

## **ME Exists: True or False?**

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**“If you repeat something often enough, you can perhaps make people believe it. What you cannot do is turn it from being false into being true”.**

This well-known adage was quoted by Robert Rowthorn in the Sunday Telegraph on 2<sup>nd</sup> July 2006 in an article exposing as false yet another claim by the UK Blair Government.

This adage being true, it ought to be the case that no matter how often the psychiatric and medical insurance lobbies (and the Governments with whom they connive) continue to deny the published scientific evidence about ME/CFS and to promote their own model of behavioural illness, their doctrine remains false.

And false it certainly is: experienced biomedical researchers regard ME/CFS as a multi-system autoimmune overlap disorder, not a mental disorder, and give it its place between lupus, diabetes type 1 and MS, referring to it as “AFS” which stands for autoimmune fatigue syndrome (see also Chronic Fatigue Syndrome: A Biological Approach edited by Patrick Englebienne and Kenny de Meirleir; pp 291; CRC Press 2002).

False though the psychiatric paradigm about the nature of ME/CFS is, it nevertheless pervades and indoctrinates all levels of society, especially medicine, social services, the judiciary and, in recent years, some of the ME/CFS patients’ support charities in the UK, with knock-on effects on sufferers and their families. The recent campaign by Action for ME (AfME) to engage Members of Parliament in a candle-lit vigil to save the Government “CFS” Centres by securing more funding for those Centres would seem to be a case in point, given that the Centres support the psychiatric fallacy about the nature of ME/CFS by imposing compulsory and often inappropriate behavioural modification strategies such as cognitive behavioural therapy and graded exercise regimes, even though the evidence continues to mount that these Centres are causing actual harm to unknown numbers of those they are supposed to be supporting (see Research into ME [RiME] information on Co-Cure, 18<sup>th</sup> August 2006: “RiME latest – NHS Clinics Condemned – Part 4” and see also [www.erythos.com/RiME](http://www.erythos.com/RiME) ).

As solicitor John Batt wrote in The Times Law Section on 11<sup>th</sup> July 2006: “False allegation ruins lives....doctors are always hostage to the knowledge that lurks in the future...it is not the legal system that is causing miscarriages of justice; it is medical opinions that are not as sound as they appear to be. If (a doctor) cannot back (his) opinion with research and laboratory results, the judge should openly rule that the opinion may be worthless. He should not allow statements like: ‘this theory is well-established’ or ‘doctors have known this for years’ ”.

Where is the evidence that supports allegations by these “Wessely School” psychiatrists (some of whom act as Government advisors and others as advisors to the medical

insurance industry) that people benefit from “adopting the sick-role” conferred by a label of ME/CFS? There is no such evidence. There is, however, abundant published evidence of the extensive losses, loneliness and extreme suffering endured by those with ME/CFS, as well as plentiful evidence of the extra burden forced on them in their fight for survival, both medical and financial.

Why is the existing evidence of biomedical anomalies in ME/CFS continually ignored by “Wessely School” psychiatrists, who claim that the only disorders that they will “accept” are those supported by “evidence-based” medicine? And yet their “de-conditioning” model of ME/CFS is not evidence-based, nor can it ever be so: it is a hypothesis that cannot be tested, let alone proven, which contrasts with the biomedical model of ME/CFS that is supported by respected literature of solid scientific evidence.

The psychiatrists involved with ME/CFS, however, have assumed control of the ME/CFS situation and they continue to reject the substantial biomedical evidence of serious organic pathology despite the fact that their own assumptions about ME/CFS have never been validated. The reaction of the UK authorities to the “Wessely School” view of ME/CFS seems grotesquely to reflect the South African government’s view of AIDS, ie. it can be cured by garlic and lemon juice.

Is it because of the far-ranging influence of “Wessely School” psychiatrists that there was such deceit and misappropriation of funds surrounding ME/CFS biomedical research in the US?

Is it because of the influence of “Wessely School” psychiatrists that funding applications to the UK MRC for biomedical research projects in ME/CFS are rejected in favour of massive and repeated funding for those psychiatrists? It is certainly true that Wessely et al have made ME/CFS unfashionable to the elite scientific community, who now see ME/CFS as a behavioural illness.

Is it because of the influence of “Wessely School” psychiatrists that both the UK Government and the US Centres for Disease Control (CDC) continue to deny appropriate and necessary investigations for those with ME/CFS? For example, as in the UK, the CDC continues to deny the need for SPECT scans that researchers have shown to be diagnostic in ME/CFS: “Some (ME)CFS researchers have observed apparent differences in the cranial blood flow between patients and controls (but) imaging tests should not be performed as a diagnostic technique for (ME)CFS” (see the post on 17<sup>th</sup> August 2006 on Co-Cure by Stephen Du Pre: “CDC’s Continued State of Denial about ME/CFS”).

It was in 1995 that nuclear imaging expert Dr Durval Costa published his brain-stem SPECT imaging paper which clearly demonstrated highly significant patterns of hypoperfusion in ME/CFS patients that were not evident in those with major depression or in age and gender matched controls. Despite countless requests for referrals to him by desperate patients, these were consistently thwarted by NHS consultants working in “CFS” clinics, including to our certain knowledge by Professor Tony Pinching (then at St Bartholomew’s Hospital in London, now medical adviser to AfME) who refused outright

to allow such referrals. Costa eventually gave up in the UK and moved to Portugal, where he is now Professor of Nuclear Medicine. Why have these important findings been buried for the last eleven years?

If the psychiatric lobby is 100% convinced that they, and they alone, are right that ME/CFS is not a serious multi-system organic disorder, why are they so opposed to biomedical research that, according to their own beliefs, would be negative, thus proving them to be right after all? Or is it simply that the psychiatrists want all the available research money for themselves?

It is a problem that is unlikely to be solved until there is a workable case definition against which patients need to be clinically examined and categorised correctly, and the establishing of such a case definition is the key to this whole argument.

Why do “Wessely School” psychiatrists not encourage research into the underlying cause of ME/CFS as any true scientist would? Why instead do they deny and denigrate instead of investigating and supporting patients? It is worth recalling that before the existence of diagnostic tests like MRI in MS and EEG in epilepsy and the discovery of dopaminergic pathways in Parkinsons, sufferers from these disorders were treated abysmally, so the diversion of funds for research that would address the aetiology of ME/CFS has historical precedence.

Is the refusal to fund biomedical research simply a case of wishing to curtail care costs and of protecting insurance company profits, or could it be that those who are unproductive but expensive to the State economy are expediently regarded as “useless eaters”? Many will be familiar with the book by Martha Gellhorn entitled “The Face of War” (first published in 1959 and re-published by Virago Press in 1986) about the Nuremberg Trials and the fact that the Nazis actually killed 275,000 of their own people who were old, feeble-minded or incurably ill, describing them as “useless eaters”.

If it is not a case of those with ME/CFS being “useless eaters”, is the unthinkable possible, namely that the remarkable (and documented) increase worldwide in incidence and prevalence of ME/CFS is in some way related to a bio-warfare agent such as borreliosis? The diagnostic picture would then be complicated by the overlap with diseases such as borreliosis, whose myriad symptoms are almost indistinguishable from ME/CFS.

Why, for example, was Elena Cook (the pseudonym adopted of necessity by the woman who was instrumental in organising the demonstration on 25<sup>th</sup> January 2006 outside Gresham College in London at Professor Simon Wessely’s lecture on the non-existence of Gulf War Syndrome) subsequently arrested and sectioned under the Mental Health Act, having received threats to her life and family? (see Co-Cure ACT: 17<sup>th</sup> July 2006: “Release of anti-Wessely lobbyist and Lyme disease researcher from psychiatric detention [UK]”. Her statement about her arrest can be seen at <http://www.lyme-rage.info/elena/statejun06.html>).

Could this have had anything to do with Wessely's known association with the US military or with the exposure of the apparent confirmation by the CDC that Lyme borreliosis is indeed a biowarfare agent?

(see "US Government Admits Lyme Disease is a Bioweapon" online at <http://www.indymedia.org.uk/en/2005/11/328067.html> and see also "Borreliosis / Lyme & M.E. in the United Kingdom" at <http://www.mesupport.bigstep.com/generic215.html> ).

As noted by Aaron and Buchwald et al (J Gen Intern Med 2001;16(1):24-31), it is the case that chronically fatiguing illnesses are associated with high rates of many other clinical conditions that cannot be attributed solely to psychiatric illness, and that patients with fatiguing illnesses may present a complex clinical picture that poses diagnostic and management challenges. However, whilst they would not be included in a case definition, the following abnormalities have been regularly documented in patients with ME/CFS: whilst the direction of causality of the high burden of co-morbidity remains unproven, such co-morbidity gives the lie to the psychiatrists' too-facile assertion that the greater the number of somatic symptoms, the greater the likelihood of somatoform illness.

Whilst correlation does not imply causality, it remains beyond reason that the existence of so many documented abnormalities in people with ME/CFS should simply be disregarded and denied, including the following:

- abnormalities of the central nervous system include abnormalities of brain cognition, brain perfusion, brain metabolism and brain chemistry; there is evidence of low blood flow in multiple areas of the brain; neuro-imaging has revealed lesions in the brain of approximately 80% of those tested and according to the researchers, these lesions are probably caused by inflammation: there is a correlation between the areas involved and the symptoms experienced; abnormalities on SPECT scans provide objective evidence of central nervous system dysfunction; there is evidence of a chronic inflammatory process of the CNS, with oedema or demyelination in 78% of patients tested; there is evidence of a significant and irreversible reduction in grey matter volume (especially in Brodmann's area 9) which is related to physical impairment and may indicate major trauma to the brain (which could also explain the low recovery rate); there is evidence of seizures; a positive Romberg is frequently seen in authentic ME/CFS patients
- abnormalities of the autonomic and peripheral nervous systems: there is evidence of dysautonomia in ME/CFS patients – see "Standing up for ME" by Spence and Stewart: Biologist 2004;51(2):65-70; according to Goldstein, ME/CFS represents the final common pathway for a multi-factorial disorder causing autonomic dysfunction
- cardiovascular dysfunction: there is evidence of haemodynamic instability and aberrations of cardiovascular reactivity (an expression of autonomic function);

there is evidence of diastolic cardiomyopathy; there is evidence of endothelial dysfunction; there is evidence of peripheral vascular dysfunction with low oxygenation levels and poor perfusion and pulsatilities; there is evidence of abnormal heart rate variability and evidence of abnormal orthostasis; there is evidence of abnormally inverted T-waves and of a shortened QT interval, with electrophysiological aberrancy; there is evidence of abnormal oscillating T-waves and of abnormal cardiac wall motion (at rest and on stress); there are indications of dilatation of the left ventricle and of segmental wall motion abnormalities; there is evidence that the left ventricle ejection fraction – at rest and with exercise – is as low as 30%; there is evidence of reduced stroke volume

- respiratory system dysfunction: there is evidence of significant reduction in many lung function parameters including a significant decrease in vital capacity; there is evidence of bronchial hyper-responsiveness
- a disrupted immune system: there is evidence of an unusual and inappropriate immune response: there is evidence of very low levels of NK cell cytotoxicity; there is evidence of low levels of autoantibodies (especially antinuclear and smooth muscle); there is evidence of abnormalities of immunoglobulins, especially SIgA and IgG<sub>3</sub>, (the latter having a known linkage with gastrointestinal tract disorders); there is evidence of circulating immune complexes; there is evidence of a Th1 to Th2 cytokine shift; there is evidence of abnormally diminished levels of intracellular perforin; there is evidence of abnormal levels of interferons and interleukins; there is evidence of increased white blood cell apoptosis, and there is evidence of the indisputable existence of allergies and hypersensitivities and positive mast cells, among many other anomalies, with an adverse reaction to pharmacological substances being virtually pathognomonic
- virological abnormalities: there is evidence of persistent enterovirus RNA in ME/CFS patients; there is evidence of abnormalities in the 2-5 synthetase / RNase L antiviral pathway, with novel evidence of a 37 kDa binding protein not reported in healthy subjects or in other diseases; there is evidence of reverse transcriptase, an enzyme produced by retrovirus activity, with retroviruses being the most powerful producers of interferon; there is evidence of the presence of HHV-6, HHV-8, EBV, CMV, Mycoplasma species, Chlamydia species and Coxsackie virus in the spinal fluid of some ME/CFS patients, the authors commenting that it was surprising to find such a high yield of infectious agents on cell free specimens of spinal fluid that had not been centrifuged
- evidence of muscle pathology: this includes laboratory evidence of delayed muscle recovery from fatiguing exercise and evidence of damage to muscle tissue; there is evidence of impaired aerobic muscle metabolism; there is evidence of impaired oxygen delivery to muscle, with recovery rates for oxygen saturation being 60% lower than in normal controls; there is evidence of prolonged EMG jitter in 80% of ME/CFS patients tested; there is evidence of greater utilisation of energy stores; there is evidence that total body potassium (TBK) is significantly

lower in ME/CFS patients (and abnormal potassium handling by muscle in the context of low overall body potassium may contribute to muscle fatigue in ME/CFS); there is evidence that creatine (a sensitive marker of muscle inflammation) is excreted in significant amounts in the urine of ME/CFS patients, as well as choline and glycine; there is evidence of type II fibre predominance, of scattered muscle fibre necrosis and of mitochondrial abnormalities

- neuroendocrine abnormalities: there is evidence of HPA axis dysfunction, with all the concomitant implications; there is evidence of abnormality of adrenal function, with the size of the glands being reduced by 50% in some cases; there is evidence of low pancreatic exocrine function; there is evidence of an abnormal response to buspirone challenge, with a significant increase in prolactin release that is not found in healthy controls or in depressives; there is evidence of abnormal arginine – vasopressin release during standard water-loading test; there is evidence of a profound loss of growth hormone; even when the patient is euthyroid on basic screening, there may be thyroid antibodies and evidence of failure to convert T4 (thyroxine) to T3 (tri-iodothyronine), which in turn is dependant upon the liver enzymes glutathione peroxidase and iodothyronine deiodinase, which are dependant upon adequate selenium in the form of selenocysteine (which may be inactivated by environmental toxins)
- defects in gene expression profiling: there is evidence of reproducible alterations in gene regulation, with an expression profile grouped according to immune, neuronal, mitochondrial and other functions, the neuronal component being associated with CNS hypomyelination
- abnormalities in HLA antigen expression: Teraski from UCLA found evidence that 46% of ME/CFS patients tested were HLA-DR4 positive, suggesting an antigen presentation
- disturbances in oxidative stress levels: there is mounting evidence that oxidative stress and lipid peroxidation contribute to the disease process in ME/CFS: circulating in the bloodstream are free radicals which if not neutralised can cause damage to the cells of the body, a process called oxidative stress: in ME/CFS there is evidence of increased oxidative stress and of a novel finding of increased isoprostanes not seen in any other disorder; these raised levels of isoprostanes precisely correlate with patients' symptoms (isoprostanes being abnormal prostaglandin metabolites that are highly noxious by-products of the abnormal cell membrane metabolism); there is evidence that incremental exercise challenge (as in graded exercise regimes) induces a prolonged and accentuated oxidative stress; there is evidence of low GSH-PX (glutathione peroxidase, an enzyme that is part of the antioxidant pathway: if defective, it causes leakage of magnesium and potassium from cells)
- gastro-intestinal dysfunction: there is evidence of objective changes, with delays in gastric emptying and abnormalities of gut motility; there is evidence of

swallowing difficulties and nocturnal diarrhoea; there is evidence going back to 1977 of hepatomegaly, with fatty infiltrates: on administration of the copper response test, there is evidence of post-viral liver impairment -- an increase of at least 200 in the copper level is the expected response, but in some severely affected ME/CFS patients the response is zero; there is evidence of infiltration of splenic sinuses by atypical lymphoid cells, with reduction in white pulp, suggesting a chronic inflammatory process

- reproductive system: there is clinical evidence that some female patients have an autoimmune oophoritis; there is evidence of endometriosis; there is evidence of polycystic ovary syndrome; in men with ME/CFS, prostatitis is not uncommon
- visual dysfunction: there is evidence of latency in accommodation, of reduced range of accommodation and of decreased range of duction (ME patients being down to 60% of the full range of eye mobility); there is evidence of nystagmus; there is evidence of reduced tracking; there is evidence of problems with peripheral vision; there is evidence that the ocular system is very much affected by, and in turn affects, this systemic condition.

The above list is by no means comprehensive but merely gives an overview of documented abnormalities seen in ME/CFS that can be accessed in the literature, as well as in the abstracts and reports of international Clinical and Research Conferences. Unfortunately not all are on MedLine. It is our intention to provide, with others, a comprehensive list of references for an article discussing the above abnormalities in ME/CFS to be submitted for publication.

By contrast, there is no credible evidence --- as distinct from belief and assertion --- of abnormal illness behaviour in authentic ME/CFS.

It is not known if, during the various stages of ME/CFS described by Cheney (for which see <http://www.meactionuk.org.uk/consideration.htm>) all patients with ME/CFS exhibit all the above mentioned abnormalities: this is unlikely to be known due to the prevailing dictum by dominant UK psychiatrists and their associates that it is neither “necessary nor appropriate” to investigate such patients other than by minimal screening (and if you don’t look, you certainly don’t find).

Instead, the UK policy is to compulsorily brain-wash those with ME/CFS into changing their rational belief that they are organically sick into the irrational belief that, once they “modify” their thinking, they are fit to work and to look after themselves.

Moreover, not only are no appropriate investigations to be performed on patients with ME/CFS, but those few NHS physicians who possess the integrity and courage to act in ME/CFS patients’ best interests and who go out of their way to help them are pilloried by their colleagues and in some instances are subjected to “disciplinary” proceedings following complaints to the General Medical Council by other doctors with known

allegiance to the pharmaceutical and medical insurance industries; some have been threatened; some have been warned off, and some have had their licence to practice medicine restricted, with the possibility of their being struck off the medical register being very real. Some have been forced to resign; others have had their NHS clinics closed down, with their patients being transferred to psychiatrists.

It seems that the powerful vested interests groups who now control the Establishment will tolerate no opposition, with the result that NHS doctors' freedom to practice medicine is increasingly proscribed.

With no hope of funding to establish a diagnostic test and with no will by the Royal Colleges or Government to formulate or accept an accurate case definition, the situation relating to ME/CFS in the UK cannot improve.

So many abnormalities have now been shown to occur regularly in cases of authentic ME/CFS that it is not only bad science to attempt to dismiss, ignore or deny a reality that can be scientifically measured, but to continue to do so must, as others have noted, border on the criminal.