Alzheimer's thing was conducted by Younan et al and was published just in November 2019.[1]

Knowing that a decline in episodic memory is a preclinical symptom of Alzheimer's disease, the authors compared memory function to fine particulate exposure. They asked whether exposure to fine particulates (PM2.5) was associated with decline in episodic memory and whether the structural changes seen in the brain after exposure are indicative of early disease. This research was conducted on 998 women ages 73 to 87 who were free of dementia, periodically giving them tests assessing learning and memory.

The women underwent annual episodic memory assessment using the California Verbal Learning Test, including measures of immediate free recall/new learning and delayed free recall (short- and long-delay). In addition to memory testing, magnetic resonance imaging (MRI) was used to detect brain atrophy, or wasting; and participant brain deterioration was scored on its degree of similarity to the atrophy characteristic of Alzheimer's disease. Environmental Protection Agency (EPA) data on air pollution at the women's home addresses was used to gauge fine particulate exposure for the three years prior to their first brain MRI. This allowed assessment of exposure to fine particulates (PM2.5) and association between memory decline and changes in brain structure. This was one large, comprehensive, and carefully done study.

The authors report that the greater the exposure to PM2.5, the greater the declines in immediate recall and new learning the women showed on testing. For each jump of 2.81 µg/m^3 in fine particulate exposure, the annual rate of decline accelerated by between 15% to 19.3%. Long-term exposure was associated with greater Alzheimer's disease pattern similarity scores. Let me say that simpler. The more bad stuff the women had breathed, the more their brains looked like they were developing Alzheimer's. Fine particulates caused neurotoxicity that contributes to early decline of the ability to recall recent things or learn new information. This fuzziness of thought was associated with atrophy of the brain's grey matter and an increased Alzheimer's disease risk down the road. We have our own mist-breathing dragon wiping our memories away in the real world.

This isn't new but it certainly sounds like bad news. The more research we see on fine particulate air pollution the worse it looks. Research published in June 2019 already suggested that long-term exposure to air pollution was associated with lower scores on tests of mental acuity. Shehab and Pope had measured fine particulate exposure in people who commute to work by traveling outdoors and demonstrated this was associated with significant declines in short-term cognitive function. Their results suggested that people living in the more polluted cities will have worse cognitive abilities on average than they might have if the air was cleaner.[2] Bottom line, from both the study by Younan et al and this earlier research, is that fine particulate exposures cause physiologic changes

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in the brain now linked to Alzheimer's disease development. Younan et al found that the greater the women's exposure to PM2.5, the lower their scores on the cognitive tests. The MRI results showed that increased exposure to fine particulates was associated with increased brain atrophy, even before clinical symptoms of dementia had appeared. The more pollution, the more their brains looked like they had Alzheimer's disease.[3,4]

In October 2019, a meta-analysis by Tsai et al concluded that fine particulate exposure is a potential determinant of Alzheimer's disease. Their data revealed that an exposure increase of 10 μg/m^3 in PM2.5 was significantly and positively associated with dementia, more than tripling risk (HR = 3.26, 95% CI: 1.20, 5.31).[5]

We ought to make a bigger fuss when our patients complain about poor memory or have a family history worrisome of neurodegeneration and directly inquire about air quality and the patient's exposure to fine particulates. We may not yet know if reducing particulate exposure will lead to improvement, but we can certainly assume that worsening exposure will worsen symptoms and doing nothing won't help.

The EPA considers the upper limit of "good quality" air to be 12 micrograms per cubic meter (μg/m^3). Here in Denver, our worst air quality day last year, reached 57 μg/m^3. Seattle's worst was 47 μg/m^3. San Francisco topped out at 200 μg/m^3. This is nothing though. On one day in November 2019, New Delhi's air reached over 900 μg/m^3, blasting past the EPA's definition of "hazardous" air (which maxes out at 500) and into unknown territory. Recall that in the study, a 2.8 μg/m^3 difference in PM2.5 exposure was associated with a nearly 20% acceleration in annual decline rate of cognitive function.

You can check your own outdoor air quality right now at this website administered by the EPA: https://www.airnow.gov.

Or generate a report on air quality exposure by location at https://www.epa.gov/outdoor-air-quality-data/air-quality-statistics-report.

In 2015 particulate pollution was responsible for an estimated 4.2 million deaths worldwide. The majority of these deaths were in Asia. Even though the United States has some of the cleanest air in the world, fine particulates are thought to have contributed to 88,000 premature deaths in 2015. This makes air pollution more deadly than diabetes. Air quality in the US had steadily improved for many years but, since 2016, air in the United States has worsened; fine particulates have increased on average by over 5%.[6] It has been suggested that this downturn resulted from lax enforcement of the Clean Air Act by the EPA.

The Environmental Protection Agency calculates that the reduction in fine particulates achieved in the past saved hundreds of thousands of lives.[7] These calculations come in part from a number of unique studies published over the years in which air quality changed dramatically in specific locations allowing the measurement of benefit and then extrapolation to larger populations. A December paper published in the *Annals of the American Thoracic Society* summarized some of these cases. What stands out in all of these studies is how rapidly the benefits were seen.[8]

Starting just a week after a ban on smoking in Ireland went into effect in March 2004, all-cause mortality in the country dropped by 13%. There was also a 26% reduction in ischemic heart disease, a 32% reduction in stroke, and a 38% reduction in COPD.[9]

In the United States, during the late 1980s when a steel mill in Utah closed for 13 months, hospitalizations for pneumonia, pleurisy, bronchitis, and asthma dropped by half. School absenteeism decreased by 40% and daily mortality fell by 16% for every 100 μg/m^3 PM10 (a larger sized pollutant) decrease.[10]

When the 1996 Olympic Games were held in Atlanta, Georgia, parts of the city were closed to traffic to relieve congestion so that athletes could arrive at their venues on time. This strategy also reduced air pollution in the city. In the following weeks, medical visits by children for asthma treatment dropped by more than 40% and trips to emergency departments dropped by 11%. Asthma hospitalizations decreased by 19%.[11]

Something similar occurred when China imposed factory and travel restrictions during the 2008 Beijing Olympics; lung function improved with a 46% reduction in asthma-related physician visits and less cardiovascular mortality.[12]

In Nigeria, when families were given clean ethanol cook stoves that reduced indoor air pollution, pregnancies ended with higher birthweights, greater gestational age at delivery, and less perinatal mortality.[13]

The Thoracic Society report also analyzed the cost of implementing the Clean Air Act. Twenty-five years after Richard Nixon signed the Clean Air Act into law, the health benefits exceed the cost of implementation by a ratio of 32:1 saving our country an estimated 2 trillion dollars. The Clean Air Act is one of the most effective public health policies ever enacted in the United States. Emissions of the major pollutants were reduced by 73% between 1990 and 2015 while the US gross domestic product grew by more than 250%.

Recent research such as this paper by Younan et al linking fine particulate air pollution to Alzheimer's disease suggests that the EPA's cost benefit analysis may actually be wrong. The cost of Alzheimer's disease and potential savings from reducing air pollution were not considered. Air pollution may cause more morbidity and mortality than previously thought and so may cost more than the EPA has estimated. Data have not taken into account the cost of decreasing cognitive function. This, of course, is a fancy way of saying getting dumber. The cost of people being dumber than they need be may eventually turn into the greatest of the dangers associated with fine particulates.

Update: This article was originally written in the autumn of 2019, before we had any inkling of the many changes the future had in store for us. The global response to the COVID-19 pandemic caused dramatic decreases in air pollution. A July 2020 report tells us that in regions of China, PM2.5 concentrations fell an average of 18.57 to 25.00 μg/m3 depending on time of day and weather conditions. The greatest decrease was in Hubei, with a 27 ug/m3 drop.[14] In Wuhan, PM2.5 levels dropped nearly 37%.[15] Particulate levels also

dropped in the US especially in urban areas though these drops varied by how complete a lockdown was instituted.[16]

Full analysis of how these shifts influenced morbidity and mortality have yet to be published. Yet we are seeing alarming hints. A paper from Francesca Dominici's team at Harvard looked at COVID-19 mortality rates and air pollution but as of this writing is still in preprint. Their team collected COVID-19 death counts from more than 3,000 counties in the United States (representing 98% of the population) up until April 22, 2020. When these rates were compared to long-term levels of PM2.5 exposure, their analysis revealed "…that an increase of only 1 µg/m3 in PM2.5 is associated with an 8% increase in the COVID-19 death rate.... The results were statistically significant and robust to secondary and sensitivity analyses."[17] These are sobering numbers if they prove to be accurate over time.

> Much more focus needs to be placed on recognizing the important role that common air pollutants hold in health, with commensurate actions being taken to reduce the levels of common air pollutants in the home—the one environment most people are in control of. It is quite possible that one of the most effective preventive medicine modalities would be the installation of a high-quality air purifier in the home.
>
> --Walter Crinnion, ND

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Curmudgeon's Corner

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Rheumatoid arthritis ($34 billion): Some of the drugs used to treat rheumatoid arthritis (e.g., Remicade, Humira, Enbrel) are extremely expensive, with costs ranging from $31,000 to $72,000 per year. In some cases, symptoms of rheumatoid arthritis can be controlled, without medication, by identifying and avoiding allergenic foods. A number of different supplements, including fish oil, borage oil, and ginger root, may improve symptoms in patients with rheumatoid arthritis.

Urinary diseases, including urinary tract infections ($86 billion): Interventions that may decrease the recurrence rate of urinary tract infections include increasing fluid intake and supplementing with a specific probiotic agent (a combination of *Lactobacillus rhamnosus* GR-1 and *L. reuteri* RC-14).

Nonmelanoma skin cancer ($22 billion): In patients at risk of developing basal cell or squamous cell carcinoma, supplementation with niacinamide (500 mg once or twice a day) decreased the incidence of these cancers by 23% in one study and by up to 86% in another study.

Migraine ($14 billion): Interventions that can decrease the frequency or severity of migraines include identification and avoidance of allergenic foods and supplementation with magnesium or riboflavin.

Otitis media ($10 billion): Identification and avoidance of allergenic foods can prevent recurrences of otitis media in as many as two-thirds of children who suffer from this condition.

Other "expensive" conditions for which dietary or lifestyle changes, nutritional supplements, or other low-cost interventions may be beneficial include diabetes ($111 billion), ischemic heart disease ($89 billion), depression ($68 billion), inflammatory bowel disease ($25 billion), osteoporosis, fibromyalgia, and irritable bowel syndrome (the annual cost for these last three conditions was not specified in the study).

There are two major obstacles to adopting these money-saving approaches to health and disease. First, many people are unwilling to make the dietary and lifestyle changes that are at the heart of controlling various chronic diseases. However, there are also millions of people who would gladly make the necessary changes if they had a healthcare professional to guide them. I saw countless patients over the years who, after experiencing improvement from nutritional therapies, told me they wished their doctor had told them about this approach long ago. Which brings us to the second obstacle: the mainstream medical community is not trained in and appears to have little interest in learning how to use interventions such as nutritional supplements and elimination diets. Fortunately, this is changing; a growing number of medical doctors are showing interest in integrative medicine. In addition, the number of naturopathic doctors and the number of chiropractors, dietitians, and other healthcare professionals who are learning about nutritional medicine continues to increase. We have great potential to become a healthier people and to free up massive amounts of money to be used for more productive purposes.

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Where Is the Money Going in Healthcare?

A recent study found that total spending on healthcare in the United States in the year 2016 was $3.1 trillion.[1] Healthcare spending increased from 13.3% of gross domestic product (GDP) in 1996 to 17.9% of GDP in 2016. The average amount of money spent on healthcare in 2016 was $9,655 per person. To give you a perspective on how much $3.1 trillion is, it is more than the GDP of every other country in the world except China, Japan, and Germany. Despite this massive amount of spending, we are not a particularly healthy country. An estimated 40% of Americans suffer from at least one chronic disease and, of those, more than half have multiple chronic illnesses.

When I was in medical school, one of the professors remarked that doctors do not cure disease. Rather, we just help people manage their illnesses. After hearing that remark, I thought to myself, "If that is true, then what am I doing here?" I never accepted the idea that we cannot cure (or at least partially reverse) chronic diseases. In my clinical practice that focused mainly on diet, lifestyle, and nutritional supplements, it was the norm rather than the exception to see people with chronic illnesses return to better health. Some patients who had been seeing several different specialists for several different conditions improved so much that they were essentially able to exit the medical system. If mainstream medicine would place more emphasis on approaches that help the chronically ill to get well, much of the money being spent on healthcare could be diverted to other socially useful endeavors. Below are some of the many examples of low-cost, low-risk interventions that are often beneficial for expensive chronic diseases. The annual cost of treating these conditions in the United States is listed in parentheses. The information below is based partly on published research and partly on clinical experience.

Hypertension ($79 billion): Diet; exercise; weight loss when necessary; and supplementation with nutrients such as magnesium, calcium, and vitamin C can often help control hypertension, thereby reducing the need for medications, decreasing the need for visits to the doctor, and decreasing the risk of developing heart disease or stroke.

Osteoarthritis ($80 billion): Symptoms of osteoarthritis often improve with supplements such as niacinamide, glucosamine sulfate, and chondroitin sulfate. These "natural" remedies may decrease the need for anti-inflammatory drugs, which can cause complications such as gastrointestinal bleeding and kidney disease. Moreover, in contrast to nonsteroidal anti-inflammatory drugs, which appear to accelerate the progression of osteoarthritis, there is evidence that these natural remedies can slow disease progression, and thereby decrease the need for joint-replacement surgery.

Heart failure ($33 billion): As many as 50% of people with chronic heart failure have iron deficiency. Identifying and treating iron deficiency has been reported to decrease the frequency of hospitalizations for worsening heart failure. Supplementation with coenzyme Q10 has also been found to decrease the need for hospitalization. In addition, a series of intravenous injections of magnesium (an inexpensive therapy) has been reported anecdotally to produce long-term improvement in patients with heart failure.

Anxiety disorders ($42 billion): A number of low-cost interventions are effective for some people who suffer from chronic anxiety. These include identifying and treating reactive hypoglycemia; identifying and avoiding hidden food allergens; avoiding caffeine; supplementing with magnesium or niacinamide; and doing aerobic exercise.

Asthma ($36 billion): A large proportion of the annual cost of treating asthma is related to hospitalizations and emergency department visits for acute asthma attacks. Low-cost interventions that can decrease the severity of asthma or reduce the number of acute attacks include identification and avoidance of hidden food allergens and supplementation with magnesium, vitamin B6, and vitamin C.

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