Guildford ME/CFS Support Group
(& West Surrey)

Newsletter

June 2014

Future dates

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

7th July 2014 (Monday) 12 noon The White Hart
White Hart Lane, Wood Street Village, Guildford, Surrey, GU3 3DZ
www.thewhitehartpub.com

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.

14th August 2014 (Thursday) 7.30pm The Weyside
Millbrook, Guildford, Surrey, GU1 3XJ
www.theweyside.co.uk

Over the last few years the Weyside was called the Boatman.

10th September 2014 (Wednesday) 11am The Seahorse
The Street, Shalford, Guildford, GU4 8BU
www.theseahorseguildford.co.uk

7th October 2014 (Tuesday) 7.30pm White Lyon & Dragon
Perry Hill, Worplesdon, Guildford, GU3 3RE
www.thaipubs.co.uk/location/white-lyon-dragon

Just along from the Worplesdon Place Hotel, The White Lyon and Dragon is a Thai restaurant in Worplesdon that has both a bar/food section and restaurant. Tuesday night is discounted making the quality food very reasonably priced. Of course, you can simply turn up to have a soft-drink or tea.

Dr Myhill's new book

"Diagnosis & Treatment of Chronic Fatigue Syndrome" is an easy to read self-help book based on years of clinical experience and supported by evidence-based research. Written in a clear, logical and concise way, the book is full of practical, comprehensive, step-by-step solutions for patients, and is equally a 'must read' for doctors and other health professionals as it provides a detailed treatment solution with an evidence-based approach.

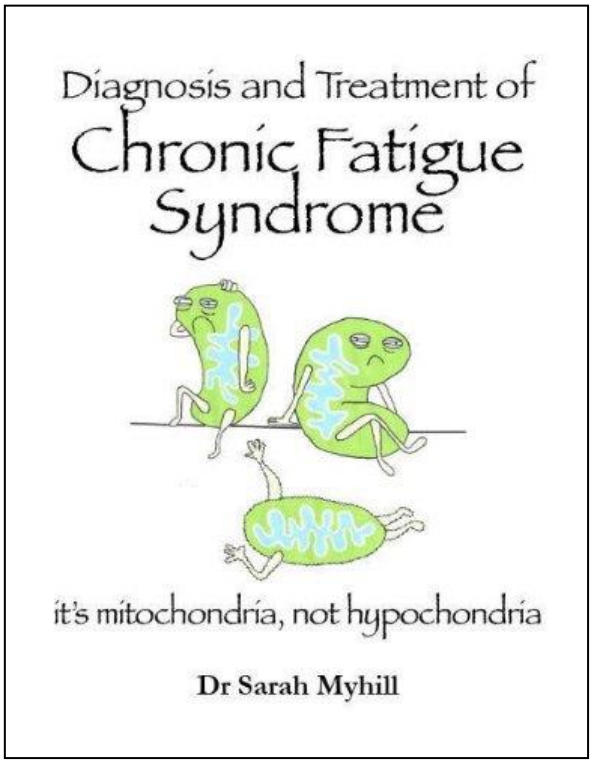
'Diagnosis & Treatment of Chronic Fatigue Syndrome - - it's mitochondria not hypochondria' - £14.99 (print) and £7.50 (ebook) from www.hammersmithbooks.co.uk. Launched 5 April 2014 in print and for Kindle, Nook, Kobo and ipad.

Example reviews

Dr. Myhill has written an extraordinary book. She is the no 1 authority on CFS in the UK. Whereas many doctors dismiss the condition, she explains what it is, what has gone wrong in the body, using appropriate tests that are not done by mainstream medicine, and tells people what they can do about it. Whereas mainstream medicine only uses drugs to deal with a particular symptom, Dr. Myhill explains the reasons 'Why'. In my opinion, you will never cure anything unless you understand and deal with the why.

Dr. P J Kingsley MB.BS.,MRCS,LRCP.,FAAEM., DA.,DObst.RCOG.

This book is logical and evidence-based but is still an easy read. Dr Myhill has made the mitochondrial story clear for the non-expert. It makes essential reading for family members or friends of CFS sufferers. This book will help these patients to obtain understanding and effective management from their physicians.



Dr Sybil Birtwistle, MB ChB, DObstRCOG, DCH, Post Graduate Tutor, British Society for Environmental Medicine.

This book is an invaluable tool to recover from fatigue-related disorders for patients and practitioners alike! It is based on years of clinical experience, backed by research; written in a clear, concise way with lots of practical instructions - and from what I can say using these methods in my practice: it works!

Dr Franziska Meuschel MD, PhD, ND, LFhom

This book is both a superb self-help guide for any person with CFS/ME and a concise manual for the liberated clinician. In her unerringly defiant style, Sarah shares her vast specialist expertise, in a lighthearted and upbeat fashion, using analogies that make complex scientific concepts sound simple. The practical tips make it a unique, up to date, resource book, a "must read" for all those in the health professions who sympathise with the quest of thousands of debilitated people for answers and better care.

Dr Apelles Econs, MRCS, LRCP. Allergist

Further reviews are available at: www.drmyhill.co.uk/wiki/CFS_book_reviews

LDN, Nootropics, Rituximab and fads: Maija Haavisto on treating CFS and Fibromyalgia

Due to the length of the original article I've selected choice parts. Please refer to the source address below for the full article. This article is provided for information purposes only, any treatments are used at your own risk.

Source: www.cortjohnson.org/blog/2014/05/28/ldn-nootropics-rituximab-fads-maija-haavisto-treating-chronic-fatigue-syndrome

By Cort Johnson 28th May 2014

Maija Haavisto is a Finish writer and Chronic Fatigue Syndrome patient who wrote the massive ME/CFS and FM compendium "Reviving the Broken Marionette: Treatments for CFS/ME and Fibromyalgia". Maija's health improved greatly with low dose naltrexone (LDN) and other treatments but pituitary damage in 2012 sent this very productive author reeling. Now she talks about what happened and gives her thoughts on ME/CFS and FM treatments.

Your book "Reviving the Broken Marionette: Treatments for CFS/ME and Fibromyalgia" provided an overview of over 250 ME/CFS and FM treatments six years ago. How have your views of ME/CFS and FM treatments changed in the past six years?

I am even more convinced now that low dose naltrexone (LDN) is "the" CFS/ME drug. Hundreds of Finns have now been using it and 2/3 have a positive experience. Some get minor benefit, others have life-changing experiences. I'm a bit less impressed by antivirals now, but more enthusiastic about beta blockers.

Besides rituximab and GC-maf I don't think there have been any major new treatments. Had Ampligen been approved the landscape might be very different now. My doctor in Finland is trialing some interesting new treatments that regulate the sympathetic nervous system.

I would say the biggest change in treatment is personalized medicine, like 23andMe, which has increased the importance of non-pharmaceutical treatments, like the various methylation protocol.

I've been hoping to update the book, but sadly it hasn't fit my schedule and finances – I can only afford to write in Finnish now. But should I update it, I will include a new chapter about verifying your diagnosis is correct. It is so common for people to get a CFS/ME diagnosis when they really have something else – sometimes several illnesses at once. Whether it's Sjögren's, Lyme, B12 deficiency, mold illness, adrenal insufficiency or something rarer.

Rituximab wasn't even a blip on the screen six years ago when you published your book. Now it's a major interest. What do you think about rituximab?

I included rituximab in the Finnish version of the book which came out in 2010, soon after the first Norwegian study, but I'm not enthusiastic about it, to put it mildly. I think CFS/ME should always be treated with immunostimulants – as pretty much all of the other major CFS/ME treatments are – never immunosuppressants.

The same goes for autoimmune diseases, as well. It goes against the dogma , but then again, first there was a dogma that autoimmune diseases could not even exist (horror autotoxicus) and for a long time the dogma was that they were extremely rare. My most recent medical book (published in Finnish last year) was about the treatment of autoimmune diseases and it presents a lot of evidence to support this idea.

I'm often asked whether I think CFS/ME is an autoimmune disease. Perhaps surprisingly, I don't have a clear answer for this, but I usually say that treatment-wise, it doesn't matter. Both are diseases of low immune function, which may in some ways manifest as "overactive immune system", but it's really just immune system that doesn't work right.

Politically, having conclusive proof of autoimmune origin would be helpful, but contrary to what some think, rituximab "working" doesn't prove CFS/ME is a physical illness – obviously we already have proof of that. Biological immunosuppressants are also being studied in psychiatric illnesses.

What do you think about microglial inhibitors?

It's a highly promising area, but needs more research to ascertain how promising. Naltrexone is a microglial inhibitor among its other modes of action. There is constant disagreement whether large or small LDN doses are more effective and some have suggested only using the enantiomer D-naltrexone (which is a microglial inhibitor with no opioid blocking effects) or using a heterochiral mixture (more D-naltrexone than L-naltrexone). Then people could take large amounts of the microglial inhibiting isomer and smaller amounts (or none) of the opioid blocker. But this is something the pharmaceutical industry must do, you can't have your pharmacy compound it.

Most drugs and supplements that inhibit microglial activation also have other potentially useful modes of action.

Your knowledge of pharmaceutical drugs in ME/CFS is incredible. Tell me one drug that you suspect is underused in ME/CFS and FM.

LDN is still underused, but at least now most people have heard about it.

I'd say nootropics (cognition enhancing drugs) are very underused. Most people, including many doctors, haven't even heard of them! Piracetam is very well-tolerated and even OTC in some countries.

Another class is NMDA antagonists. I hope to see proper studies on this front soon. Medical marijuana is probably very underused, sadly we all know why. I believe we probably have many great underused drugs, but even I don't know they're underused! There are several medications in my book that I believe would work well, but the data is still missing.

We hardly ever hear about nimodopine but it plays a key role in a Dr. Downing-Orr's book and treatment protocol. How helpful was nimodopine for you?

It was very helpful from the first dose. It wiped off that "foggy" feeling and it helped with language problems in particular, but in a different way from piracetam, which also helped on that front. It didn't help my fatigue or any other symptom except for orthostatic hypotension, which was always mild for me. The best part was that after half a year of taking it at half a tablet a day I was able to retain all the cognitive benefits even though I stopped taking it.

Several people on my forum have had the same experience of lasting benefits. One person found nimodipine very helpful for fatigue and blurry vision. Another woman found it made it easier for her to walk.

You wrote 'Don't go chasing expensive fad treatments". Is there anything in particular you want to put in that category?

I feel that applies for most areas of medicine. Not just CFS/ME but e.g. autoimmune diseases.

People think that if a new super expensive biological drug is approved, it must be the best treatment. Some new biologics do work well, others don't. Just because something is a biologic doesn't mean it works better than a pill. Targeted drugs aren't automatically more effective (or safer) than non-targeted ones and new meds don't always beat the old.

The range of treatments that can REALLY help some people with ME/CFS and/or FM is astonishing

Things that have had dramatic beneficial effects on at least one person on my forum, besides the LDN, include e.g. L-carnitine, D-ribose, methylfolate, wild vegetables, gluten-free diet, ozone therapy, oral saline, beta blockers, sumatriptan (for all symptoms), tramadol (similarly), modafinil, hydrocortisone, amitriptyline, fluoxetine, clonazepam, prednisone, testosterone, IVIG, nimodipine, antibiotics.

Some of these are common CFS/ME treatments, some are not.

My Finnish doctor has started treating people with saline solution (normal salt + water), usually just orally, and a few people have benefited a lot.

It shows that even though this is a very serious illness, sometimes simple things can help a lot.

Many people don't even want to try any treatments because they are just waiting for Ampligen and rituximab.

It's not so much which is the best treatment but what is the right fit for you.

What role do you think supplements and non-pharmaceutical treatments play in ME/CFS and FM treatment?

Some supplements that could help may be under many people's radar. I think for most of us medications are going to be the most helpful form of treatment (aside from rest/pacing), but that doesn't mean other treatments are useless. Some people benefit massively from diet changes, others not at all. Mitochondrial nutrients like carnitine, D-ribose, lipoic acid, NADH and PQQ help many of us, and if mitochondrial issues are your main problem, they may help more than anything else.

Less popular supplements that can be helpful include e.g. Epicor, benfotiamine, astragalus, berberine, cat's claw and inosine. Bromelain is great for post-exertional muscle soreness. As with meds, there are supplements that may carry a lot of promise, but hardly anyone uses them.

My Finnish doctor uses supplemental oxygen, which several of my friends have tried and feel it is very helpful. Ozone therapy may have use in some cases. A friend benefited a lot from neurofeedback. Acupuncture may help some. My Dutch doctor uses intermittent hypoxia.

You also talk about 'accepting' ME/CFS and FM and still fighting to get better. Many people think of acceptance as a kind of passive stance that doesn't include taking action. Can you talk about what you mean by 'acceptance' and how that doesn't mean for you caving in to this illness?

A Finnish friend (who used to have severe CFS/ME but is now much better) put it well. To get better you need a positive attitude. The attitude itself isn't helpful – you just need it to pursue treatment. Of course for many people getting treatment is difficult or impossible, because of finances or lack of local doctors, but others just give up, thinking that CFS/ME isn't really treatable, or that their own case is too difficult. They already tried several meds. Well, there are 250 meds in my book.

First direct evidence of Neuroinflammation – 'Encephalitis' – in ME/CFS

Source: http://phoenixrising.me/archives/24936
By Simon McGrath 29th April 2014

Simon McGrath reports on the new study that indicates low-grade encephalitis in ME/CFS …

A small study with just nine patients has captured the attention of patients and researchers alike after reporting direct evidence of inflammation in the brain of ME/CFS patients. The finding was one of the highlights picked out by Professor Anthony Komaroff in his IACFS/ME conference round up.

Back to the future
What makes this study so fascinating is that it provides tantalising evidence supporting not only of current views that inflammation in the brain is central to understanding the disease, but also of Melvin Ramsay's original name of 'myalgic encephalomyelitis'.

Encephalomyelitis is inflammation of the brain and spinal column, and critics of the name pointed to the lack of direct evidence for inflammation of either. This study only looked at the brain, not the spinal column (so could only find encephalitis), but the immune cells found to be activated in the brain are also present in the spinal column.

The study
To see if there is immune activation in the brain, researchers need to look inside the brain — which is not so easy if you want patients to still be alive when your study is done.


Dr. Yasuyoshi Watanabe

The scientists in this study, led by Dr. Yasuyoshi Watanabe from the RIKEN institute in Japan, used PET & MRI imaging to peer into the brain.

What make this study work is the use of tiny quantities of a radioactive tracer that binds to specific proteins that appear on activated microglia (the main immune cells of the brain) but crucially doesn't bind to non-activated microglia. The marker also binds to activated astrocytes, which play an immune role in the brain. The brains of nine ME/CFS patients meeting both Fukuda and International Consensus Criteria were compared with those of 10 healthy controls.

The results showed that neuroinflammation markers were higher for patients than controls across many brain areas including the thalamus, the pons and the midbrain. They also found that the severity of symptoms correlated with the degree of inflammation in multiple brain regions, particularly for cognitive functioning.

It was the correlation between a biological finding — neuroinflammation — and clinical problems that Komaroff found so exciting about this work, because it suggests a biologically plausible explanation for the symptoms of ME/CFS:

> "[If replicated] it would, for me, say that there is a low-grade, chronic encephalitis in these patients, that the image we clinicians have of encephalitis as an acute and often dramatic clinical presentation that can even be fatal has — may have — blinded us to the possibility that there may be that long-lasting — many years long — cyclic chronic neuroinflammation is underlying the symptoms of this illness."

Intriguingly, the midbrain, thalamus and amygdala — all regions where cognitive problems correlate with neuroinflammation — are also all part of neural circuits involved in awareness, arousal and attention. Concentration problems are typical of ME/CFS, and one of the problems found most consistently in laboratory testing.

Replication needed

While tantalising, these findings are far from conclusive, as the authors acknowledge. The study has only nine patients, albeit diagnosed with ICC criteria. The tracer used to identify activated immune cells produces a very 'noisy' signal, giving rather indistinct readings, and the overall level of neuroinflammation was relatively low.

Although cognitive issues correlated with neuroinflammation in several areas, generally other symptoms, including fatigue, did not significantly correlate with inflammation. There was almost no sign of inflammation in the prefrontal cortex, the region of the brain most involved in higher cognitive functions, that might be expected to be a problem in ME/CFS. And there was a potential technical weakness in the way the study was run.

Commenting on the neuroinflammation, Komaroff emphasised the need for replication:
"If it were confirmed by multiple other investigators … these data are consistent with [encephalitis], but I would feel more strongly if other labs using same technology came up with the same result."

The good news is that the authors of this study are already working on a new study using the same patients but with a newer and more sensitive tracer to pick up neuroinflammation. They will address the earlier technical issue, and to make the study more powerful they will also be looking at neurotransmitter activity in the brain, following up their previous findings of neurotransmitter abnormalities.

Hopefully independent groups will try to replicate this finding too – and in the U.K., Dr. Charles Shepherd of the ME Association has already said it would welcome applications to fund a replication attempt.

Microglia — key to ME/CFS?

So neuroinflammation — specifically activation of microglia — correlates with cognitive problems, but how might microglial activation cause the problem?

The most plausible answer is through what is termed 'sickness behaviour' — a characteristic set of responses to infection, including fatigue, malaise joint and muscle pain and problems concentrating — which might just sound familiar to ME/CFS sufferers. ('Sickness behaviour' is a lousy name for biological phenomenon, as Dr. Dan Peterson has noted).

Microglia are known to play a key role in regulating sickness behaviour, and that's a big reason this study has attracted so much attention in ME/CFS.

The fatigue, malaise, problems concentrating, etc., of sickness behaviour help us survive an infection by forcing us to rest so our body can devote all its resources to the energy-greedy immune system.

'Sickness Behaviour' is driven by biology: infection leads to a rise in pro-inflammatory cytokines in the blood, triggering activation of brain microglia and their production of cytokines. This triggers sickness behaviour.

However, sickness behaviour is normally a short-lived response to an acute infection, designed to temporarily divert resources to ensure a swift recovery. If that doesn't happen, e.g., if there is a chronic infection, or the process goes wrong, for instance, if microglia remain activated after an infection has been cleared, then sickness behaviour can itself be a problem. ME/CFS may be an example of this.

Cytokines in the spotlight
Cytokines are a key trigger for sickness behaviour, and researchers have often found elevated cytokines in patients, but the findings have been inconsistent and in small studies. The new studies reported on by Dr. Jose Montoya at the Stanford conference and Dr. Mady Hornig at the IACFS/ME conference are helping to firm up these findings in huge cohorts.

Probably the most important piece of work on the role of sickness behaviour — and cytokines — in ME/CFS came from the landmark "Dubbo" studies.

The researchers found that about 11% of those with glandular fever and two other infections developed CFS after six months. And crucially, what predicted the length of the illness (and chance of developing CFS) wasn't psychological factors, but the severity of the initial 'acute illness', or sickness behaviour.

The researchers also showed that those with more active genes for the pro-inflammatory cytokine Interferon-gamma had a more severe sickness behaviour (and longer illness) than those with regular versions, linking cytokine response to sickness behaviour and ME/CFS.
The Dubbo study did not look at inflammation in the brain, but the authors did speculate that the cause of CFS could be long-term activation of microglia and astrocytes. And that is exactly what was found in this new PET imaging study.

As with all research findings, replication is essential, and a new version of the Dubbo study is currently under way in Sydney, Australia.

The new imaging study from Japan has found provisional evidence of activated astrocytes and microglia cells (both types of glial cell) in the brain of ME/CFS patients. This is support for the suggestion from the Dubbo team that ME/CFS develops from certain infections as a result of activation of brain microglia.

Dr. Michael VanElzakker's recent vagus nerve infection hypothesis also features glial cells heavily. And recently Professor Hugh Perry, who has studied microglial cells in neurodegenerative diseases such as Parkinson's disease, proposed that primed microglia and sickness behaviour lie at the heart of ME/CFS.

Neuroinflammation and Sickness Behaviour the final common path in ME/CFS?
It may prove to be that 'neuroinflammation' — i.e., activated microglia in the brain/spinal column — is a common endpoint of numerous triggers, including glandular fever (EBV), other infections, vaccines — or even, as Dr. Lipkin has proposed, disturbances in the microbiome.

Discovering if this is the case — and firming up the finding of neuroinflammation is key — could be a big step forward in understanding and then treating ME/CFS. And those it is still very early days, it is possible this approach could eventually show that Dr Ramsay was right about 'encephalomyeltitis'.

Watch out for a new blog on sickness behaviour, microglia, cytokines and their role in ME/CFS, coming soon.

Simon McGrath tweets on ME/CFS research: @sjmnotes

Further information
Simon McGrath has written a more in-depth overview of Microglia, Cytokines and Sickness Behaviour here:

http://phoenixrising.me/archives/25148
http://phoenixrising.me/archives/25310

In Brief: The Adrenal Glands and ME

Source: http://phoenixrising.me/archives/24193
By Andrew Gladman April 1st, 2014

The second in a new series of 'In Brief' articles, where Andrew Gladman provides a helpful insight into the science behind fairly common topics, exploring how they relate to ME/CFS. This time he discusses the adrenal glands and why they can be such a talking point …

While the frequent topics of conversation relating to ME/CFS appear to now be infectious agents, autoimmunity and often a dysfunctional nervous system, many patients and researchers still turn their attention to problems within the endocrine system, namely the adrenal gland.

As the gland within the body centred around stress responses, it is initially quite a logical place to look for problems. There can be no denying that patients suffering with diseases of the adrenal glands certainly do share many symptoms and complaints of those afflicted with ME/CFS.

There is then good reason to query where the adrenal glands may relate to ME/CFS. While the initial reason for querying this may seem simple, the answers raise interesting questions for the potential pathophysiology and reasons for the symptomology of ME/CFS.

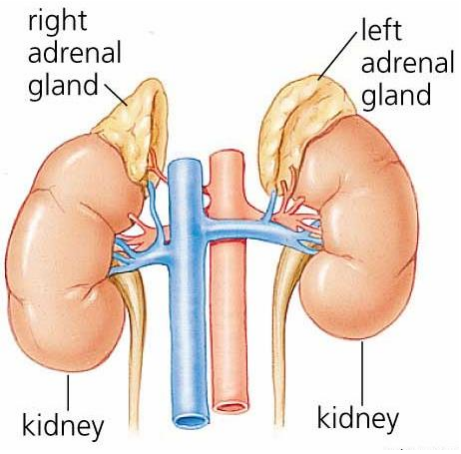


Diagram showing the position of the adrenal glands above the kidneys

What are the Adrenal Glands?

The adrenal gland is one of the body's most vital endocrine glands. The adrenals are glands which secrete their hormone directly into the bloodstream, often as a result of the glands being innervated with blood vessels. Sitting above each kidney, these glands' primary function is releasing the hormones and chemicals which in turn stimulate a stress response, often known as a fight or flight reaction.

However the adrenal glands do carry out numerous other functions.

The stress response is initiated through the production and release of corticosteriods, such as cortistol, and a larger group of chemicals known as catecholamines of which adrenaline (epinephrine) and noradrenaline fall under.

The adrenal gland is composed of numerous layers although it is often discussed simply as being split into two main regions, the medulla and the cortex.

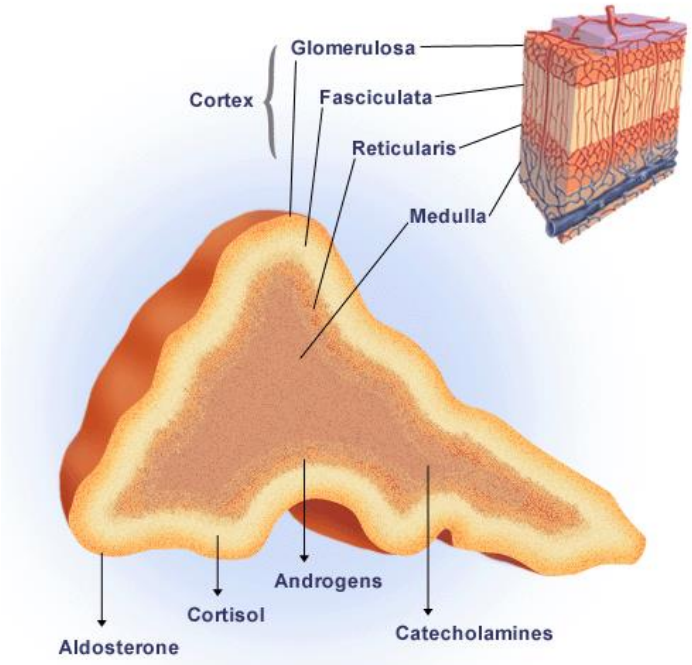


Diagram showing the division between the adrenal medulla and cortex, along with the chemicals produced by each

The medulla is the centre of the gland and the cortex is the outermost layers. The medulla is responsible for the production of adrenaline and noradrenaline. It achieves this through complex innervation with neurons of the sympathetic nervous system which stimulate the adrenal medulla in times of acute stress, increasing the rate of synthesis and release of these chemicals.

The cortex, on the other hand, is tasked with the production of corticosteroids, including cortisol and aldosterone, along with androgen hormones (the male sex hormones). The cortex is further sub-divided into three separate layers differentiated by which of the discussed chemicals is being produced by the specialised cells in that region, although the specifics of this sub-division are very rarely discussed.

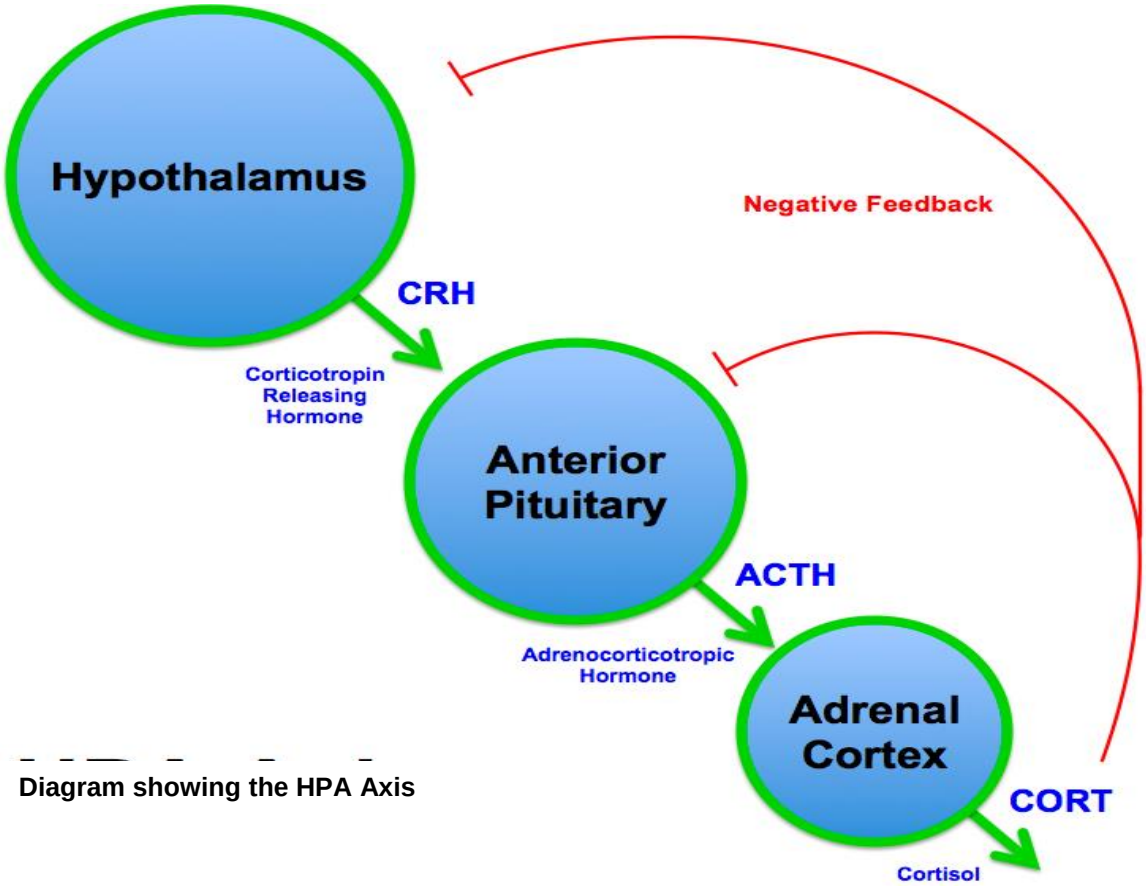


Diagram showing the HPA Axis

Given the importance of the adrenal gland in producing these vital chemicals, there has evolved a complex set of interactions between other endocrine glands, the adrenal gland and the brain to help regulate one another through a process known as negative feedback.

This set of interactions occurs between the hypothalamus (a region at the centre of the brain), the pituitary gland (a small gland located at the base of the brain) and the adrenal gland. This set of interactions between these three areas is known commonly as the hypothalamic-pituitary-adrenal axis, often abbreviated to HPA axis. It is this HPA axis that is frequently discussed in relation to ME/CFS.

Given the wide range of chemical messengers produced by the adrenal gland through the complex interactions it shares within the HPA axis, it is clear that the gland has far-reaching consequences within the homeostasis of the body. These include playing roles in helping to control and regulate body temperature, digestion, immune system responses, mood sexuality and energy usage, along with the adrenal's primary function of controlling the physiological reaction to stress, trauma and injury.

It is clear to see why any dysfunction in either the adrenal gland alone or in the HPA axis as a whole could potentially cause a plethora of problems and symptoms. Dysfunction in the HPA axis is well known to be involved in a wide variety of psychological illnesses and is now being understood to play quite a large role in many physiological conditions too — understandably, given the stress that disease itself causes.

Why are the Adrenal Glands important in ME?
For many years now studies have indicated finding abnormalities in HPA axis function within the ME/CFS cohort. These abnormalities are often described in the literature to include mild hypocortisolism, heightened negative feedback, and blunted HPA axis responsiveness.

Fundamentally, this means that in those patients observed, cortisol levels are persistently lower than is to be expected. This appears to be traced back to the hypothalamus and pituitary gland becoming somewhat unable to appropriately detect or respond to the low cortisol levels.
Many studies have not just identified this as a fairly consistent finding in ME/CFS but have furthermore observed a correlation between this dysfunction and symptom severity. It is of note however that some studies dispute this finding.

This dysfunction in the HPA axis has been used somewhat deviously by some groups to verify the effectiveness of talking-based therapies such as cognitive behavioural therapy (CBT).
This is in part due to the effectiveness these therapies can appear to have in conditions such as anxiety disorders in which HPA axis dysfunction has also been proven to play a somewhat central role. However such a line of thought, while helpful in many diseases where HPA axis appears to be a disease mediator, is unlikely to be helpful in a disease where the HPA axis does not yet appear to be central.

While there are a large number of studies confirming their independent findings of HPA axis dysfunction, few make the leap to develop a hypothesis for this being the central disease mechanism. This is likely a result of the multitude of other research indicating a deeper physiological defect in ME/CFS of which HPA axis may simply be an unfortunate by-product.
One interesting study that bears thought as to HPA axis dysfunction developing as a secondary condition is a somewhat unrelated study by Dunn et al. This study focuses upon how cytokines can in turn activate the HPA axis, initiating a stress response. Cytokines are a rapidly emerging line of study in ME/CFS and we recently produced an article exploring why they are gaining increased exposure in the ME/CFS research field.

It’s an interesting notion that cytokines can directly influence the functioning of the HPA axis. When we consider the emerging results of researchers such as Prof. Lipkin outlining significant cytokine abnormalities in ME/CFS, perhaps we have a logical line of reasoning as to why the HPA axis appears somewhat blunted.

If the observed cytokine abnormalities prove repeatable and appear chronic, then it stands to reason that the HPA axis is under significant strain for a prolonged period of time. This could account for lower cortisol levels as time progresses, alongside a blunting of the HPA axis response when challenged with a stressor.

This hypothesis would also be supported by the HPA axis dysfunction seen in other chronic conditions with significant cytokine disturbance, such as lupus.

Overall, while problems with the adrenal glands and HPA axis function don’t yet appear to be a sole causative agent for ME/CFS, they do provide an interesting explanation for certain symptoms of ME/CFS from which patients suffer. As time progresses, and through the thorough research done every day, we learn more and more about the complex interconnections between different organs and systems such as the HPA axis.

The connections between these systems, while seemingly unrelated to many diseases, appear to play quite a substantial role in the symptomolgy of ME/CFS, perhaps tying together with increasing evidence of autonomic dysfunction within ME/CFS.

DVD of the 2014 Invest in ME conference

The conference took place on the 30th May 2014 in Westminster, London.
A DVD is available to buy and is due for delivery sometime in July.

Currently the price displayed on the Invest in ME website is £12 which was a discount price for May. I've just ordered a copy in the first few days of June and it cost £14.

At time of printing the link for information about the DVD was:
www.investinme.eu/DVD-earlybird.html

An overview of the presenters and topics is included below.

Keynote Speech - Professor Jonathan Edwards

Finding Antibodies in Neurological Diseases - Professor Angela Vincent

Infection-induced autoimmunity in ME - Professor Jonas Blomberg

Pathogen Discovery in ME - Professor Mady Hornig

EBV and ME/CFS - Professor Carmen Scheibenbogen

Gut Microbiome and ME/CFS - Professor Simon Carding

Innate and Adaptive Immune Cells in ME - Professor Sonya Marshall-Gradisnik

Brain Imaging and ME - Professor James Baraniuk

ANS and ME - Professor Julia Newton

Markers of Post-Exertional Malaise in ME - Professor Maureen Hanson

Diagnosis/Treatments and ME in USA - Dr. Andreas Kogelnik

Diagnosis/Treatments and ME in UK - Dr. Amolak Bansal

Diagnosis/Treatments and ME in Clinical Practice - Panel Discussion (Dr. Andreas Kogelnik / Dr. Amolak Bansal / Dr. Saul Berkowitz)

External View of ME Research Strategy - Dr Julian Blanco

Payment
Payment can be made online at: www.investinme.eu/DVD-earlybird.html

Or, payment can be made by a cheque for £14 sent to:

Invest in ME
PO BOX 561
Eastleigh SO50 0GQ
Hampshire
UK

Please supply your name and address (and email address if possible)

Cheques should be made payable to Invest in ME

Simmaron Research Foundation study targeting roots of immune system breakdown in ME/CFS

Source: http://simmaronresearch.com/2014/06/simmarons-next-immune-study-wheres-weak-link
By Cort Johnson 13th June 2014

Simmaron Research's new immune study builds on exciting research that is changing how we think about ME/CFS.

Twenty years ago the internationally known virus hunter, Dr. Ian Lipkin of Columbia University, didn't find Borna Virus in people with ME/CFS, but he never forgot the immune dysfunction he found. Twenty years later he found more immune dysfunction in another study.

He doesn't know why it's there but he does believe that all ME/CFS cases – no matter what pathogen or other factor has triggered them – devolve to a 'common pathway'. The fact that pathogens of all types – from Epstein-Barr Virus, to SARS, to Giardia – can trigger ME/CFS suggests a core immune deficiency lies at the heart of the illness.

Isabel Barao, PhD, the Simmaron Research Foundations Scientific Director believes a genetic predisposition to immune problems could underlie ME/CFS

Every genetic study suggests an inherited susceptibility to Chronic Fatigue Syndrome is present. Dr. Mady Hornig of the Center for Infection and Immunity at Columbia University believes that a genetic predisposition in combination with an environmental trigger (such as an infection) occurring at just the right (wrong) time is probably key to coming down with ME/CFS.

For thirty or forty years you might be able to easily slough off this bug or that pathogen, but at some point for some reason the stars aligned; you were depleted in just the right way, the pathogen hit and with your immune system genetically predisposed to crack under the pressure – it did – and your entire system faltered.

Simmaron Research's next pilot study is looking for that immune crack in the dike – the genetic underpinnings of the system collapse that occurred. Led by Simmaron's Scientific Director, Isabel Barao, PhD, in collaboration with researchers at the National Cancer Institute and University of Nevada Reno, it will determine if your NK and B-cells and macrophages are genetically predisposed to respond poorly to a virus, toxin, or cancer cell.

Dr. Barao is studying whether people with ME/CFS have polymorphisms – unusual gene formations – that make their key immune cells less likely to respond well to viruses and other threats. That immune 'hole' many people have talked about with regards to ME/CFS could start here. We all know about the rampant NK cell problems in ME/CFS, but this study could help explain the B-cell problems recently uncovered in a German research study – and perhaps even shed light on why Rituximab may be working in some patients.

It's the initial part of a projected three-part study that could end with drugs for ME/CFS. Once genetic alterations have been found, they'll be correlated with immune findings. If that holds up, it'll be time to look for drugs to fix the problem, two of which are currently in clinical trials.

Think about it. The high heritability rates in ME/CFS indicate genetic problems exist somewhere. Where better to look than the immune system? This study is a no-brainer to me. It's relatively cheap – it has a quick six-month turnaround – and the data it produces will lay the foundation for an NIH grant on topics they've shown they're willing to fund.

Acetyl-Glutathione

Certain supplements, such as B12 and Glutathione, are difficult to absorb (e.g. mostly destroyed by the digestive system). Our last newsletter introduced 'Liposomal B12' as a new way to get the B12 past the digestive system, delivering the B12 directly to cells of the body.

There is also a Liposomal form of Glutathione. For example: "ReadiSorb Liposomal Glutathione" from Amazon UK for £48.99

However, Acetyl-Glutathione may be better yet. Both are expensive supplements. Perhaps, when they are not quite so new they will be available at a more accessible price. For Acetyl-Glutathione a brief overview, example and link to further information is included below.

Glutathione is our main anti-oxidant and detoxifier.

Overview

"Acetyl-glutathione is orally active, unlike plain glutathione, and is stable in the intestine and plasma when absorbed and delivered directly to the cells for natural de-acetylation intracellularly. Plain glutathione delivered to the plasma by precursors, liposomal products or intravenously must be broken down by enzymes to the basic amino acid components for absorption into the cell and require more energy expenditure to be re-constructed back to rGSH.

It is known that disease states can block the re-assimilation of components into rGSH. Therefore, it is a better dietary/therapeutic decision to provide the orally active and absorbed Acetylglutathione which increases intracellular rGSH directly and naturally without increased energy expenditure and without being compromised from disease states."

Source of overview: see 'Further Information' link below

Example

Allergy Research Group - Acetyl-Glutathione 100 mg 60 tabs £82.17 From Amazon.co.uk

Further information

www.nleducation.co.uk/resources/reviews/oral-glutathione-equivalent-to-iv-therapy

---

The Guildford & West Surrey ME/CFS Group newsletters aim to inform members of relevant news and treatment options. Use of the treatments is done at your own risk.