Guildford ME/CFS Support Group
(& West Surrey)

Newsletter

December 2013

Future dates

The following ME meetings are open to all members and carers.

8th January 2014 (Wednesday) 7.30pm The Boatman
Millbrook, Guildford, Surrey, GU1 3XJ

3rd February 2014 (Monday) 11am The Seahorse
The Street, Shalford, Guildford, GU4 8BU

27th February 2014 (Thursday) 7.30pm The Bridge Barn
The Bridge Barn, Bridge Barn Lane, Woking, Surrey, GU21 1NL

Directions from Guildford:
1. Head northwest on North St/A320 toward Chertsey St
2. Turn right onto Chertsey St/A320. Continue to follow A320. Go through 7 roundabouts
3. At the roundabout, take the 1st exit onto Wych Hill Ln
4. At the roundabout, take the 2nd exit onto Trigg's Ln
5. At the roundabout, take the 3rd exit onto Goldsworth Rd
6. At the roundabout, take the 1st exit onto Bridge Barn Ln

20th March 2014 (Thursday) 12 noon The White Hart
White Hart Lane, Wood Street Village, Guildford, Surrey, GU3 3DZ

Directions from Guildford - Join A323 towards Aldershot. Turn left at the roundabout just after The Rydes Hill Prep School, onto Broad Street, sign-posted to Wood Street. Drive through Wood Street until you get to the Village Green, then turn left immediately after the green and right onto White Hart Lane. The pub is 50 yards down on the right.

Annual General Meeting (AGM)

8th April 2014 (Tuesday) 7.30pm The Seahorse
The Street, Shalford, Guildford, GU4 8BU

All members and carers are welcome to the AGM. The committee members will provide a brief overview of the group's activity over the last year and discuss the group's future direction with those who attend the AGM.

Dr. Lapp on the cognitive problems in ME/CFS and what to do about them

Source: www.prohealth.com/me-cfs/library/showarticle.cfm?libid=18439
By Cort Johnson October 26, 2013

Dr. Lapp has been treating chronic fatigue syndrome and fibromyalgia patients for decades at the Hunter-Hopkins Center, and regularly participates in clinical trials. In his latest newsletter he addresses the cognitive problems found in ME/CFS, briefly reviews the scientific literature, and offers up some treatment ideas.

It's tough to lose your mental sharpness to a disease but all may not be lost. Dr. Lapp suggests some ways to combat the cognitive declines common in chronic fatigue syndrome. The research says the cognitive deficits in ME/CFS are relatively subtle compared to some other illnesses, but my guess is that a series of subtle deficits in information processing, short term memory, concentration, etc., can lead to pretty big cognitive and emotional hits.

Dr. Lapp notes cognitive problems are amongst the 'most functionally disabling and disturbing symptoms found in ME/CFS.

The easy distractibility, and flitting from half-completed task to half-completed task is something that I struggle with every day. It's perhaps not surprising that several studies suggest high rates of ADHD are present in both fibromyalgia and chronic fatigue syndrome.

Short-term memory problems and intrusive body sensations can make it difficult to follow conversations and understand the printed word. Of course, there's also the sheer mental fatigue; the inability to, at times, meaningfully incorporate, grasp or even understand outside events. It's as if a wall came down, and all one's mental energy is devoted to keeping one's body going.

The loss of spontaneity and richness that disappears from interactions, and the kind of blasted or half-self that's left over sometimes can be devastating emotionally. For me, when it gets that bad, all I can do is cultivate patience.

Documenting the brain drain
Dr. Lapp notes that neuropsychological tests can find deficits, but other tests can provide insights into the causes of the cognitive issues in ME/CFS. Brain MRI's find Unidentified Bright Objects (UBO's not UFO's) – small brain lesions – in up to 80% of ME/CFS patients, and they appear to be correlated with physical impairment. The amygdala may be one area of the brain damaged in ME/CFS.

Researchers have not known what to do with these small lesions. Because they tend to occur in different places in different people, and because healthy people can have them, it's not clear how important they are. However important or not important each specific lesion is, though, the fact that people with ME/CFS have significantly more small lesions than healthy people, in itself, suggests something significant has gone awry.)

SPECT scans often show reduced blood flow (i.e., reduced metabolism – reduced brain functioning) in the temporal lobes, amygdala, hippocampus and midbrain. PET scans show reduced metabolism in the brainstem, which controls many of the basic functions of the body (heart rate, breathing, sleeping.) Some brain volume appears to have been lost in the most severely ill – perhaps because of reduced physical activity) – and can be prevented or reversed by physical activity – if you can do it. Japanese studies indicate low levels of carnitine (acetyl-carnitine) are associated with reduced cognition.

Improving Cognition in Chronic Fatigue Syndrome and Fibromyalgia Supplements:

- Carnitine – Dr. Lapp confirms that carnitine (1000 mg/day) can help with thinking, concentration and memory. Acetyl-carnitine increases glutathione levels (reduced in brains of ME/CFS patients), increases brain blood flow (reduced in ME/CFS), ATP production, and acetylcholine activity.
- Gingko Biloba – can increase brain blood flows – again, a documented problem in ME/CFS.
- Phosphatidyl serine (PS) – brain PS levels are associated with normal nervous system signaling, glucose consumption and other factors important in brain functioning.
- B-12 - methylcobalamin 1000mcg to 5000mcg daily / methylfolate (400mcg daily)
- Procera AVH (acetyl-carnitine + vinpocetine+ huperzine) – Derived from the periwinkle flower, vinpocetine increases cerebral blood flows, and, according to Procera's manufacturers, has been shown to increase memory and brain processing speed – both of which are impaired in ME/CFS.
- Vinpocetine also is an anti-oxidant/neuro-protective agent, and can increase glucose metabolism (energy levels) in the brain. Derived from Chinese Club Moss, huperzine A (HUP) increases the levels of the neurotransmitter acetylcholine in the brain, and is an antioxidant.

Prescription drugs
Pharmaceutical companies are more and more interested, as our population ages, in finding drugs to enhance cognition.

B-Vitamins
Defects in folate and B12 metabolism are sometimes found in chronic fatigue syndrome, and some patients find B-12 protocols work very well. Both defects are thought to contribute to cognitive issues.

- B-12 - Deplin (15 mg of methylfolate), Metanx (3 mg methylfolate / 2 mg B12 / 35mg pyridoxil-5-phosphate), and Cerefolin NAC (methylfolate 5.6mg, B12 2mg, N-acetyl cysteine 600mg).

Brain enhancers
Pharmaceutical companies have been plumbing the 'brain enhancement' market more and more as our population ages, and studies reveal nutritional deficits in the brain. The 'medical foods' below target nutritional deficiencies associated with disease.

- Vayacog (omega-3 fatty acids 26mg + phosphatidyl serine 100mg) – this 'medical food' was created to repair lipid (fatty acid) imbalances that have been associated with memory problems.
- Axona (an alternative energy source that the brain can use instead of glucose, 40 gm twice daily) – FDA approved to treat Alzheimer's, Axona attempts to alleviate the effects of the reduced brain glucose levels associated with that disease. Axona metabolizes to ketone bodies which the brain can use when it's not using glucose. Axona comes in small packets and is mixed with liquids. Half the ME/CFS patients in one study demonstrated reduced cerebral glucose uptake in one part of their brain for another in one study, and 90% did in another earlier one.
- Prevagen (a jellyfish extract that affects calcium channels).

Drugs used in Alzheimer's
Dr. Lapp reported that galantamine (Razadyne), donezepil (Aricept), and memantine (Namenda) have all undergone brief trials in ME/CFS, but while some patients respond to them, most do not, and in general, unless dementia is present, he doesn't recommend them. Dr. Lapp has run the Hunter-Hopkins Center for the treatment of Chronic Fatigue Syndrome and Fibromyalgia for almost 20 years.

Mind-stimulation the old fashioned (and new-fashioned way)
Dr. Lapp recommends challenging your mind with crossword puzzles, word games, card or board games and computer games. (Like anything else – if you don't use it – you can lose it.) He also recommends mind-stimulating websites such as Lumosity whose authors, Dr. Lapp reports, have 'a special interest' in ME/CFS).

Not mentioned
Interestingly, Dr. Lapp did not recommend some of the more well-known cognitive enhancing drugs such as Ritalin (methylphenidate), Provigil (Modafinil) and Adderall. [Note: All of these are stimulants, which are known to cause rebound fatigue in ME/CFS patients]

- Carnosine - Carnosine supplementation in Gulf War Syndrome (GWS) increased one cognitive outcome substantially (and lead to a decrease in diarrhea as well)
- Yoga, Meditation/Mind/Body Techniques – have been shown to increase concentration and improve cognition as well.

About the Author: Cort Johnson has had ME/CFS for over 30 years. The founder of Phoenix Rising and Health Rising, Cort has contributed hundreds of blogs on chronic fatigue syndrome, fibromyalgia and their allied disorders over the past 10 years. Find more of Cort's and other bloggers' work at Health Rising

Drug under development spells hope for pain relief in fibromyalgia

Source: www.prohealth.com/library/showarticle.cfm?libid=18427
By Cort Johnson October 20, 2013

From opioids to anticonvulsants to antidepressants, etc. doctors throw a wide variety of drugs at nerve pain, yet the prognosis is generally poor, with 40-60% of patients receiving only partial relief. (Some studies indicate alpha lipoic acid and benfotiamine (thiamine) can be helpful for some, as well.)

Nerve pain comes in many shades and can produce burning, tingling, numbness, shooting, stabbing, allodynia, etc. Usually associated with central sensitization (increased pain sensitivity), inflammation in the brain/spinal column appears to play a significant role but few drugs are effective at reducing inflammation there.

Lyrica's incredible success, in spite of issues with side effects and efficacy, highlights the great need for better means of dealing with neuropathic pain. Increasing restrictions on opioid use makes the development of more effective means of pain relief imperative.

Probably the most intriguing [new development] is a compound called neuroprotectin D1 (NPD1) – the subject of increasingly intense investigation. NPD1 has been mostly investigated as a protective agent in central nervous system, eye and kidney disorders but a recent study suggested it may be effective against the hardest to treat pain of all – nerve pain.

Enter a potentially cheap drug derived from a fatty acid often used in chronic fatigue syndrome and fibromyalgia.

A 'Good' Fatty Acid derivative
"These compounds are derived from omega-3 fatty acids found in fish oil, but are 1,000 times more potent than their precursors in reducing inflammation," Ru Rong Ji

Derived from DHA, an omega-3 fatty acid found in fish oils, NPD1 has neuroprotective properties.

In contrast to omega-6 fatty acids, which have pro-inflammatory effects, omega-3 fatty acids have anti-inflammatory effects. Studies have not generally borne out their efficacy in ME/CFS but they are commonly recommended and used. With NPD1 clocking in at about 1,000 times the potency of its precursor, DHA, NPDI – if it ever gets to market – will be like fish oil on speed.

NPD1 is potentially much more than a pain reliever; indeed, pain is only the latest symptom NPD1 is being thrown at. An aptly named drug, neuroprotectin D1 is produced in response to a variety of conditions, some of which occur in chronic fatigue syndrome and fibromyalgia, including oxidative stress (high in ME/CFS/FM), protein misfolding (perhaps occurring in ME/CFS), seizures and brain ischemia-reperfusion (conjectured to occur in ME/CFS/FM).

Reduced NPD1 levels may be a factor in Alzheimer's, and just last month the Michael J. Fox Foundation awarded a NPD1 grant in hopes the compound will slow neuron loss in rodent models of Parkinson's.

Just this month, NPD1's effectiveness in reducing pain in mice was assessed.

Mouse study produces results
"Notably…. treatment, started a few hours after the nerve trauma, eliminated neuropathic pain."

What they did to these mice was not pretty and some people may want to skip this part. First they induced long-lasting allodynia in the mice by surgically damaging their sciatic nerve. They found that applying NPD1 prevented the allodynia from occurring. Far fewer of the mice given NPD1 chewed their toes (sometimes off) as they usually do when suffering from neuropathic pain, and the mice that did chew their paws, chewed them less.

Dorsal Horn of the Spinal Cord
A key neuropathic pain-regulating area, the dorsal horn of the spinal cord, was examined less. The dorsal horn refers to a horn of the spinal cord that received sensory information from the body via the dorsal ganglia.

Long-term potentiation and microglial activation sets the stage for increased pain signal production in the dorsal horn but neither were found in the NPD1 treated mice (while both were found in the untreated mice.) Levels of a key cytokine involved in invoking neuropathic pain, IL-1B, was reduced in the NPD1 treated mice, as well. Finally, the macrophage infiltration that is a trademark of neuroinflammation did not occur.

NPD1, then, was very effective at reducing or eliminating three important aspects of neuropathic pain and central sensitization.

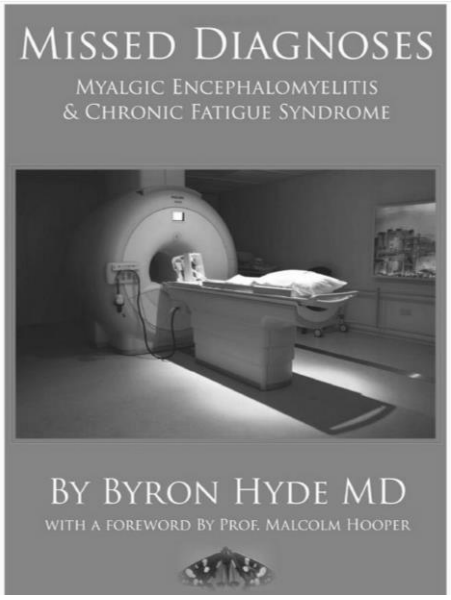
DHA is not enough – The pure stuff is needed
Even large doses of the precursor to NPD1 – DHA – failed to elicit significant pain reduction. A common neuropathic pain reliever, gabapentin, did reduce some of the allodynia but only at very high doses.

NPD1 appears to be able to stop the production of immune factors that attract the proinflammatory macrophages that tweak the nerves more. NPD1 also reduces neuron firing – a important factor in an over-active system.

Keep your eyes on NPD1
Duke researchers said they hoped to start a clinical trial to measure NPD1's effects on pain.

NPD1 is also being studied in animal models in Parkinson's and Alzheimer's. It will take time, but given the amount of research NPD1's received in the last nine years (seven studies in this year alone), it's a good bet that NPD1 will show up in drug form at some point, possibly bringing a new and, according to these researchers, a quite safe approach to neuropathic pain and neuroinflammation.

Book: Missed diagnoses ME/CFS by Byron Hyde MD



Source of the overview below:
www.hfme.org/booksbest.htm#420114714

This book is essential reading for doctors and patients alike, and those interested in M.E. as well as those interested in 'CFS' or that have been misdiagnosed as 'CFS.'

(As this book explains, there is no such distinct disease as 'CFS' - and every diagnosis of 'CFS' is a Misdiagnosis. This book also explains that M.E. is not a similar disease to 'CFS' nor a mere 'fatiguing disorder.')

The Nightingale Definition of Myalgic Encephalomyelitis paper in particular cannot be recommended highly enough.

Finally this is a modern and TESTABLE definition of Myalgic Encephalomyelitis, created by the world's leading and most experienced M.E. expert, Dr Byron Hyde. This is NOT a redefinition of CFS but is instead a pure M.E. definition.

It draws on the long history of M.E., collates the evidence from each of the world's leading M.E. experts (past and present) and combines this with details of the most modern medical tests. This definition also rightly gives no importance at all to the bogus notion of mere `fatigue' having any importance in the diagnosis/definition - unlike each of the `CFS' definitions, including unfortunately the Canadian `ME/CFS' definition which just mixes in a few M.E. facts with what is still primarily a `CFS' redefinition.

Dr Hyde explains that:

"I believe it essential to define clearly Myalgic Encephalomyelitis, returning the definition to its clinical and historic roots and complementing this information with the certitude of modern scientific testing. That is what the Nightingale definition of M.E. sets out to do. But let me first ask you a very important question.

What is the purpose of any medical definition? What is the purpose of any disease definition if it is not to allow the physician to rapidly and accurately diagnose a specific illness in order to attempt to effectively treat the patient before the illness becomes chronic or to call in the appropriate specialists? Our definition solves this problem."

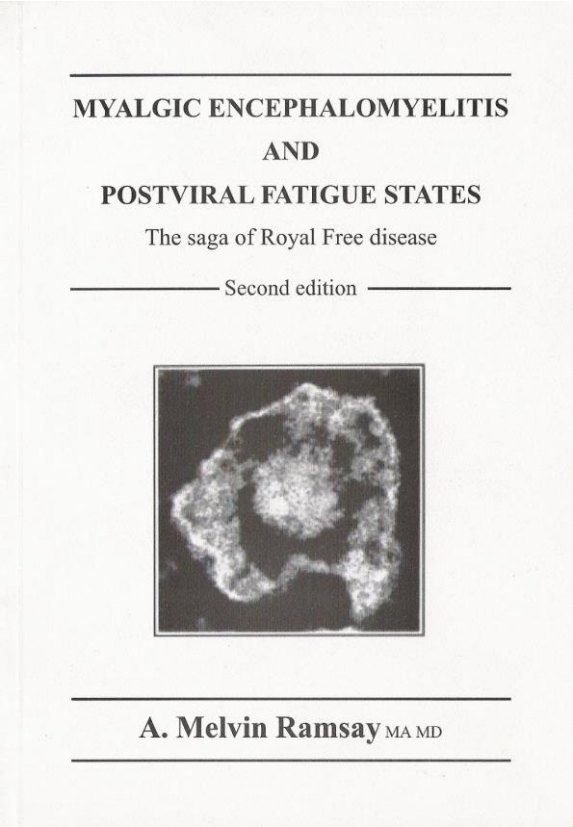
"There is a third purpose for any disease definition. That is to clearly define the disease so that various physicians and researchers can clearly understand that they are talking about the same illness spectrum and so launch research into what will become an effective treatment. Our definition gives a clear baseline for investigation.

The Nightingale definition is based upon the following two criteria: (a) The excellent scientific and clinical work of respected physicians and scientists who investigated the various M.E. epidemics. (b) The results of modern scientific testing techniques and the knowledge accruing from examining thousands of M.E. patients using these and more historical techniques. The proposed M.E. definition is designed to improve early diagnosis and treatment for the tens of thousands of patients stricken with M.E. It is not a new definition of CFS nor should it be conceived as a rewording of any previous CFS definition.

Available from www.amazon.co.uk for £9.99
Or from the following website address for £12.99:
www.lulu.com/gb/en/shop/byron-hyde-md/missed-diagnoses-myalgic-encephalomyelitis-chronic-fatigue-syndrome-second-edition/paperback/product-18463888.html

Book: ME and postviral fatigue states - The saga of royal free disease by A. Melvin Ramsay

Source of the overview below:
www.hfme.org/wramsay.htm



This book was published in the 1980's and summarises Ramsays three decades of work on Myalgic Encephalomyelitis.The book explains the difference between M.E. and post-viral fatigue states and that they do not represent the same illness.

Dr Byron Hyde MD writes:

Myalgic Encephalomyelitis (M. E.) This is a term used to describe an epidemic and sporadic disease process that is associated with a chronic debilitating illness of children and adults. Variants of this term M.E. were first used following a series of repeating epidemics starting in May 1955 in the Royal Free Hospital in London England. New outbreaks of this illness continued until 1958 in various London area hospitals. M.E. and these epidemics are well described by A. Melvin Ramsay in his book Myalgic Encephalomyelitis and Post-Viral Fatigue States.

Secondhand copies of the book may be available through www.amazon.uk and the book has also recently been republished by The M.E. Association in the UK.

The book is available from the MEA for £6.00. (all profits to MEA Ramsay Research Fund)

They write: Since 1990, The ME Association has been active in raising money for scientific research and during 1999, The ME Association's research fund was renamed in honour of our founding President, the late Dr Melvin Ramsay. Dr Ramsay worked tirelessly to obtain recognition for ME/CFS and his spirited determination to help people with ME/CFS has been a guiding light to many who follow in his footsteps.

(However, it is important to be aware that the MEA has been widely criticised of now actually working against everything Ramsay stood for with regards to M.E.: From the BMJ: [Some] accuse the association of drifting away from the purpose of founder members such as Dr Melvin Ramsay, who first proposed myalgic encephalomyelitis as a discrete physiological condition. Instead, they say, the association has come to accept a blurring of the distinction between ME and chronic fatigue syndrome and has adopted some of the arguments of that section of the medical establishment that believes the condition to be a somatisation disorder. As the BMJ went to press, Louie Ramsay, daughter of the late Dr Melvin Ramsay, announced that she was to resign as a patron of the association with immediate effect. She has also asked that all reference to the Ramsay family name be removed from the Ramsay Research Fund. This text is available: http://bmj.bmjjournals.com/cgi/content/full/326/7401/1232-c)

Six common misconceptions about the chronically ill

(What those who are healthy rarely understand about those who are sick or in pain)

Source: www.psychologytoday.com/blog/turning-straw-gold/201312/six-common-misconceptions-about-the-chronically-ill

by Toni Bernhard, J.D. December 5, 2013

More often than not, chronic illness and chronic pain go hand-in-hand, so when I use the term "chronically ill," I'm including people who are in chronic pain. My hope is that it won't be long until these common misconceptions become uncommon ones, as people become educated about what life is like for those who suffer from chronic illness (130 million in the U.S. alone).

Misconception 1: If people look fine, they must feel fine

Whether healthy or sick, it's good for most people's morale to try and look nice when they go out. I go out so seldom that I make an effort to look my best when I do. Sometimes I feel like a young child again, playing dress-up. That said, I always hope that if I see people I know, they'll remember that looks can be deceiving.

I've had people say to me, "You look great." I know they're trying to be nice, so I make an effort to respond graciously (with something other than, "Well I don't feel great," spoken in an irritated tone of voice). But the truth is...there I am, "looking great," while my body is pulsating with flu-like symptoms, my muscles are aching, and my heart is pounding so hard that sometimes it feels as if it must be visible to others on the outside of my body!

When people see someone whom they know is struggling with his or her health, I hope they'll remember that they have days when they leave the house looking great but feeling terrible, perhaps from a bad night's sleep or from lingering symptoms of an acute illness. If they understood that this is how most chronically ill people feel all the time, this common misconception would be well on its way to becoming an uncommon one.

Misconception 2: If people's illness or pain were truly physically based, their mental state wouldn't affect their symptoms

If you're not sick or in pain, I invite you to try this simple two-part exercise, so you can test this misconception out for yourself.

> **Part one**
> The next time you feel under stress — maybe you're angry at someone or worried about something — stop; close your eyes; and pay attention to how your body feels. Can you feel that your muscles have tightened? In addition, your heart may be beating faster and your whole body may be pulsating. You may even have broken out in a sweat. These are just some of the ways that mental stress manifests in the body of a healthy person.
>
> **Part two**
> Keeping that stressful mental state in the forefront of your awareness, now imagine that you suffer from chronic pain and/or illness. What would happen? Your body would respond to the mental stress the same way it did for you as a healthy person. But now, that response would be in addition to your chronic, everyday symptoms. And if those symptoms happen to overlap with the physical symptoms that accompany mental stress — tightened muscles, racing heart, pulsating body and maybe even sweating — you can see how a person's mental state can easily exacerbate the physical symptoms of chronic illness.

This is why keeping mental stress to a minimum is so important for the chronically ill. It's important, but often impossible. Why? Because we live in the same stressful world that healthy people live in.

Misconception 3. Preparing for an event by engaging in "radical rest" will assure that when the occasion arrives, the chronically ill will be in better shape than had they not rested
I can "radically rest" for several days in a row before a commitment (I've had some events for my new book that I've been doing this for) and yet, on the day of the event, feel terribly sick. Resting may increase the odds that I'll be less sick than usual on the day of the event, but it's no guarantee.

When my granddaughter, Cam, turned six in September, I asked my husband take me to her birthday party for a short time since it's only an hour's drive away. It would have been a treat to watch her interacting with her friends (something I rarely get to see) and to meet their parents. I rested for four days before the event. But that morning, I called my son in tears to tell him that I was too sick to attend.

This misconception can lead to serious misunderstandings. For example, a week later, I was able to attend an event for my book. This could make it appear that I was choosing the book event over my granddaughter's birthday party, but I was not (and thankfully my son understood this).

The truth is that the same amount of resting before each of the two events simply did not yield the same results. That's the unpredictability of living day-to-day with chronic pain and illness. Not only can it be a source of disappointment and sadness, but if we don't treat ourselves kindly and with compassion, it can lead to self-recrimination and be a source of terrible guilt.

Misconception 4: If chronically ill people are enjoying themselves, they must feel okay
When an important occasion arises, people who are chronically ill have learned to put up with the symptoms of illness, including terrible pain, so they can try to enjoy what they're doing, especially the enriching experience of being in the company of others. Please don't assume that a person who is laughing is a person who is pain-free, ache-free, or otherwise feeling good physically.

Misconception 5: Stress reduction techniques, such as mindfulness meditation, are a cure for chronic pain and illness
Stress reduction techniques can be effective tools to help with symptom relief and to help cope with the mental stress of ongoing pain and illness. However, unless a person suffers from a distinct disorder called somatization (in which mental or emotional problems manifest as physical symptoms), stress reduction techniques are not a cure.

Misconception 6: Being home all day is a dream lifestyle
This misconception arises because, when healthy people entertain this thought, they're not contemplating being home all day feeling sick and in pain! Put another way, would they say: "I wish I could be home all day with pain that no medicine can relieve"; or "I wish I could be home all day with flu-like symptoms that keep me from being able to read a book"? I doubt it.

My heartfelt wish is that people will become educated about what life is like for the chronically ill so that, some day soon, we can say that these are six uncommon misconceptions.

Thank you for reading my work. My most recent book is titled How to Wake Up: A Buddhist-Inspired Guide to Navigating Joy and Sorrow.

I'm also the author of the award-winning How to Be Sick: A Buddhist-Inspired Guide for the Chronically Ill and their Caregivers.

ME/CFS and MS show similar Neuroimmune characteristics

Source: www.prohealth.com/me-cfs/library/showarticle.cfm?libid=18498
Original Source: BMC Med. 2013 Sep 17;11:205. doi: 10.1186/1741-7015-11-205. Morris G, Maes M.

Editor's comment
For decades, researchers have noted not just an overlap of symptoms between MS and ME/CFS - e.g. disabling fatigue, exercise intolerance, and cognitive impairment, among others - but similiarities in brain scans (both patients with MS and ME/CFS show bright spots on MRIs). The major difference between the two illnesses is obvious demyelination, as well as the progressive nature of MS. Nonetheless, the authors conclude that "the strong similarities between both disorders in terms of phenomenological, neurobehaviour and neuroimmune characteristics further underscore that ME/CFS belongs to the spectrum of neuroimmune disorders. In addition, the data show that the comorbidity between both disorders and the high prevalence of ME/CFS symptoms in patients with MS may be explained by neuroimmune mechanisms."

Myalgic encephalomyelitis/chronic fatigue syndrome and encephalomyelitis disseminata/multiple sclerosis show remarkable levels of similarity in phenomenology and neuroimmune characteristics.

Background
'Encephalomyelitis disseminata' (multiple sclerosis) and myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) are both classified as diseases of the central nervous system by the World Health Organization. This review aims to compare the phenomenological and neuroimmune characteristics of MS with those of ME/CFS.

Discussion
There are remarkable phenomenological and neuroimmune overlaps between both disorders. Patients with ME/CFS and MS both experience severe levels of disabling fatigue and a worsening of symptoms following exercise and resort to energy conservation strategies in an attempt to meet the energy demands of day-to-day living. Debilitating autonomic symptoms, diminished cardiac responses to exercise, orthostatic intolerance and postural hypotension are experienced by patients with both illnesses.

Both disorders show a relapsing-remitting or progressive course, while infections and psychosocial stress play a large part in worsening of fatigue symptoms. Activated immunoinflammatory, oxidative and nitrosative (O+NS) pathways and autoimmunity occur in both illnesses. The consequences of O+NS damage to self-epitopes is evidenced by the almost bewildering and almost identical array of autoantibodies formed against damaged epitopes seen in both illnesses.

Mitochondrial dysfunctions, including lowered levels of ATP, decreased phosphocreatine synthesis and impaired oxidative phosphorylation, are heavily involved in the pathophysiology of both MS and ME/CFS. The findings produced by neuroimaging techniques are quite similar in both illnesses and show decreased cerebral blood flow, atrophy, gray matter reduction, white matter hyperintensities, increased cerebral lactate and choline signaling and lowered acetyl-aspartate levels.

Summary
This review shows that there are neuroimmune similarities between MS and ME/CFS. This further substantiates the view that ME/CFS is a neuroimmune illness and that patients with MS are immunologically primed to develop symptoms of ME/CFS.

ME/CFS, MS and the nervous system

The following is part of an article about the Central Nervous System, ME and MS. As such, it complements the comparison of ME and MS that was discussed in the previous article of this newsletter.

Source: http://phoenixrising.me/archives/20115

There is a general consensus among ME/CFS researchers that the symptoms seem to reflect an ongoing immune response, perhaps due to viral infection. Thus, most ME/CFS research has focused upon trying to uncover that putative immune system dysfunction or specific pathogenic agent. However, no single causative agent has been found. In this speculative article, I describe a new hypothesis for the etiology of ME/CFS: infection of the vagus nerve. When immune cells of otherwise healthy individuals detect any peripheral infection, they release pro-inflammatory cytokines. Chemoreceptors of the sensory vagus nerve detect these localized pro-inflammatory cytokines, and send a signal to the brain to initiate sickness behaviour.

Quite recently, there have also been a number of articles appearing discussing the similarities between ME/CFS and Multiple Sclerosis (MS), although this is no recent occurrence, as the similarities have been noted many times previously to this. As the majority likely know, MS is caused through an autoimmune response targeted towards the myelin sheath secreting schwann cells, that wrap around some neurons. This allows for the previously discussed saltatory conduction, hence allowing for faster nerve impulse transmission. The destruction of these schwann cells, therefore, has far reaching consequences within the nervous system, causing the distressing symptoms that MS patients suffer. The relative morbidity between ME/CFS and MS is clear to see, given that both have profound adverse effects upon the quality of life. However unlike MS, ME/CFS has yet to receive irrefutable evidence regarding the pathophysiology of the ongoing disease process. For this reason, it is helpful in some respects to compare the two diseases. However it is also important to contrast the two, highlighting the differences between the diseases.

For instance, in MS, there is clear evidence of ongoing inflammatory mechanisms within the nervous system. However, despite this being reported in several ME/CFS studies, it is in no way conclusive. It appears that if an autoimmune mechanism truly lies at the heart of ME/CFS, that it functions in a more complex, subtle and devious way than MS appears to. Although it is of note that despite MS being recognized as an autoimmune disease, the actual understanding of the complex ongoing process still eludes researchers. One important difference that I as a personal observer have come to, is that while MS typically adversely affects the central nervous system, ME/CFS appears to have consequences in both the sensory nervous system (with sensory overload being a common complaint) and autonomic nervous system. Unfortunately, dysfunction of the central system appears somewhat easier to casually observe, whereas autonomic and sensory dysfunction appear somewhat more convoluted. Perhaps this is why ME/CFS is only now losing the incorrect label of a psychological condition.

There are remarkable phenomenological and neuroimmune overlaps between both disorders. Patients with ME/CFS and MS both experience severe levels of disabling fatigue, a worsening of symptoms following exercise, and resort to energy conservation strategies in an attempt to meet the energy demands of day-to-day living.

Given the research discussed here and the plethora of historical research into autonomic dysfunction within ME/CFS, it is clear that dysfunction and perhaps even mechanical damage to the nervous system, specifically the autonomic and sensory systems, plays a central role within the ongoing disease process within ME/CFS patients. An interesting point to make is that many of the previous articles centre on areas that have direct interaction with these neurological systems. Any abnormality in one can have profound effects upon another – with the cardiovascular, nervous and immune systems all being closely intertwined and interdependent upon one another. Of all the areas within biology and medicine, the nervous system is likely the most complex and is therefore a fast advancing field. Hopefully, as time progresses and ME/CFS research continues, it won't take too long to discover that one key piece of evidence that points us towards the cause of ME/CFS, and hopefully leads to treatments in the future.

An author escapes from CFS

Source: http://well.blogs.nytimes.com/2011/02/04/an-author-escapes-from-chronic-fatigue-syndrome/?_r=1
By Tara Parker-Pope

Laura Hillenbrand, the best-selling author of “Seabiscuit: An American Legend,” is known for her exuberant storytelling and dynamic characters. Her newest book, “Unbroken: A World War II Story of Survival, Resilience and Redemption,” is a riveting tale of the life of an athlete and war hero, Louis Zamperini.
Ms. Hillenbrand’s ability to transport her readers to another time and place is all the more remarkable in light of the fact that she is largely homebound, debilitated by chronic fatigue syndrome, or C.F.S.

The illness, a devastating and little understood disorder, is characterized by overwhelming fatigue and various nonspecific symptoms like muscle pain, memory problems, sore throat, swollen lymph nodes, achy joints and unrefreshing sleep. I recently spoke with Ms. Hillenbrand about her latest book and why she is speaking out about the challenges of life with C.F.S. Here’s our conversation.

Q. Why have you started talking about your illness?

A. I had never been public about my illness at all before “Seabiscuit.” I didn’t want to talk about it very much because I had the experience of being dismissed and ridiculed. People don’t understand this illness, and the name is so misleading. I realized I had this opportunity because I was going to be getting press attention for the book. I’m going to talk about it because I can. Maybe that will save the next person from going through what I did.

Q. Do you think it’s hard for people to understand how debilitating chronic fatigue can be?

A. This is why I talk about it. You can’t look at me and say I’m lazy or that this is someone who wants to avoid working. The average person who has this disease, before they got it, we were not lazy people; it’s very typical that people were Type A and hard, hard workers. I was that kind of person. I was working my tail off in college and loving it. It’s exasperating because of the name, which is condescending and so grossly misleading. Fatigue is what we experience, but it is what a match is to an atomic bomb.

This disease leaves people bedridden. I’ve gone through phases where I couldn’t roll over in bed. I couldn’t speak. To have it called “fatigue” is a gross misnomer. Most people, when they hear the disease name, it’s all they know about it. It sounds so mild. When I first was sick, for the first 10 years or so, I was dismissed. I was ridiculed and told I was lazy. It was a joke.

Q. When did you learn you had chronic fatigue syndrome?

A. I got it when I was 19, and I was diagnosed at 20 by the head of infectious disease at Johns Hopkins. It was the most hellish year of my life. I went from doctor to doctor. I got very thin and lost 22 pounds in a month. One doctor thought I was anorexic and lectured me about it. After my appointment he followed me to the bathroom and put his ear to the door. When my doctor at Johns Hopkins finally said, “You have a real disease,” that was an important moment for me.

Q. What were your first symptoms?

A. As it does in most people, it had a very sudden onset. I was an athlete and had always been healthy. I was riding in a car on my way back from spring break my sophomore year of college and felt very nauseated. I guessed it was food poisoning. I woke up a few days later, and I literally could not sit up, I was so weak. It hit me that fast. I had to drop out of school because I couldn’t make the walk to the classes.

Q. What happened once you left school?

A. I was bedridden the first two years. I was having fever all the time and huge lymph nodes; the reddest, rawest, terrible sore throat; typical sweats and chills like the flu, but it didn’t go away, month after month. I had the most extreme exhaustion and balance problems, strange cognitive things, trouble concentrating. I couldn’t read analog clocks anymore. I’d try to say one word and a different word would come up. I had brain fog that was terrible in 1987 and 1988, and then it started to slowly get better.

I did better until 1991, when I tried to take a road trip to Saratoga. I had a catastrophic C.F.S. crash, went into shock, and went back down to the bottom to worse than ever. Then vertigo started, and ever since the room appears to be moving around me. I feel like I'm moving all the time.

Q. How are you now?

A. I'm housebound now. I had a relapse while I was working on the book in 2007. I got weaker than I've ever been. I've been too weak to leave the house for two years. I only leave the house about once a month. I'm just not very strong. A lot of days I don't get down the stairs. It's a slow process to recovery. The book publicity is quite difficult for me. I'm not able to do that much of it. It's taking a whole lot out of me.

Q. It's hard for me to imagine how you could have done the research and writing for two books during this time. How did you do it?

A. It's a trade-off for me. While it's really hard to do, at the same time, I'm escaping my body, which I really want to do. I'm living someone else's life. I get very intensely into the story, into the interviews and the research. I'm experiencing things along with my subjects. I have a freedom I don't have in my physical life.

Writing is a godsend to me that way. Without it I wouldn't have anything. I am completely still almost all the time. A lot of time I don't leave the upstairs. What I have is the story I'm working on. It's a wonderful thing for me to get out of my body for a while.

Q. Do you think having C.F.S. influences your writing?

A. Because my life is so silent and so still, I think I'm able to get deeper into what I'm working on. My mind is willing to get out of here and go into there. It becomes such an intense experience.

Q. Did you always want to be a writer?

A. At the time I got sick, I wanted to be a history professor. I was 8 years old when I went across the street from my house to a fair, and they always had a used book sale. For a quarter I bought a book called "Come On Seabiscuit." I loved that book. It stayed with me all those years. I was sick and housebound and looking for something I could write about. I wrote an article. I was partway through it and realized there was a huge untold story.

Q. How did you do the reporting for the book?

A. Lots and lots of interviews, at least a hundred, and going through newspaper archives. The family of Seabiscuit's owner sent me 30 enormous leather scrapbooks. I bought so many things on eBay — vintage things, magazines. I did many interviews with very, very old men.

Q. Who helps you manage your life?

A. I'm married. I have a wonderful husband. The house is all set up for me. There is a refrigerator upstairs. My desk has everything I need. Toaster, utensil and bowls, teapot — everything I need. I got married in 2006. We've been together since before I got sick. He's my college sweetheart. We waited to get married until I got reasonably well. I was too sick to go to the reception. I was just at the wedding for a few minutes. He has been through this with me. Some couples it would drive apart; it has drawn us together. We have a deep understanding. He doesn't see me as a sick person. He sees me as everything else I am. It's a really wonderful relationship. We had to learn how to do it. It's not easy at all to be a couple with a disease.

Q. Why did you decide to write about Louis Zamperini?

A. Seabiscuit led me to him. My subject was one of the greatest runners in the world in the 1930s, likely to break the four-minute mile. Seabiscuit was famous at the same time. All the newspapers that covered Seabiscuit also covered Louis. I kept reading about him. When I got done with Seabiscuit, I wrote him a letter and called him, and he told me his life story. I had to write this book.

It's the most amazing survival story. Louis was an Olympic runner who hung up his shoes and became a bombardier. He crashed in the Pacific and floated on a raft for 47 days. Sharks jumped on board to pull him off. He was attacked by a Japanese bomber. He nearly starved to death. He went through a typhoon and was captured. His captors experimented on him, enslaved him, and he was a prisoner for two years. The things that happened to him, and his defiance — it's an amazing story.

Q. It sounds like on some level you could relate to him.

A. I think because of what I'm dealing with, I'm really interested in people who become trapped in extremity and have to rely on their character to pull them out of it. I'm fascinated by the struggle, and the attributes that enable people to survive these things. I want to look to them for inspiration. I think that's why I'm drawn to it.

Louis has told me he felt I was someone who was easy to open up to because he knows I've suffered. With someone else, I think he might have been a little more taciturn. But he felt, "She gets it. She's been to this place herself."

Having to go all the way to the bottom of yourself to find the resources to survive: this is something I understand well. I understand desperation. It's an emotion I have dealt with a thousand times in the last 24 years. You feel like you don't know where you're going to get the strength to go on. We've been to the same place in different circumstances. I'm not comparing myself to a prisoner of war, but there are common emotions that enable me to identify with him.

Q. Do you think your writing would be different if you didn't have this illness?

A. I don't remember what it's like to feel well. I'm 43. I was 19 when I got sick. It's a lifetime ago. It's hard for me to imagine what I would have been as a writer without the history I have now. We're all sitting in our particular circumstances and writing from that place.

Q. Your personal story is so compelling. Have you thought about writing something autobiographical?

A. My husband wants me to. I just don't know that I want to do that. I have to spend so much time being vigilant on my body and worrying about my body and suffering. So much of my own autobiography would be about my health, and I don't know if I want to spend my professional life thinking about that. I write to escape my circumstances.

Body willing, and if I can find a subject that compels me, I'll keep writing. It's a great way to touch the world, because I'm not in this world. I went out recently to the CVS drugstore for the first time, and they had these new checkout things with no person at the checkout counter. I was baffled by this. Writing is my way of communicating with the world, and I don't have any other way to do it, so I want to keep doing it.
