Guildford ME/CFS Support Group
(& West Surrey)

Newsletter

September 2018

Drawing by CFS sufferer Shantel Palmer

Future dates

Open to all members and carers

10th October 2018 (Wednesday) 7.30pm The Weyside
Millbrook, Guildford, Surrey, GU1 3XJ
http://www.theweyside.co.uk
(Parking at Millbrook car park £1 for evening)

5th November 2018 (Monday) 11.15am The Seahorse
The Street, Shalford, Guildford, GU4 8BU
www.theseahorseguildford.co.uk

3rd December 2018 (Monday) 7.30pm The Weyside
Millbrook, Guildford, Surrey, GU1 3XJ
http://www.theweyside.co.uk
(Parking at Millbrook car park £1 for evening)

NHS to update classification system to reflect that M.E. is neurological disease

Source: www.meassociation.org.uk/2018/08/nhs-to-update-classification-system-to-reflect-that-m-e-is-neurological-disease-06-august-2018

6th August 2018

It was confirmed last week by DX Revision Watch, that the recording of M.E. and CFS as previous examples of a 'multisystem disorder' will now be replaced in SNOMED CT by the more appropriate, 'disorder of the nervous system'.

This follows the welcome efforts of advocate Suzy Chapman, who writes the authoritative blog, DX Revision Watch, and Sonya Chowdhury, on behalf of Forward ME and Action for M.E.

- Chronic fatigue syndrome classified under Neurological disorder in SNOMED CT International Edition
- GP system updated to reflect M.E. as neurological

SNOMED CT is a comprehensive electronic clinical classification system used by the NHS that records known diagnoses and symptoms with the aim of making clinical information consistent across healthcare settings.

In some respects it would seem to be more relevant to UK residents than the World Health Organisation international classification system, as electronic NHS patient records should be updated to reflect the change.

While chronic fatigue syndrome unfortunately remains the parent term in this clinical vocabulary, M.E. is recognised as a synonym along with other recognised terms.

The change will take place in the UK in October, and, as SNOMED CT is also used internationally, it will also be reflected in the United States, Canada, New Zealand, Australia and other European countries.

It is too soon to say what practical effect, if any, this might have on patient relations and treatment within the NHS, but it at least means that ME/CFS will now be listed under a more appropriate heading.

Dr Charles Shepherd, Hon. Medical Adviser, ME Association:

> "This is a subject that the Countess of Mar and the Forward ME group of charities have been closely following for some time. So, it is good news to see that M.E. will now be classified as a neurological disorder in the SNOMED CT system – a position that appears consistent with WHO classification of M.E."
>
> "As our summary states, it is difficult to know what practical effect this will have in the consulting room – because doctors are not normally following, or reading about, changes to the SNOMED classification system."
>
> "But it will be helpful when we still have to challenge a media or medical profession statement that ME/CFS is a mental health condition. It may also be helpful for people with ME/CFS when they are having to challenge a faulty benefit, insurance or legal decision which is again being based on an inaccurate mental health classification of M.E."

5 tips for keeping your motivation for self-care high

Source: www.prohealth.com/me-cfs/library/5-tips-keeping-motivation-self-care-high-85248
By Julie Holliday

We all want to be as well as we can be, but even when we feel highly motivated to reach as high a level of functioning as possible, that doesn't always translate to being highly motivated to great self-care. We want the results but can get impatient with what it takes to get them. Especially when you factor in the dreaded R word – rest. The fact that its very nature involves lack of doing makes it feel as though it's not doing anything useful; it doesn't seem like an active step to getting results. Even when repeated experience points out that not doing it is a recipe for disaster!

With illnesses like ME/CFS, Fibromyalgia and Lyme disease, it can sometimes seem as though all your efforts at self-care just don't seem to be making a difference (or at least not enough of a difference). Keeping motivation to keep it going can be really challenging when the results seem so slow to come. It can be so easy to get disheartened when we don't perceive success. Recently I've discovered a very simple secret to keeping motivation high: detach the idea of success from the outcome of better functioning, and link it to the fact that you actually did the self-care. For me it all comes down to the tick box chart (check box). I feel successful when I see the ticks piling up, I give myself the message that I am achieving something important and it is that sense of achievement – that sense of succeeding in something – that keeps my motivation high. Experience has also shown me that in general, a box full of ticks does correlate with feeling better. I've learned that without the disappointment of aiming for a particular outcome and not seeing it fast enough, I actually make better progress. However, I try not to fixate on that; it works a lot better when I keep the focus just on being successful at doing the self-care.

5 tips to boosting motivation with your tick box chart

1. Start by including all the things you already do pretty consistently for your self-care.
Include just one or two things that you want to improve on and do more consistently.
The whole point of the tick box is to help you feel good about your self-care, so set it up so that you will see lots of ticks and so that it encourages you to get even more.

2. Anchor filling out your tick box to regular events in your day.
Before or after meal times is a good bet, then you get 3 opportunities to remind yourself of anything you haven't done yet.

3. Set achievable weekly targets and offer yourself a reward.
If you have 6 things in your tick box, 4 of which you are already pretty regular with and two that you are aiming to do better with, that's a potential of 42 tick in a week. I'd probably set a target of about 30 ticks for the first week (depending just how regular my first 4 are). Pick your reward in advance and make sure you give it to yourself if you do achieve your target.

4. Only ever add one new thing to the tick box at a time.
When you start your tick box there might be lots of aspects of your self-care that you want to improve, but keep focused. Decide on one aspect that is most likely to make a difference for you and work on getting it established before adding anything else. When I'm just trying to tighten up on things that I already do but not very consistently, I will add something every week or two if I'm getting results. If I'm aiming to introduce something new, I'll give it several weeks before I add something else.

5. Share your success, find someone to celebrate with.
Identify somebody who you can hold yourself accountable with, someone encouraging who understands the importance of good self-care. Tell them whether you've reached you tick target or not. Being open with what you're doing somehow makes it more valuable. Celebrating your victories is also really important in terms of keeping motivation high and much more enjoyable when its shared with someone who also recognises its importance.

My holistic self-care tick box

As a holistic life coach, self-care means a lot more to me than just taking care of myself physically. I have two categories on my tick box – one for things that I know my body needs to optimise conditions for healing and one for things that boost me spiritually and add to my happiness. I know that they are all connected and need to be in balance. I have them in separated so that I can see if I'm paying too much attention to just one side of the equation, then I know what I need to pay most attention to the following week to balance things up.

I have been using the tick box system for a long time now so my list is quite long, but here's what it looks like this week:

Optimising conditions for healing physically	Mon	Tues	Wed	Thus	Fri	Sat	Sun
Tai chi	√	√	√				
20mins+ meditation am	√	√	√				
15 hr intermittent fast	√	√	√				
Dancing in my break			√				
Yoga pm	√	√	√				
Stimulation free rest	√		√				
Drinking lots of water	√	√	√				
Walk	√	√	√				
Energy exercises	√						
Energy testing food	√	√	√				
Spiritual/mindset/happiness							
Affirmations	√	√	√				
Visioning	√	√	√				
My daily commitment	√	√	√				
Connecting with nature	√	√	√				
Meditation pm	√						
Journaling			√				
Playfulness and trust	√	√	√				
Being love	√	√					
Gratitude	√		√				
Pleasure	√	√	√				

My target is an average of 15 ticks a day: 105 ticks in the week, and my reward will be buying a new trashy novel to keep me entertained during the part of my afternoon rest that isn't stimulation free.

What will you put in your tick box chart?

Major research group highlights inflammation energy production connection in ME/CFS

Source: http://simmaronresearch.com/2018/06/inflammation-energy-production-chronic-fatigue-syndrome-me-cfs

30th June 2018

> "We propose that chronic low-grade inflammation induces and/or maintains persistent fatigue by inducing an imbalance between cellular-energy availability and cellular- and behavioural energy expenditure." Lacourt et al. 2018

Inflammation, the brain and energy metabolism – it's like the trifecta in chronic fatigue syndrome (ME/CFS) research. It seems like virtually everyone in the ME/CFS field believes that all three are involved but that belief only carries so much weight in a small field. What this field really needs is buy-in from outside researchers who can help move it forward.

That appears to have happened recently when a major research group lead by Robert Dantzer penned a review paper proposing that low-grade inflammation is causing energy production problems in chronic fatigue syndrome (ME/CFS) and probably many other diseases. The authors didn't shy away from the chronic fatigue syndrome (ME/CFS) connection. In fact, they lead their review paper off with it, placing the fatigue in ME/CFS in the same context as the fatigue in cancer, MS, rheumatoid arthritis and others.

The study was published in the Frontiers in Neuroscience journal series which is touted as the 1st most cited series in the Neurosciences journal field.

The Dantzer group's involvement in the intersection between inflammation and energy production is welcome but not entirely surprising; it's a logical outcome of their past work. Dantzer spearheaded the now accepted idea that the immune system produces the symptoms of "sickness behaviour" (fatigue, headache, muscle aches, sore throat, etc.) that occur during an infection which serve to reduce our energy usage and to keep us isolated from others (they posit to prevent pathogen spread).

What's new is his group's focus on the energy production process itself – a focus, interestingly, made possible largely by the work of ME/CFS researchers. The piece, with lead author Tamara LaCourt, shows how low-grade inflammation can cause the same energy problems we're seeing in ME/CFS: a metabolic switch from energy-efficient, oxygen-based energy production process to a fast-acting, inefficient glycolysis-based approach.

Immune cells aren't like other cells; jumping into action causes them to rev their motors up tremendously, placing enormous stress on their energy production systems. As they do this, they switch from a focus on aerobic energy metabolism to what the authors call "aerobic glycolysis" in order to churn out energy more quickly. That process results in less mitochondrial energy production and the increased production of toxic by-products like lactate. Plus, over time this process results in reduced nutrient availability and less energy for the rest of the body.

Several studies from the Solve ME/CFS Initiative are examining whether the energy production of immune cells in ME/CFS is up to the task.

Prolonged inflammation also tends to result in two other energy production problems: increased insulin resistance and reduced glucose tolerance. Reduced glucose tolerance smacks glucose uptake by immune cells at the very time that they're clamoring for it, causing the body to break down fats and proteins, thus removing resources it would ordinarily use elsewhere. In yet another whack at the energy production, inflammation increases reactive oxygen species production which can hammer mitochondrial energy production.

The authors believe that neurons – which rely on glycolytic processes in astrocytes to get their energy – may be hit hardest by chronic inflammation. This is because insulin resistance – a common outcome of chronic inflammation – destroys the glycolytic process in astrocytes, causing neurons to get their energy from fats – a slower and less efficient process.

Miller's work on ME/CFS suggests that problems with the basal ganglia – the dopamine-producing centre of the brain – may be causing problems with movement, reward and fatigue in ME/CFS. That's a particularly interesting finding given that dopaminergic neurons in the brain are particularly vulnerable to inflammation. Shungu's studies, which have consistently found high lactate and low gluthathione levels in the ventricles of ME/CFS patients brains, suggest that high levels of oxidative stress could be causing inflammation in the brain itself.

Plus, even low-level inflammation can disrupt a key element in ME/CFS and FM – sleep – which, in turn, increases fatigue. Simply altering one's circadian rhythm (i.e. one's sleep times) can have significant metabolic effects, leading to increased glucose levels and decreased insulin sensitivity. The effects don't end with sleep; sleep deprivation results in the need for increased energy expenditures the next day.

Then add in the extra ten percent in extra energy needs that chronic low-level inflammation imposes on the body – and the potential for a dramatic drop in energy production rises. (We'll find out more about total energy production in ME/CFS during the metabolic chamber tests in the NIH's intramural study).

The authors believe that impaired energy production represents a "final common pathway" in persistent fatigue.

Leader in the Field

> "In sum, most evidence for an association between fatigue and mitochondrial functioning comes from CFS, indicating lower levels of antioxidants and possible reductions in mitochondrial ATP production." The authors.

We understandably don't think of researchers in the small ME/CFS research field as being pioneers in the medical research field at large, but some have ploughed brand new ground. Suzanne Vernon's computational biology work at the CDC was so novel that an entire issue of the Pharmacogenomics journal was devoted to it. Gordon Broderick and Travis Craddock's expansion of that work at Dr. Klimas's Institute of NeuroImmune Medicine has taken computational biology further – much further – in ME/CFS than in any other field. Ron Davis and Mark Davis at Stanford are using new HLA gene typing and T-cell technologies to try and nail down what is activating ME/CFS patients' immune systems.

ME/CFS researchers' attempts to understand the intersection between mitochondrial problems and fatigue are clearly breaking new ground as well. According to the authors of this review article, 21 of the 25 studies examining the intersection between mitochondrial problems and fatigue have been produced by ME/CFS researchers. Researchers we all know (e.g. Naviaux, Montoya, Hornig and Lipkin, Fluge and Mella) were cited again and again in the overview.

The authors even cited Workwell's groundbreaking 2013 study which indicated that a shift to glycolytic energy production occurred during the second day of a two day exercise test in ME/CFS. They also singled out the 2017 Tomas study which found that under conditions of cellular stress, the mitochondria in ME/CFS patients' cells were unable to rise to the occasion.

Turning to the metabolomics studies, the authors cited three ME/CFS studies which have pointed to "reduced metabolic activity". They believe the metabolic changes seen in ME/CFS reflect a chronic over-reliance and eventual depletion and abandonment of lipid metabolism, which results in a greater use of carbohydrate stores; hence the greater reliance on glycolysis and impaired aerobic energy production.

In short, the authors believe the metabolomic studies in ME/CFS are demonstrating the same metabolic shift that the authors propose occur in states of chronic low-grade inflammation.

Interestingly, the authors proposed that many ME/CFS patients are probably exceeding their daily energy stores. That, of course, makes perfect sense given Staci Stevens's and Workwell's findings that, for some patients, simply sitting upright puts them into an aerobic energy deficit.

For all its possible connections, the idea that fatigue in ME/CFS is simply the result of "low-grade inflammation" seems untenable given the disability present – unless that inflammation is found in the brain. The Simmaron Research Foundation is bringing the brain, the immune system and metabolism together in a way that's never been seen before in ME/CFS.

The Simmaron Research Foundation's first ME/CFS cerebral spinal fluid study suggested that an immune dysregulation, the likes of which approached that found in multiple sclerosis, may be present in the ME/CFS patients' central nervous systems. Their second outlined an atypical ME/CFS subset. Their current CSF (cerebrospinal fluid) study – an expanded version of the first study which includes a metabolomic component – will be the first to potentially merge immune and metabolic findings in the most energetically active part of the body – the brain.

Plus, stay tuned for a report suggesting that inflammation is not just present, but pervasive, in ME/CFS patients' brains.

MPs demand more biomedical research for cruel 'death sentence' disease

Politicians said that controversial psychotherapy and exercise therapies recommended by the NHS after a flawed medical trial must stop NOW

Source: /www.prohealth.com/me-cfs/library/mps-demand-biomedical-research-cruel-death-sentence-disease-83211

Press Release: ME Association, June 21, 2018. Less than £1 is spent each year on people suffering from the devastating invisible illness M.E. (myalgic encephalomyelitis), a condition which leaves tens of thousands bedbound, housebound and unable to work.
Parliament heard in a landmark three-hour debate of the chronic lack of funding for medical research and how many doctors still don't know how to diagnose or manage the condition.

End to controversial therapies and to stigma
Politicians said that controversial psychotherapy and exercise therapies recommended by the NHS after a flawed medical trial must stop NOW – because they are making patients worse. And they called for an end to the stigma and myths surrounding M.E., which at worst, leaves sufferers to endure a tortuous existence.

Westminster Hall was told how people with M.E. are six times more likely to commit suicide.
Carol Monaghan (SNP Glasgow North West) told how ME costs the UK £3.3bn per year.
She said: "Despite the number of people affected and the devastating effect of the disease on sufferers and their families, it is very much a hidden illness, which is characterised by some as 'yuppie flu' and misunderstood by doctors, the public and politicians alike."

M.E. tragedies
Westminster Hall heard of the tragic case of 21-year-old Merryn Crofts, who last month had M.E. listed as the cause of her death at inquest, and how ME Association fundraising manager, Helen Hyland, broke the news of her husband's suicide to her children.

Yet Ms Monaghan added: “Some people consider M.E. to be a psychological condition, despite the fact that people with M.E. are not allowed to be blood or organ donors.”

“Unfortunately, those who hold such beliefs often are in influential positions and have a blinkered view of the condition.”

“I wonder what they have to fear from proper biomedical research into M.E. If such research showed they were correct, their views would be vindicated. However, if it threw up new information that had an impact on M.E. treatment and care, as medical professionals they should surely support that.”

The discredited PACE trial

Ms Monaghan drew particular attention to the PACE trial results in 2011, that examined graded exercise therapy (GET) and cognitive behaviour therapy (CBT).
The researchers claimed the results demonstrated both treatments were ‘moderately’ effective and led to recovery in over a fifth of patients.

But the trial has since faced intense criticism, and not only from patients in the UK. Clinicians, researchers, as well as charities, like the ME Association, have all expressed concern about how the results were obtained, analysed and presented.

Parliament has previously heard claims that the PACE trial data was deliberately flawed to “remove people from long-term benefits and reduce the welfare bill”. The PACE trial endorsement of GET and CBT helped form the basis of the NICE clinical guideline – which is now being reviewed.

PACE has had a wide-reaching influence

Ms Monaghan said one of the key authors behind the PACE trial, Professor Michael Sharpe, admitted that some involved in the trial had worked for insurance companies.
She said: “The PACE trial, which recommended CBT and GET, influences how health insurers and the DWP make their decisions.

“Insurance companies refuse to pay out unless a programme of GET has been undertaken, and many people who apply for benefits are told that they must carry out GET—or, indeed, that they appear well enough to work.

“PACE is unique in UK medical history, in that it was part-funded by the DWP.

“The links of some of it’s main authors to health insurance companies are troubling. One of those authors, Professor Michael Sharpe, states in his briefing for the debate:

“Several of the investigators had done small amounts of independent consultancy for insurance companies, but this was not relevant to the trial. The insurance companies played no part in the trial.”

“I will leave hon. Members to make up their own minds about that.”

Westminster Hall was told how the U.S. Center for Disease Control (CDC) and the Dutch Health Council have both abandoned GET as a treatment.

Ms Monaghan added: “If those countries acknowledge the flaws of GET, why are ME sufferers in the UK having to fight so hard for similar acknowledgement? The ME community hopes that GET will not feature in the NICE guidelines for ME treatment after they are revised.”
‘Unbecoming’ behaviour?

“Interestingly, Professor Sharpe, one of the authors of the PACE trial whom I already mentioned, emailed me this week and told me that my behaviour is “unbecoming of an MP”.

“I say to Professor Sharpe that if listening to my constituents, investigating their concerns and taking action as a result is ‘unbecoming’, I stand guilty.”

“If Members of Parliament are not willing to stand up for the most vulnerable in society, what hope do any of us have?”

Ministerial response
Minister for Health and Social Care, Steve Brine, was asked how the Department for Health is supporting training for medical practitioners on ME care and treatment, and asked if he would support proper funding for medical research into the diagnosis and treatment of ME.
Mr Brine said the government invests £1.7bn each year into health research.

And stated that the National Institute for Health Research and the Medical Research Council would welcome “high-quality” research into “all aspects of ME… to make a scientific breakthrough”.

He described the need to find a breakthrough as a “matter of good Christian humanity” and promised to help increase the awareness of M.E. with GPs.

Read the full Ministerial response and Ms. Monaghan’s closing remarks HERE.

He reiterated that patients with M.E. symptoms should be referred to NHS specialist services – within six months for mild symptoms, three months for moderate symptoms and immediately for severe symptoms.

But Mr Brine admitted that access to services remained “a big and ongoing issue” and that the configuration of services was down to local commissioning groups. He added that all schools must have arrangements in place to support, with flexibility, children with ME.

NICE guideline review
On the NICE guidelines, he said it was a “jolly good job” the position on M.E. is being updated but said it would be “wrong for ministers to interfere” with the process.
On benefits, Mr Brine said the “DWP recognises that ME is a real and disabling condition” and that every patient must be assessed on an individual basis.

He added: “When assessing claimants, healthcare professionals are expected to be mindful of the fact that many illnesses—including ME—produce symptoms that vary in intensity over time, and they are instructed not to base their opinion solely on the situation observed at the assessment.

“The DWP assures me that all healthcare professionals are required to read an evidence-based protocol on ME as part of their training, as well as engaging in a programme of continuing medical education that includes modules on the condition.”

‘A completely unacceptable situation’
The ME Association campaigns to make the UK a better place for people with M.E. A spokesman said: “A three-hour parliamentary debate on M.E. is not before time. We are grateful to Carol Monaghan for securing the debate, to those MPs who took part, and to ME Association members for engaging with their parliamentary representatives ahead of the debate.

“Despite being recognised by the World Health Organisation as a neurological disease – and an earlier report to the Chief Medical Officer calling for more research and a network of hospital-based clinics – many doctors still don’t know how to diagnose and manage M.E. and lack of biomedical research means that we still don’t have any effective forms of treatment.

“This is a completely unacceptable situation for a disease that is twice as common as multiple sclerosis and where a new report has estimated that M.E. is costing the UK economy billions in lost taxes, and through healthcare and benefit expenditure.”

Widespread neuroinflammation found in ME/CFS

Source:
www.healthrising.org/blog/2018/09/24/brain-fire-neuroinflammation-found-chronic-fatigue-syndrome-me-cfs

The following is a slightly abbreviated version of the full article found at the source above

Neuroinflammation – The Japanese Way
Researchers have thought for decades that neuroinflammation is probably present in chronic fatigue syndrome (ME/CFS), but it's only recently that the technology has been able to pick up the lower levels of neuroinflammation believed present in diseases like ME/CFS and fibromyalgia. The Japanese were the first to take a crack at it.

They have long believed that inflammation produces central fatigue (fatigue emanating from the brain), which plays a major role in ME/CFS. In 2013, Watanabe proposed that inflammation in the brain was whacking the "facilitation system" which pops up when we are fatigued to boost signals from the motor cortex to keep our muscles moving. He also hypothesized that an inhibition system was turning up the fatigue in ME/CFS.

A 2016 study rounded the circle when it found evidence of reduced dopaminergic activity from a part of the brain (the basal ganglia) which activates the motor cortex. That fit in just fine with Miller's results, which suggested that problems with the basal ganglia could be producing both the fatigue and the motor activity problems in ME/CFS.

The big breakthrough came in 2014 when the Japanese startled just about everyone with a PET scan study which found widespread neuroinflammation in the brains of ME/CFS patients. The study was small (n=19) but the findings appeared strong.

The neuroinflammation was widespread but was highest in the areas of the brain (thalamus, amygdala, midbrain, hippocampus) that had shown up in ME/CFS before. Plus, the Japanese were able to link specific regions of inflammation to specific symptoms. Inflammation in the thalamus was associated with cognitive impairment, fatigue and pain; inflammation in the amygdala was associated with cognitive issues; and inflammation of the hippocampus was associated with depression.

Anthony Komaroff called the findings the most exciting in decades. The Japanese began a much larger (n=120) neuroinflammation study. This year they published a large number of papers on ME/CFS in the Japanese Journal, "Shinkei Kenkyu No Shinpo" (Brain and Nerve). One of the papers was specifically on neuroinflammation but the findings have not yet been published in English journals.

Neuroinflammation – The Younger Way
Jarred Younger – who runs the Neuroinflammation, Pain and Fatigue Lab at the University of Alabama at Birmingham has also long believed that neuroinflammation plays a major role in chronic fatigue syndrome (ME/CFS) and fibromyalgia (FM).

Microglia immune cells (the immune system of the brain) are sensitive to so many factors and can be triggered in so many ways that virtually any stressor, from an infection to toxins to psychological stress, can potentially trigger a state of microglial sensitisation in the right individual. With their ability to produce dozens of different inflammatory mediators, Younger believes that the difference between ME/CFS and FM could simply come down to small differences in how the microglia are tweaked.

Both diseases could be triggered by high rates of immune activation which, over time, sensitizes the microglia to such an extent that they start pumping out inflammatory factors at the first sign of a stressor.

Younger hypothesised that because inflammation produces temperature increases, he could try and create a heat map of the brain. Looking through the literature, he realized that thermometry was already being used in the brain to assess stroke and cancer patients. It turns out that the brain's attempts to repair the damage from stroke and cancer results in huge temperature increases. The stroke and cancer researchers, though, were just focused on small areas of the brain.

Because Younger didn't know exactly where in the brain to search in ME/CFS, that technique wouldn't work for him. He had to develop a method that would produce a heat map and a chemical signature **of the entire brain**, and found a Florida researcher who developed a way to do that, called magnetic resonance spectroscopic thermometry (MRSt), using an MRI scanner.

With this technique, it takes just 20 minutes in the machine to get an entire 3-D heat and chemistry map of an ME/CFS patient's brain. After The Solve ME/CFS Initiative (SMCI) provided funding, he got to work and ultimately scanned the brains of 15 ME/CFS women and 15 age and sex matched healthy controls.

It turned out that Younger's brain-wide search technique was right on. Looking at single areas of the brain in ME/CFS patients would have produced misleading data. It turned out there was no single area or even a group of areas in the brain that were abnormal in ME/CFS: most of the brain was.

Younger found lactate – a product of anaerobic metabolism – widely distributed across the brains of people with ME/CFS. He opened a chart showing an amazing array of lactate-engorged brain regions. He picked out a few: the insula, hippocampus, thalamus, and putamen, which had particularly high levels. They were virtually the same regions the Japanese had found in their 2015 study. The fact that the temperature increases overlapped with the lactate increases provided further confidence that Younger had identified some key areas.

The interior cingulate cortex, in particular, which Younger called "the seat of suffering" in the brain, showed up in spades. It's associated with a lot of nasty symptoms (malaise, fatigue and pain) and it's shown up in both ME/CFS and fibromyalgia studies in the past. The high choline signal in that region of the brain suggested that inflammation there was producing a pattern of destruction and replacement; i.e. quite a bit of damage – even possibly neuronal damage – was happening there.

Overall, the lactate levels weren't as high as in other diseases – they were just consistently present. Younger didn't expect to see really high levels; really high lactate levels would have meant irretrievably damaged neurons – the kind of neuronal damage seen in M.S., Parkinson's and Alzheimer's – the kind of neuronal damage that is really hard to reverse. The fact that Younger saw inflammation in ME/CFS but not neuron-destroying inflammation is good news indeed for people with ME/CFS.

It's possible that some damage such as neuronal reprogramming and synaptic pruning could be occurring, but determining that would take an autopsy. (Some groups are collecting ME/CFS brains at a couple of autopsies that have been done.)

Remarkably, the healthy controls didn't show evidence of a single analyte such as lactate being elevated or a single area of the brain being heated up. It's highly unusual to find zero evidence of an abnormality in the healthy controls. Usually the results of studies apply to groups, not individuals; some healthy controls typically will have findings that are similar to the MEC/CFS patients and vice-versa, but not here – the two groups were absolutely distinct. Even though this was a small study, such black/white results strongly suggest that neuroinflammation of the brain is a key element of ME/CFS.