Guildford ME/CFS Support Group
(& West Surrey)

Newsletter

June 2017

Future dates

Open to all members and carers.

17th July 2017 (Monday) 11.15am The Seahorse
The Street, Shalford, Guildford, GU4 8BU
www.theseahorseguildford.co.uk

8th August 2017 (Tuesday) 7.30pm The Weyside
Millbrook, Guildford, Surrey, GU1 3XJ
http://www.theweyside.co.uk
(Parking at Millbrook car park £1 for evening)

13th September 2017 (Wednesday) 11.15am The Seahorse
The Street, Shalford, Guildford, GU4 8BU
www.theseahorseguildford.co.uk

Biomarkers

Source: http://solvecfs.org/wp-content/uploads/2017/03/Chronicle-02-2017-1.pdf

What is a biomarker?
When is the last time you took your temperature using, your thermometer? Temperature is the most commonly used biomarker—one that we all know and trust. We all have a thermometer at home and have used it many times to confirm that our temperature is abnormally high, indicating that we have an infection somewhere in our body. This self-administered biomarker measurement is used to determine whether our kids should stay home from school, we should call the doctor, or we should stay home from work. This is a great biomarker, as it meets all the criteria we seek.

- So, what are those criteria? What makes a good biomarker?
- In the example above, the biomarker is elevated temperature as measured by the home thermometer. The process for testing this biomarker is wonderful for many reasons:
- It's non-invasive; putting a tab under the tongue or in the ear is not painful
- It has little risk of harm
- It's very inexpensive; most everyone has a thermometer at home
- It's reliable; we trust the number we see
- It's easily accessible; we don't even have to drive anywhere
- Everyone agrees that a fever indicates infection; it's well accepted in the medical community
- It's specific, meaning that it identifies an infection and not something else (like a cataract)

Why do biomarkers matter so much?
Biomarkers can be important to diagnose, verify, and track the presence of disease; having a great biomarker can even transform an illness.

For instance, the creation of the PSA test for prostate cancer meant that men could be diagnosed much sooner. And the Pap smear, designed to screen for cervical cancer and based on research dating back to the 1920s, has saved thousands of lives. Acute promyelocyte leukemia, once a death sentence, is practically eradicated thanks to identification of its biomarker, fusion oncoproteins (proteins that can turn cells into cancer cells), and, later, successful corrective treatment.

In diabetes, the detection of chronically elevated sugar levels makes diagnosis straightforward. And the discovery of the biomarker responsible for cystic fibrosis, the inherited gene for the CFTR protein, revolutionised that disease. Biomarkers can be useful for diagnosis and treatment even when the underlying cause of the disease is not understood.

So, a biomarker for ME/CFS that has all the qualities listed above—it's non-invasive, inexpensive, reliable, accessible, well-accepted, and specific while also having little risk of harm, would transform the diagnosis and treatment of our disease. Just imagine going to your doctor and doing a simple test then having her announce, "Yes, you have ME/CFS." Certainly, that is the very opposite of the experience of most patients now!

Do we have any biomarkers for ME/CFS now?
Well, yes and no. We don't have any biomarkers that meet all the criteria above. But we do have a few that fall short. They are all either too invasive, too expensive, too potentially harmful, or not well accepted. Most existing biomarkers for ME/CFS fall, broadly speaking, into established categories such as neurological (e.g., neuroanatomical, neuroendocrine, or neuro-cognitive), metabolic (e.g., altered regulatory functions of key enzymes, suboptimal processing of nutrients), immunological (e.g., altered activities in cytokines, B-cells, or natural killer cells), hemodynamic (e.g., reduced total blood volume, reduced cerebellar perfusion, or cell-free markers like specific microRNAs in blood), or pathological (e.g., microbiome changes, viruses, and pathogens).

Here are some of the difficult biomarkers we have for ME/CFS now:

The spinal fluid of severe ME/CFS patients shows elevated levels of autoimmune markers and white blood cells. But this test fails on most criteria. It is quite invasive, there is risk of harm, it's expensive, and it's not generally accepted. The key positive attribute is that it is reliable MRIs for ME/CFS patients show reduced gray and white matter in the brain. This biomarker is a bit better than a spinal tap, as it's non-invasive, there is little risk of harm, and it's reliable. However, it's quite expensive, it's not easily accessible, and it isn't clear that the medical community has accepted it.

ME/CFS patients' natural killer (NK) cells show reduced functionality. NK cells act as the first line of defence in the immune system and are a key component of one's blood. This biomarker is one of the earliest uncovered in ME/CFS and has stood the test of time. However, determining the functionality of these cells requires a sophisticated laboratory setup, and data interpretation is not straightforward, requiring special expertise. In addition, other immune-related conditions are also characterized by reduced NK cell function, making this biomarker not unique to ME/CFS.

The majority of ME/CFS patients have markedly different "anaerobic thresholds."
This measurement, taken during cardiopulmonary exercise testing (CPET), determines the complex capacity of the whole body for energy production.

Common in exercise physiology research and athletic performance analysis, this measurement also requires sophisticated equipment and specialised expertise for data interpretation. In the ME/CFS community, it is well known that patients experience post-exertional malaise (PEM) after pushing their energy boundaries.

‘Outrageous interference’ with rule of law as DWP sets 80% benefit appeal rejection target

Source: www.politics.co.uk/news/2017/05/16/dwp-sets-80-benefit-appeal-rejection-target

16 May 2017. Story by Natalie Bloomer.

The Department for Work and Pensions (DWP) has set staff a target of upholding 80% of the benefit decisions that they are asked to reassess, it has been revealed.

In response to a Freedom of Information request, the department stated that a key measure used to monitor the performance of Mandatory Reconsiderations (the first step in the benefits appeals process) is that 80% of the original decisions are to be upheld.

It went on to confirm that between April 2016 – March 2017 staff exceeded that target with 87.5% of original decisions being maintained.

Henry Brooke, a former judge and member of the Access to Justice Commission, wrote in a blog post yesterday that the revelations appeared to be an “absolutely outrageous interference” with the rule of law.

Since 2013, benefit claimants who wish to appeal against a decision made by the DWP have had to request a mandatory reconsideration before being able to take their case to a tribunal. Staff then look at the application again and have the power to overturn the original decision if they think it was incorrect. Many of the cases relate to claims for disability benefits.

The DWP has always insisted that mandatory reconsiderations make the appeal process fairer but critics have warned for some time that it is confusing and can deter people from appealing.

Last year, the National Association of Welfare Rights Advisors (NAWRA) submitted evidence to a consultation by the Social Security Advisory Committee (SSAC) on decision-making in the DWP. It included widespread concerns that mandatory reconsiderations add “confusion, obstruction and complexity to the appeals processes” with “little or no added value compared with the older automatic revision process”.

Disability Rights UK (DR UK) said that disabled people should not be delayed access to justice.

“That the DWP has an actual target for refusing benefit decision reconsideration requests is bad enough, but that it has set and exceeded a figure of 80% is appalling,” Ken Butler, a welfare benefit adviser at DR UK, said.

“Disabled people should not have delayed access to justice of sometimes several months in order to first pursue an often futile mandatory reconsideration request to the DWP. We would urge all disabled people who are rejected at the mandatory reconsideration stage to seek advice about making an appeal to an independent tribunal.”

A spokesperson for the DWP said:

“Mandatory reconsiderations look at all the evidence afresh, including any new evidence provided by the claimant. Our key performance measures are strictly used to assess the accuracy of the original benefit decisions.

“We want to ensure we get decisions right first time around, and performance measures help to monitor this.”

Invest in ME Conference 2017 – the DVD

The 12th Invest in ME Research International ME Conference 2017 - took place on 2nd June 2017 in London and again attracted delegates from eighteen different countries - from Europe, North America, Middle East and Australasia.

The conference day was preceded by the 7th Invest in ME Research Biomedical Research into ME Colloquium - two full days of closed researchers' meeting with over 70 eminent researchers from fourteen countries in attendance.

The theme was Centres of Excellence for ME - reflecting the establishment of a UK Centre of Excellence for ME and linking to other centres being set up around the world. This underlines the charity's work in developing international collaboration in research into ME via establishment of the centre in UK - see http://www.cofeforme.eu

Speakers

The conference was opened by **Dr Ian Gibson.**

The first presentation was by **Dr Ian Charles** (leader of the Quadram Institute, Norwich, UK), who outlined the development of the UK Centre of Excellence for ME/CFS. He told us there are likely to be approximately 250,000 with ME/CFS in the UK, of which 25,000 are children and young people. Up to 84% may not yet be diagnosed. Possible causes were outlined, which included dysbiosis of the microbiota and possible auto-reactivity to commensal microbes.

Dr Vicki Whittemore (NIH, USA) is programme director of the National Institute of Neurological Disorders and Stroke (NINDS). The NIH (made up of 27 institutes and centres) seeks knowledge about the nature and behaviour of living systems. The NIH is committed to ME/CFS research, both intramurally and extramurally. A current study is looking at 40 patients, 20 controls and 20 recovered Lyme disease patients. An extensive analysis and bio-specimen collection is being undertaken.

Professor Donald Staines (Griffith University, Qld, Australia) discussed their paper “Impaired calcium mobilization and dysregulation of transient receptor potential melastatin 3 (TRPM3) ion channels in Natural Killer (NK) cells from CFS/ME”. Their group were the first to identifyTRPM3 on NK cells. There is impaired calcium mobilization. They also have studied NK cells, and find function and expression significantly reduced.

Professor Nancy Klimas (Miami, Florida) – reported on their genetic signature study. She pointed out that genetic studies are expensive, and large numbers of patients are needed – as many as 30,000 to be finding significant differences, therefore much funding is needed. She is part of a very large team. She outlined the symptoms of the illness. Studies are being undertaken to determine diagnosis, genetic risk, explanation of the illness and prediction of therapy.

Dr Jakob Theorell (Karolinska Institutet, Sweden) works focussing on the understanding of the mechanisms of disease in patients suffering from chronic immunodeficiency syndromes. As an example of his work he initially discussed a rare potentially fatal disease in babies - Familial hemophagocytic lymphohistiocytosis (FHL), a disorder in which the immune system produces too many activated T cells. He has been studying NK cells and cytotoxic T-cells in ME/CFS. Professor Jo Cambridge/Fane Mensah (University College Hospital, London) – works in the area of B-cell depletion treatment (Rituximab) in rheumatoid arthritis. Rituximab only works in sero-positive patients, not sero-negative. After treatment there is a delayed return of B cells – memory cells having been left behind, leading to gradual repopulation of the B cells. The number of B cells returning is not related to relapse. Rituximab works if auto-immunity is present. It stops new B cells differentiating to plasma cells. It also stops the interaction of B cells with T cells. Relapse occurs when B cells start to mature.

Professor Simon Carding (Norwich, UK) presented his team of young researchers from the University of East Anglia, who are researching the gut virome. For example: Fiona Newberry discussed the differences between the healthy and unhealthy gut. She described how a viral infection could cause inflammation and change in the microbiome. There could then be bacterial leakage into the bloodstream with auto-immunity.

Associate Professor Mady Hornig (Columbia University, NY, USA) discussed gut metabolome-immune disturbances in ME/CFS subsets. She firstly reviewed the metabolome and immune disturbances. She discussed the influences of genetic factors, epigenetic regulation and environmental exposures during pregnancy and later life.

Professor Olav Mella (University of Bergen, Norway) – started by giving a review of their clinical studies focusing on the use of RituxME (rituximab) and CycloME (cyclophosphamide). Earlier studies indicated that B-lymphocyte depletion may result in symptom improvement in a subgroup of ME/CFS patients. A Phase 3 double-blind, placebo-controlled intervention study with rituximab is now underway and due to be unblinded in October 2017. In the earlier rituximab trials, responses were transient and as B cells returned there was likelihood of relapse. Maintenance treatment therefore seems likely to be needed.

Dr Ingrid Rekeland (Bergen, Norway) discussed more of the Norwegian work on behalf of Prof Øystein Fluge, who was recovering from injury. She discussed the likely obstruction of metabolic pathways in ME/CFS.

Professor Warren Tate (University of Otago, New Zealand) started by explaining how he, as a biochemist had come to be involved in ME/CFS research. His 14 year old daughter had developed the illness after EBV. He graphically likened the research to the piecing together of a jigsaw puzzle. He is researching a small number of patients employing the technique of Precision Medicine (many tests on a small number of patients). MicroRNAs control all our physiology, and their study reflects what is going on in the body. Data has been collected on the transcriptome and the proteome as well as plasma microRNAs and cytokines, with the aim of integrating the data to elucidate linkages between different classes of molecules, and to give insight into physiological changes.

Professor Ron Davis (Stanford, California, USA) also has pioneered his ME/CFS research as a result of having a severely ill son. He says this is no doubt a molecular disease, and there have been many molecular breakthroughs. New technology is developing fast. The aims should be to look at the mechanism of the disease, a diagnostic tool and then treatment.

The DVD

The DVD cost £14. The DVD contains 4 discs and is in PAL format (UK relevant format) and contains the full presentations from the 2016 conference plus plenary sessions, and the pre-conference dinner keynote speech by David Tuller.

To order by website:

Go to: http://investinme.eu/IIMEC12.shtml#report

To order by post:

Send a cheque for the requisite amount (see above) to -
Invest in ME Research 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 Research

Mindfulness course in Guildford

Our group has been contacted by Rachel Crookenden, a Guildford based 'Clinical Specialist Physiotherapist and Mindfulness Coach'. For some, Mindfulness could be an important part of an overall coping strategy.

Mindfulness

Mindfulness provides us with effective techniques to combat the stresses and strains of our everyday, 21st century lives. Mindfulness helps us cope with the external pressures of our work and family commitments and it can also help us when we feel overwhelmed by our internal thoughts and feelings. With some simple mindfulness skills, we can learn to bring our attention into the present, to what is actually happening around us in any given moment and away from our worries, about either the past or the future.

Mindfulness teaches us to focus our awareness on what is happening 'in the now', rather than being 'caught up' in our thoughts and allowing the present moments to slip past, unnoticed. Mindfulness techniques can be practised anywhere, at any time; simple exercises that bring you into the present, so that you feel calmer, more content.

Benefits include:

- Feel calmer, more content.
- Less worry about the past (rumination)
- Less anxiety about the future (catastrophising)
- Improved concentration
- Improved working memory – the ability to focus on a task
- Less emotional reactivity
- Better able to cope with persistent pain or discomfort

About Rachel

I have worked as a Clinical Specialist Musculoskeletal Chartered Physiotherapist in the Guildford area for 15 years and my particular area of interest and expertise is the assessment and treatment of spinal problems – painful necks and backs. My work with complex spinal and general musculoskeletal conditions has enabled me to understand the distress and functional limitations that people with chronic pain or discomfort can experience. In order to learn more about the various contributing factors to long-standing conditions and to give me a wider choice of therapeutic strategies to offer, I completed a Masters level course at Hertfordshire University in Psychological Therapies, which included Mindfulness, Cognitive Behaviour Therapy (CBT) and Motivational Interviewing. I have subsequently completed several other Mindfulness training courses, including one specifically for the use of Mindfulness with long-term medical conditions – Breathworks Mindfulness for Health. For further details of my qualifications and how Mindfulness can be beneficial for those suffering from chronic symptoms, please go to:

www.timetobemindful.com/about

I have been running 8 week Mindfulness Courses in Merrow, Guildford, for 2 years now and am adding a new course in September specifically designed for those who suffer from chronic, long-term pain and / or symptoms. This course will start on Monday, September 25th 3 - 4.30 pm. For more details about this course – venue, dates and FAQ, please go to:

www.timetobemindful.com/chronic-pain-course

I hope that my 8 week Mindfulness Courses may be of interest to you. If you have any questions, or would like any further information, my contact details are below, or you can use the 'Contact' page on the website: www.timetobemindful.com/contact

I look forward to hearing from you.

Rachel
Phone: 07795 106478 Email: rachel@timetobemindful.com

David Tuller investigates the PACE trial

Source: www.meaction.net/2017/06/08/interview-with-tuller-on-pace-work

For many years now ME/CFS people have suffered both the illness and the surreal (if not sinister) situation where exercise and psychotherapy are touted as cures for the biological (likely autoimmune) illness that leave many housebound/bedbound if not tube fed. Perhaps because they are the cheapest dismissal of the widespread long-term disabling illness.

If the weight of world research and internationally agreed diagnostic criteria weren't enough to make the layperson cringe, pro-CBT and pro-Graded Exercise efforts such as the PACE trial are both: shown to be astonishingly phony; and remain the mainstay of government action (inaction).

It is with this in mind that David Tuller (private investigator) has been at work addressing the PACE trial…

David Tuller has been hard at work investigating the very many flaws of the PACE trial (which he has pointedly called "a piece of crap") for more than three years. We at #MEAction are grateful for the work he has done to fight for good science and for people with ME. We know too many people who have gone downhill (including becoming housebound and sometimes bedbound) due to following PACE-based recommendations to exercise. When we found out that David needed to raise money to continue his effort "to expose the methodological and ethical problems in PACE," we knew we'd do anything we could to help plan and publicise his fundraising campaign.

> The website address:
>
> https://www.crowdrise.com/virology-blogs-trial-by-error-more-reporting-on-pace-mecfs-and-related-issues1

David's first in-depth report on the PACE trial, a 15,000-word series published over three days in 2015 on the science site Virology Blog, methodically dismantled many of the study's absurd premises and assumptions. As soon as those of us working on #MEAction read the first article in the series, we felt it had the potential to finally break through the seemingly impenetrable wall protecting the PACE trial from the valid criticism that knowledgeable patient-researchers had been making for years. The #MEAction petition with more than 12,000 signers asking for retractions of the misleading claims made by the PACE team got its inspiration from the series, and was eventually delivered to the Lancet. We worked with knowledgeable volunteers to summarize the articles and to gather signatures from dozens of researchers and clinicians on Virology Blog's open letter to the Lancet last year demanding an independent investigation of the trial.

David has continued to investigate different aspects of the PACE debacle, as well as related studies like FINE and FITNET, largely as an unpaid "public service" project. David recently learned that his contract with UC Berkeley is not being renewed due to budget cuts. This means he cannot continue to rely on his earnings from his academic position to cover his bills while he continues to dig into the PACE trial and fight for a much-needed retraction of the papers based on the trial. To help support his work, the School of Public Health at UC Berkeley has agreed to create a new position "focused on investigating the PACE trial and other issues related to ME/CFS," financed by his crowdfunding campaign. The Centre for Scientific Integrity, a non-profit that publishes the site Retraction Watch, has agreed to serve as fiscal sponsor for this effort, which means all donations will be tax deductible. The campaign's goal is to raise $60,000 to fund the position.

For further information please refer to the source listed at the beginning of this article.

Columbia & Simmaron gut study

Source: http://simmaronresearch.com/2017/04/chronic-fatigue-syndrome-microbiome-gut-subset

Ian Lipkin and Mady Hornig's Centre for Infection and Immunity (CII) and collaborator Simmaron Research have used the latest technological advances to possibly find a sub-set of ME/CFS. The new study combined microflora, metabolic and immune analyses in fifty chronic fatigue syndrome (ME/CFS) and healthy controls from four clinical sites (Dr. Peterson, Dr. Lucinda Bateman, Dr. Nancy Klimas and Dr. Susan Levine). The goal was to take the deepest look yet at gut bacteria and their effects on metabolic pathways and the immune system.

All chronic fatigue syndrome (ME/CFS) gut studies to date have used a process called 16 S rRNA sequencing to characterize the gut microbiome. Unfortunately this process, which focuses on one section of the bacterial genome, is unable to differentiate approximately 40% of the species within each bacteria genera. Because different primers can also produce discordant results, results of 16 S rRNA studies can also vary from study to study.

These studies have been valuable; they've have indicated that something is off in the ME/CFS patients guts, and have given us some idea about the bacterial species involved, but because they can't differentiate between some of the helpful or harmful species in a genera, they lack specificity.

Enter Ian Lipkin. It's perhaps no surprise that technological ace Ian Lipkin would be the first to produce a study that really gets at gut species in ME/CFS. (Lipkin has invented several viral identification tools). Lipkin used a more expensive tool called metagenomic sequencing which analyses the entire genome. It has even been used to identify species new to science.

Lipkin's ME/CFS study identified more than 350 bacterial species. How cutting-edge Lipkin's approach was showed up when I asked him if finding 350 species was unusual. He said he couldn't say; the technique hasn't been used enough in other diseases to tell. He was confident, though, that the species the study identified were correct.

The study indicated that the guts of people with chronic fatigue syndrome (ME/CFS) were harboring a significantly different flora than the healthy controls. As in other studies, the relative abundance of species from one phylum (Firmicutes) chiefly defined the ME/CFS.

Moving from the top down, topological analyses and prediction models found that the relative abundances of seven bacterial genera (Faecalibacterium, Roseburia, Dorea, Coprococcus, Clostridium , Ruminococcus, and Coprobacillus) differentiated ME/CFS patients from healthy controls as well.

Getting into the species level, four gut species in particular (C. catus, P. capillosus, D. formicigenerans , and F. prausnitzii) and four others (C. asparigiforme, Sutterella wadsworthensis, A. putredinis, and Anaerotruncus colihominis) mainly differentiated the ME/CFS patients from the healthy controls.

Thankfully, the study's general conclusions jived with the results of past ME/CFS studies which also found reductions in Faecalbacterium and increases in Alistepes bacteria.

Whether they had IBS or not, chronic fatigue syndrome patients had a different microbiome than the healthy controls. Topological analyses, however, indicated that having IBS, changed a great deal.

The relative abundance of four bacteria (Faecalibacterium species, R. obeum, E. hallii, and C. comes) were lower in the ME/CFS + IBS group than the ME/CFS – IBS group. One bacteria (D. Longicatena) that was increased in ME/CFS patients – IBS, was actually decreased in the ME/CFS + IBS patients. This appears to suggest that ME/CFS patients with IBS specialized in having lower abundances of "good bacteria".

Encouragingly some of those same bacteria are low in IBS studies. Low levels of these protective bacteria have been associated with gut hypersensitivity, bloating and discomfort in both animal and human studies.

That suggests that having inadequate levels of these bacteria may result in inflammation which attacks the gut lining and allows bacteria to escape to the blood. Once in the blood the bacteria are believed to trigger a systemic immune response that may be able to affect the central nervous system. Evidence of leaky gut has shown up in several ME/CFS studies.

For the rest of the article please refer to the source. The conclusion is included below…

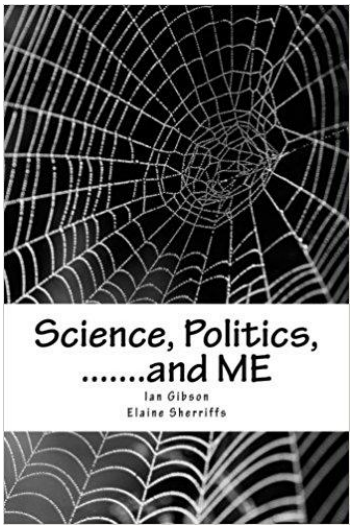
Conclusion
The Centre for Infection and Immunity was able to distinguish ME/CFS patients with and without IBS from healthy controls using analyses of their gut flora. Underlying alterations in gut flora were common to all ME/CFS patients but having IBS as well had a major effect on the gut flora and possibly on ME/CFS patients' metabolism.

Using a technique that was better able to identify more gut species than past studies, the group found marked differences not just in the gut flora of ME/CFS patients with IBS but in the metabolic pathways those differences are believed to effect. Problems with ATP production and the urea cycle might be more associated with ME/CFS + IBS patients while problems with fatty acid metabolism appear to be common to all ME/CFS patients. The study suggested that infectious gut illnesses might be common triggers of both ME/CFS and IBS.

https://www.healthrising.org/blog/2017/02/28/biomarker-aussies-chronic-fatigue-syndrome/

Science, Politics, ……..and ME
A new book by Dr Ian Gibson

Source: www.investinme.org/IIME-Newslet-1508-01.shtml
Source: www.amazon.co.uk



Dr Gibson led an inquiry in 2006 without official funding and at a time when unbiased and independent analysis on the way ME was being treated and reported on, by the establishment organisations and media, was lacking. Dr Gibson provided a checkpoint which attempted to get publicity and change which would help ME patients.

The Inquiry's report made several recommendations, that the then Labour government ignored the report and its recommendations will forever cast a shadow on the health minister at the time and the government itself.

Since that time Dr Gibson has been influential in assisting 'Invest in ME' get high-quality biomedical research established in Europe. He has also chaired the 'Invest in ME' conferences.

Now, after:

- 10 years of 'Invest in ME' conferences;
- the tenth anniversary of the Gibson Inquiry; and
- when change is slowly managing to creep into establishment organisations;

Dr Gibson feels it is necessary to look at the effects that politics and the actions of some have influenced the way ME has been, and continues to be the subject of misrepresentation, inappropriate media reporting, ineffective research funding and a pervading prejudice that needs to be exposed.

The book will look at where we have come since that Inquiry and why.

The book
Few diseases can have been so maligned by false information, so manipulated by an insidious establishment-controlled ideology, or so poorly dealt with by those holding the purse-strings for research into the disease, than Myalgic Encephalomyelitis (ME). This book examines a scandal in our generation – a scandal still being played out by corrupt, apathetic, inept or ignorant attitudes in governments and Medical Research Councils and health services.

One of the authors (Dr Ian Gibson) in his 'Retirement' has written this book with a political friend (Elaine Sherriffs). Ian Gibson has a passing interest in the current political scene across the world and regularly speaks on these issues. When it comes to universal health, he has pointed out on many occasions that governments often ignore scientific evidence. ME, as described in the book, is a major problem where evidence is relegated to psychiatric explanations. It is a desperate need for scientists as far as health issues are concerned to look for biomedical evidence and ME is a major example.

This book describes the political manoeuvring which features just like those in the TV programme The House of Cards in the USA & the UK which described the games that are played in both parliaments.

He has previously addressed these problems in an early book in 1981 – called 'Class, Health & Profit'. Ian and Elaine have penetrated the murky world of politics which features in the world of ME. It is long past the time to treat this as a serious illness and the need for serious biomedical research. This will only come about when politicians and the media stop trivialising the illness. Science, Politics …… and ME is a book which will serve as a reference for the dark times, when patients were ill-served by the clash of interests between truths and untruths. It is also a book which comes at a time where a brighter future may be in the making for people with ME and their families.

The book is £7.99 from Amazon UK
www.amazon.co.uk/Science-Politics-ME-scandal-generation/dp/1543183786/ref=cm_wl_huc_item

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The doctors and Mr. Hyde: Amy Brown's ME enterovirus story

Source: www.prohealth.com/me-cfs/library/showarticle.cfm?libid=30350
By Cort Johnson

Byron Hyde M.D. is a Canadian physician who has focused his practice on chronic fatigue syndrome (ME/CFS) patients for the past 30 years. In 1988, he founded the Nightingale Research Foundation to "explore, understand and treat the patients disabled with Myalgic Encephalomyelitis, Chronic Fatigue Syndrome (M.E. and CFS), fibromyalgia-type illnesses and post-immunization injuries".

In 1992 he edited and published the 752-page The Clinical and Scientific Basis of Myalgic Encephalomyelitis and Chronic Fatigue Syndrome which in some ways has yet to get matched. He has an extensive database of over 3,000 ME/CFS/FM patients dating back over 20 years.

Missed Diagnoses and ME
His controversial hypothesis is that ME/CFS is made up of two classes of illness: a condition called myalgic encephalomyelitis (M.E.) caused by an enteroviral infection, and another class of illness – known as chronic fatigue syndrome – which represents a false diagnosis.

Hyde believes that M.E. patients are being mostly felled by enteroviruses belonging to a newly discovered branch of the enteroviral tree. (Other branches include the paralytic polio causing enteroviruses.)

Hyde, who is still practicing, is known for his extensive testing regimens. He asserts that people with true ME have a recognizable onset and distinctive brain signature that PET or SPECT scans will pick up. All the others almost invariably have another illness which testing, if enough of it is done, will pick up. (Some ME/CFS physicians will probably balk at that second conclusion…)

Hyde believes that a "large number of these reputed CFS patients have a treatable disease and with proper examination could be back to school and work." He's essentially agreeing that ME/CFS is a wastebasket diagnosis. He differs from others who've used that term in that he believes that people with ME/CFS are really do have an illness – just not ME/CFS.

It's a stark thesis, one which one commenter pointed out, would, if taken to its logical conclusion, end research into this "disease" (!). (It would also boost research into enteroviruses and "M.E."). It also appears to leave aside the possibility that the comorbid diseases associated with ME/CFS might have a common core.

Hyde's thesis presents an opportunity and a problem. If he's right then a significant subset of "ME/CFS" (how large is unclear) have a disease that may be able to be treated, if they can only get diagnosed.

Of course, some people with chronic fatigue syndrome (ME/CFS) and/or fibromyalgia see many doctors and go through years of expensive testing to no avail. Others, perhaps in the majority, get more limited testing done. The difficult question, given the expense of getting tested in the U.S. and elsewhere, is when is enough testing enough?

Hyde said it's impossible to say how many people diagnosed with chronic fatigue syndrome actually have M.E. or vice versa. He does provide some ways to determine what group he thinks you're in, though. Since enteroviral infections generally occur from June to November with a blip at Christmas, and often (but not always) occur in epidemic form (i.e. other people are getting sick around you), if your illness didn't start then, then you probably don't have M.E.

A SPECT or PET scan done according to his protocol will then confirm or deny ME. If you don't have ME you should, he believes, get more extensive testing done. He described his findings from some recent "chronic fatigue syndrome" patients in an email.

I just got off the line with a US patient I was seeing on SKYPE/FACE TIME: he has been diagnosed with CFS, but in reality he has Hughes Syndrome (antiphospholipid syndrome). This is a very nasty genetic illness and his grandmother had a diagnosis of lupus. Dr. Hughes from St Thomas Hospital in London only first discovered the disease after 1985. So it is understandable it was missed for his grandmother who may have had Hughes Syndrome and not lupus.

Yesterday I saw a lovely teenage girl who looked like she was going to melt in front of me and was totally incapable of standing. She had been ill for 5 years and it was like her muscles were made of ice melting under a hot sun. She had not only severe POTs, missed by over 10 physicians, she also had one of the most classic cases of Ehlers Danlos with her carrying angle bent backwards by 220 degrees, (also missed), and I have only started to examine her.

On examination she also had a significant heart defect that had been totally missed. She's been Ill for 5 years and no one had done a cardiac workup.

Two weeks ago another CFS patient was here for the first time and she had a missed heart disease and she has never been examined.

When I asked if any missed diagnoses in particular stood out, Dr. Hyde said…

Cort, there are so many it is ridiculous. I would have to go through a few hundred or even thousands of patients to adequately answer that question. He suggested though, that one area that people with gradual onset might want to focus on is the heart.

One thing is certain, any patient with a diagnosis of acute onset or gradual onset CFS should have a complete cardiac assessment including, since most of these things can be either corrected or cured: example: (1) ECHO, (2) 48 HR Holter when they are told to run up steps, stand at attention for 10 minute without moving, go to a shopping centre (all these things will have an effect on the Holter(3) Stress ECG on a tract or bike, (4) Carotid Doppler. Up to 10% will have a significant missed cardiac illness.

Another recently emerging possibility is hepatitis B immunisation:
I am also a very pro immunization physician but recombinant hepatitis B immunization is another matter. I have had 3 deaths and over 100 cases who have chronic, chronic illness following within one week of the second or third immunization with RHB. If you don't know this, these people are called CFS.

The basic problem, Dr. Hyde believes, is that most doctors are not curious enough. The biggest problem with a chronic fatigue syndrome diagnosis is that for most doctors it ends the search. Either the doctor believes the illness is mental and sends them to a psychiatrist/psychologist, or doesn't know what to do with it and discharges the patient, leaving them on their own.
What is more the problem is how physicians examine patients. The lack of physical examination and history taking has become pervasive. They tend to have a set group of blood tests and if they are normal then you treat the patient with an anti-psychotic of which there must be many, many dozens.

I think all physicians, when they do medical school, should be obliged to read the 1990's book, On Fatigue by the incredible Italian author from the University of Turino, Dr. Angelo Mosso, one of the most beautiful books ever written on the understanding of the cause of fatigue.

Mosso devised a technique to open up the skull and insert water to study the shrinkage of the brain when abnormal fatigue set in. You can see this today if you do a circulating blood volume on real ME patients. They all have significantly decreased blood perfusion to the brain, some even as high as 55% decrease on SPECT circulation study.

Looking For Original Incline Village/Lake Tahoe ME/CFS Patients
Despite the fact that Dr. Hyde has been studying patients from the original M.E. outbreaks for years (he says 80% had enteroviral infections) he's never been able to talk to any of the original Incline Village / Lake Tahoe patients. Please contact him at the Nightingale Foundation if you're interested. There would be no charge for the consultation.

Hyde recently published a long and for many patients possibly quite familiar story of physician inattention which (a) outlines his concerns regarding the medical profession and (b) provides a dramatic backdrop for his assertion that undiagnosed enteroviral infections are rampant in M.E. Along the way, Dr. Hyde suggests that if the polio vaccine engineered decades ago had covered a few more enteroviral strains "M.E." would not exist, and worries that new enteroviral outbreaks in Asia may soon start producing more M.E. cases in the West would not exist either.

Check out "Amy's" story at:
www.healthrising.org/blog/2017/05/23/doctors-hyde-amy-browns-m-e-enterovirus-story

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.