

**Consideration of Some Issues Relating To**  
**The Published Views of Psychiatrists of**  
**The "Wessely School"**  
**in relation to their belief about the nature, cause and treatment of myalgic**  
**encephalomyelitis (ME)**

**with Appendices by Val Broke-Smith**

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## The Current Situation

A recent report from the House of Commons Select Health Committee looked at adverse clinical incidents, unexpectedly poor outcomes to treatment and failures in medical care and was critical of poorly-performing doctors and of the NHS complaints procedure. The Select Committee heard evidence and took representations from at least eight sources about myalgic encephalomyelitis.

A qualified medical practitioner is clearly entitled to his / her own professional opinion; when, however, that opinion appears to be elevated to the status of medical certainty in the absence of legitimate scientific evidence, and when the opinion held is contrary to the established scientific evidence (ie. when that opinion relies upon unsubstantiated beliefs about the nature, cause and treatment of a disorder), and when over-enthusiastic claims regarding the curative efficacy of a practitioner's preferred method of treatment are widely promoted on the basis of a medical practitioner's personal opinion, then problems can arise for patients.

In this post-Shipman era, continued self-regulation by the General Medical Council in relation to the issue of patient protection is the focus of attention.

Currently, there is widespread concern over the GMC's poor record of dealing effectively with unsatisfactory doctors, but people (whether medical or lay persons) who are perceived as "whistleblowers" still face an almost impossible task and are commonly ignored by those whose job it is to pay attention.

Concerning the disorder myalgic encephalomyelitis (ME), since 1987 there have been significant problems associated with a group of UK psychiatrists known colloquially as the "Wessely School" who are associated with Simon Wessely of King's College Hospital (Guys, Kings and St Thomas' School of Medicine and Institute of Psychiatry, London), where he is now Professor of Epidemiological and Liaison Psychiatry. Despite many representations of legitimate concern to official bodies, his power and influence seem to increase rather than to diminish. It is believed that Wessely was the prime mover behind the much-criticized 5 6 7 8 9 10 1996 Report of the Joint Royal Colleges on Chronic Fatigue Syndrome 11 (and that one of the co-authors of that report is his sister-in-law Elena Garraida, who is Professor of Child and Adolescent Psychiatry at St Mary's Hospital Medical School, London). That Report is deficient in mention of references to the organic basis of ME/CFS : although it cites 256 references, half are by the same or associated group of authors, with

10% being by Wessely himself; nine had not been published or reviewed 12.

Other psychiatrists prominent in the "Wessely School" are Professor Anthony David, also of King's College Hospital; Dr Michael Sharpe, formerly of Oxford but now at Edinburgh; Professor Richard Mayou of Oxford; Dr Keith Hawton, also of Oxford; Dr Tony Pelosi of Glasgow; Dr Stephen Lawrie of Edinburgh; Dr Peter White of St Bartholomew's Hospital, London; Dr Anthony Cleare of King's College Hospital; Dr Matthew Hotopf, also of King's College Hospital and Dr Steven Reid, Clinical Research Fellow at King's College Hospital. Other regular co-authors are behaviour therapists such as Alicia Deale and Trudie Chaider, who are also on Wessely's team at King's College Hospital.

From the BBC Panorama programme "Sick and Tired" on 8th November 1999, it seems that another paediatric psychiatrist who subscribes to the belief that children with ME/CFS should be treated by "active rehabilitation" is Dr Michael Prendergast, formerly of Great Ormond Street Hospital, London, and that Prendergast has used an experimental and scientifically unproven "active rehabilitation" regime for children with ME. The programme exposed the quite appalling treatment carried out by Prendergast and it revealed the harrowing stories of several families whose very sick children had been removed from their homes and locked away in "secure" psychiatric units where minimal parental access was permitted.

Following the Panorama programme, Harvey Marcovitch, a consultant paediatrician and Editor of Archives of Disease in Childhood, wrote an article in the British Medical Journal 13 stating "BBC's Panorama performed a hatchet job on Dr Michael Prendergast, previously a child psychiatrist at Great Ormond Street Hospital (who) uses active rehabilitation as a treatment for chronic fatigue syndrome....It's about time the (medical) profession hit back at those who are vilifying our colleagues".

Wessely rushed into electronic print to support Marcovitch 14, stating "I congratulate Harvey Marcovitch on his exposition used by some activists to hound those paediatricians who are prepared to consider that parents do not always act invariably in the best interests of their children"; referring to the BBC Panorama programme, he said "This was a particularly biased and pernicious account of an area where balance and reason are needed, not polemic and distortion.....Any parent who watched the one-sided Panorama programme might be forgiven for thinking that the management of CFS in children involves coercion and the Courts...."

This same Harvey Marcovitch was apparently head-hunted and now, with Wessely, is a member of the Chief Medical Officer's Working Group on ME/CFS (see later).

Perhaps unsurprisingly, Elena Garraida also wrote to the BMJ 15 stating "television can also fuel the fire of pressure groups bent on combatting and discrediting medical diagnoses and treatments support H.Markovitch's conclusions that defence societies should consider defending doctors who are defamed publicly. In addition, highly biased programs (sic) such as Panorama's are likely to scare families and deter them from seeking the best help available.."

The treatment of children with ME/CFS is disastrous, as shown in the Panorama programme; the presenter (Matthew Hill) confirmed to the present author that there were so many cases to chose from that his difficulty was in deciding which families to use for the programme.

There is no question that children with ME/CFS have been forcibly removed from their parents and home - this pressing issue was raised by consultant paediatrician Dr Nigel Speight at the Chief Medical Officer's Working Group (see later) in April 1999, who reported that the frequency of psychiatrists diagnosing Munchausen's Syndrome by Proxy now amounted to an epidemic: this was reported in the 1999 (Autumn) issue of Perspectives, (the magazine of the UK ME Association).

Speight also reported that there was enormous pressure on sick children to attend school, with mandatory involvement of a paediatric psychiatrist (and consequent rejection of input by a paediatrician).

There are over 400 young people between the ages of 5 and 25 in the Association of Youth and ME, all of whom are too ill to attend school or university.

As long ago as 1988, young people with ME were being subjected to psychiatric "distraction therapy"; the most well-known case is that of Ean Proctor from the Isle of Man, then a twelve year old boy who, against his parents' wishes and with no prior warning, was forcibly taken from his parents. A policeman was standing by and a Court Order had been obtained (which was supported - in writing-by Wessely).

Before being referred to doctors in London, Ean had been subjected to terrifying ordeals: his local doctors did not believe in ME so they devised activities which were designed to prove that the child's symptoms were simulated. One such "distraction therapy" involved taking the petrified child on a ghost train in the expectation that he would cry out in fear on 3rd June 1988 Wessely had written a letter saying that Ean's inability to speak was " elective mutism").

Ean's parents turned for help to the Isle of Man Tynwaid, whose report on the case reveals even more horrors (ref. Report of the Select Committee of Tynwaid on the Petition for Redress of Grievance of Robin and Barbara Proctor, 1,C April 1991). This official report states: "At the time, Ean

could not keep his balance, his legs were getting weak, his speech was much slower, he found it difficult to read and he could not keep his concentration. He could not feed himself because he could not move his arms; he could not stand. He was subsequently unable to speak". The report documents that during one admission to Nobles Hospital on the Isle of Man, whilst "paralysed, he was put in the swimming pool with no floating aids whatsoever. Mfs Proctor said that at this time, Ean could not move a finger and could not speak. Ean sank under the water" (page 14,3.15).

Regrettably, not much seems to have changed in the last twelve years as far as the treatment of children with ME is concerned. A comparison of the views of UK psychiatrists as set out in the joint Royal Colleges' report on CFS with an American report (Chronic Fatigue Syndrome: Information for Physicians. NIH, Public Health Services, US Department of Health and Human Services, September 1996) shows just how little the approach of psychiatrists of the 'Wessely School' has altered.

For example, the US report states on page 7 that it advocates a "supportive approach", whereas the UK report states that children may need to be forcibly removed from their parents, stating "CFS in children covers a broad spectrum of problems. Even Munchausens by Proxy Syndrome" (10.2).

The US report states on page 8 "the physician should work with the school to limit class time, if necessary, and to resume school attendance gradually", but the UK report urges "an immediate return to school" (page 31, 10.12)

The US report advises "Home tuition may be an alternative" but the UK report states "We discourage home tuition" (page 31, 10.12).

For over a decade, Wessely has claimed that ME and CFS are the same condition, and that this condition is psychiatric: despite the fact that serious concerns about the methodology and validity of his well-published views on ME and CFS have been published in mainstream international medical journals, and despite the fact that many of his papers have subsequently been shown to be gravely flawed (see later), the UK medical establishment and Government departments are continuing to turn a blind eye.

Editors of UK medical journals appear to afford psychiatrists of the "Wessely School" a seemingly open door to publish papers claiming a primary psychiatric aetiology for ME/CFS but appear regularly to reject submitted papers from other researchers showing the organic basis.

For Wessely to be permitted to promote his personal view of ME/CFS is notable, given that the World Health Organisation has formally and

definitively classified ME as a neurological disorder under Diseases of the Nervous System at section G93 (Other disorders of brain), sub-titled Postviral fatigue syndrome (G93.3) sub-titled benign myalgic encephalomyelitis 16, whereas fatigue syndromes are formally classified under Mental and Behavioural Disorders at section F.48 (Other neurotic disorders), subtitled Neurasthenia F.48.0, subtitled Fatigue Syndrome 17.

Wessely, however, believes that the WHO got it wrong about ME 18, writing in The Lancet.. :

"The inclusion in the tenth revision of the International Classification of Diseases (ICD 10) of benign myalgic encephalomyelitis as a synonym for postviral fatigue syndrome under Diseases of the Nervous System seems to represent an important moral victory for self-help groups in the UK ... The nineteenth century term neurasthenia remains in the Mental and Behavioural Disorders chapter under Other Neurotic Disorders ... neurasthenia would readily suffice for ME. Applying more stringent criteria for CFS in the hope of revealing a more neurological sub-group succeeds only in strengthening the association with psychiatric disorders. We believe this latest attempt to classify fatigue syndromes will prevent many people from seeing the world as it actually is."

Despite the fact that ME has been documented in the world medical literature as a nosological entity for over 40 years, Wessely proclaims that ME does not exist; he says it exists only because well-meaning doctors have not learnt to deal effectively with "suggestible patients" 19. He states that "ME is a description, not a diagnosis" 20, and asserts that ME is nothing more than a dysfunctional "belief " that one is ill 21, but that CFS is an operationally-defined (psychiatric) syndrome.

From the time that Wessely came to prominence in 1987, it can be seen from his publications that there has been no real change in his opinion about ME/CFS: despite the enormous body of published mainstream literature which has emerged in the last 13 years, and despite many international conferences on ME/CFS at which world experts announced significant advances in medical understanding of the complex organic nature of this disorder 22, Wessely is pursuing a relentless course, and the evidence speaks for itself 23. Psychiatrists of the "Wessely School" continue to ignore the findings presented at these international conferences by eminent ME/CFS scientists and clinicians which illustrate the organic aetiology of ME/CFS, preferring instead to concentrate on meetings which involve like-minded Psychiatrists (for example, the CIBA Foundation Symposium held in London on 12th-14th May 1992).

Psychiatrists of the "Wessely School" believe that ME and CFS and chronic fatigue are all interchangeable names for the same psychiatric condition, which as recently as September 1999 Wessely describes as a "functional somatic syndrome", equating it with such "medically unexplained

symptoms" as pre-menstrual tension; he believes that conditions such as ME/CFS should not be "dignified by their own formal case definition and body of research" 24. Wessely assiduously promotes his belief that these disorders are nothing more than "artefacts of medical specialisation" 25, urging that they should be dealt with by a form of psychotherapy called cognitive behavioural therapy (CBT), which is sometimes known as "brain-washing", as it aims to alter the way people think. It is used together with a programme of supervised graded exercise in which patients are obliged to continue exercising at specified times and to a pre-set level determined by the psychotherapist irrespective of symptoms, whether severe or not. Wessely also advocates the use of anti-depressants (whether or not depression is actually present, and in apparent contempt of the published evidence that anti-depressant therapy is unwarranted in ME/CFS, irrespective of whether depressive symptoms are present, because it does not lead to improvement in any area of the patient's functioning 26. The extensive evidence that Wessely promotes CBT is cited in volume 2 of Denigration by Design 27.

The joint Royal Colleges report on CFS advises Government Departments and NHS commissioning officers that no investigations need to be performed on those with a diagnosis of "CFS" 28 and in the Linbury Trust "research portfolio", the message is clear: this group of like-minded psychiatrists whose work on "CFS" is financed (to the tune of over 4 million pounds) by the Sainsbury-owned Linbury Trust are certain that psychotherapy and anti-depressants will control the patients' mis-attributions and that searching for causes is not only futile but may prevent recovery 29.

Substantial evidence 30 refuting the joint Royal Colleges' report has been put before the Chief Medical Officer in person (then Sir Kenneth Calman) and before the Presidents of the three Royal Colleges who were responsible for publishing it, but despite a valiant petition 31 signed by 12,500 people asking for this flawed report to be withdrawn, the Health Minister (then Baroness Jay) said it was a matter for the Presidents of the Royal Colleges, who declined to withdraw it.

There is an urgent need for the UK medical establishment and its regulators to address the continued ignoring by psychiatrists of the "Wessely School" of the overwhelming collective published evidence from international ME experts in America and Australia which shows that Wessely is simply wrong, and that his claim to be practising "evidence-based medicine" 32 is unsustainable. Of particular concern is the fact that the supposedly-independent Working Group on ME/CFS set up by the former CMO (Sir Kenneth Calman) is being funded by Wessely's friends and supporters in The Linbury Trust (not by the Department of Health); the Chairman is Professor Allen Hutchinson, Director of Public Health, School of Health and Related Research at the University of Sheffield, who

almost from the outset made it clearly known that he will not hear any criticisms of Wessely's work. Further, it has been publicly announced that over 3,000 reference papers which are to form the data base of this Working Group have been supplied by Simon Wessely from his own personal collection.

Although announced on 16th July 1998, this Working Group is not to report until June / July 2001, but at the Steering Committee meeting held on 22nd February 2000 at the NHS headquarters in Waterloo Road, London, there was no concealment of the Groups' aims, which were stated to be as follows:

there is no Government intention to pursue any epidemiological studies (even though on 10th January 2000 Professor Hutchinson had written to one member of the Working Group saying "I share your view that some clarification of the epidemiology would greatly help the commissioning and configuration of services for CFS/ME").

there is no Government intention to pursue any aetiology studies.

there is no Government intention to study sub-groups of ME/CFS if any such sub-groups exist (declared to be necessary by the 1994 Report of the National Task Force on ME/CFS 33, particularly the sub-group known to be chronically and severely affected).

there is no Government intention to review the problems over state benefits for those with ME/CFS.

there is no Government intention to pursue the need to change the name from CFS (favoured by psychiatrists of the "Wessely School" but which many believe is inaccurate).

the severity and chronicity of ME has to be played down: it was said to be much too expensive to bring any such severely affected sufferers to meetings, so that their view could be heard (as had been promised).

all the important work of the Key Group (eg. reviews of treatment, diagnostic criteria etc) is to be franchised out to the York Centre. (Author's note: This is the Centre for Review and Dissemination (CRD), which collaborates with a number of health research and information organisations across the world and is a member of the International Network of Agencies for Health Technology Assessment (INAHTA). CRD is a sibling of the Cochrane Collaboration, which is an international body set up to prepare a database to encompass the results of all clinical trials; this Cochrane database will form an internationally available meta-analysis of what the Cochrane Collaboration considers to be the most effective treatment / management in all medical disciplines. Its results will therefore become the definitive worldwide medical



database on all medical conditions. Simon Wessely is believed to be in charge of the section on ME/CFS. The CRD plays an important part in disseminating the contents of the Cochrane Reviews to the NHS. The Director of the Cochrane Collaboration is Dr Iain Chaimers, a longterm member of Healthwatch 34. The CRD at York University is believed to be run mainly by doctors in the "Healthwatch" group. Healthwatch is known to be funded by drug companies 35; in its literature, one of its clearly- stated aims is to oppose "Diagnoses that are misleading or false, or that may encourage unnecessary treatment for non-existent diseases" 36. It must be remembered that Wessely has had connections with Healthwatch from its inception in 1989: soon after the press launch, he was one of the leading campaign activists 37 and Wessely assiduously teaches that ME is a non-existent disease 38).

the overall view is that "we can't change medical opinion overnight". This shows an extraordinary lack of awareness that there is plenty of evidence that laboratories all over the world are coming up with findings which support the wholly organic pathoaetiology of ME.

It needs to be borne in mind, however, that such aims fit in with Wessely's own stated aim (which is to "eradicate" ME 39) and that Wessely himself is a member of the CMO's Working Group, as are Professor Elena Garraida from St Mary's Hospital, Dr Anthony Cleare from King's College Hospital (currently a Linbury Trust Fellow), Dr Peter White from St Bartholomew's Hospital, Dr Harvey Marcovich (referred to on page 3 above) and behavioural therapist Trudie Chalder, all of who could fairly be said to subscribe to the Wessely School ideology.

It also seems to fit in neatly with the Green Paper which is currently out for consultation until the end of March 2000 40, which is drawn so widely that if it is adopted as law, it will give psychiatrists far greater powers to enforce compulsory psychiatric treatment upon both adults and children: it proposes that psychiatrists will be able to drug people, including children (against the wishes of their parents) if they have "any disability or disorder of the mind or brain, whether permanent or temporary, which results in an impairment of mental functioning" 41. This reformed legislation might do away with the current safeguard which states that people can only be given treatment against their will if they show "seriously irresponsible or abnormally aggressive behaviour". It is a matter of utmost concern, because if this become law, it is going to give ideologists uncontrollable powers to implement their own personal ideals, notwithstanding the evidence that those ideals do not represent good science.

It is well-known that Wessely and Anthony David are trying to overturn the WHO formal classification of ME as a neurological disorder and to re-designate it as a psychiatric condition 42; for those who believe that they

have the prerogative to define reality, if this Green Paper becomes a White Paper which becomes law, then the potential consequences for people with ME /CFS are alarming.

It seems incomprehensible that so much of the world-wide evidence of the organic basis of ME can be so repeatedly dismissed, ignored or trivialised by psychiatrists of the "Wessely School", and that Government bodies and editors of UK medical journals accept so uncritically what Wessely feeds them but reject well-designed UK studies reflecting the organic basis of ME/CFS; quite certainly, eminent UK scientists and clinicians of great experience and expertise have had their papers rejected by UK medical journals, once on the grounds that there was insufficient interest in the topic. As a result, these UK experts have been forced to publish abroad, for example in The American Journal of Medicine. Wessely's influence is phenomenal (see volume 1 of "Denigration by Design?" 43); this influence may be unlikely to abate, given that in 1998 Wessely joined a Board of the Medical Research Council.

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## Footnotes

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as footnote (23) above

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as footnote (23) above.

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## 2. Evidence that psychiatrists of the "Wessely School" are wrong

In the UK, some of the most compelling evidence of an organic aetiology for ME/CFS is to be found in the work of The Chronic Fatigue Syndrome Research Foundation (formerly known as The Persistent Virus Disease Foundation) - see pp 269-271 of volume 2 (Update) of "Denigration by Design?". 44

It is also to be found in the work of Dr W.R.C.Weir, specialist in ME/CFS at The Royal Free Hospital, London (Coppetts Wood), who has demonstrated that changes in different immunological parameters correlate with particular aspects of disease symptomatology and with measures of disease severity, lending further support to the concept of immunoactivation of T-lymphocytes consistent with a viral aetiopathogenesis of ME/CFS 45.

It is to be found in the work of a team from Glasgow 46 which provides firm laboratory evidence demonstrating delayed muscle recovery from fatiguing exercise: these findings show convincingly that in ME/CFS, there is continued loss of post-exertional muscle power (giving an additional loss of power), with delayed recovery for at least 24 hours, whereas sedentary controls recovered full muscle power after 200 minutes. The findings of this Glasgow team are in line with the authoritative advice of Dr Paul Cheney on aerobic exercise (see later).

In the US, the evidence that psychiatrists of the "Wessely School" are wrong is found most notably in the research of Dedra Buchwald, Associate Professor of Medicine and Director of the Chronic Fatigue Clinic at the University of Washington; Nancy Klimas, Professor of Medicine at the University of Miami and Director of the Department of Immunology, VA Medical Centre, Miami, and Anthony Komaroff, Editor-in-Chief, Harvard Medical Publications, Boston: overall, there is extensive evidence demonstrating chronic, low-grade immune activation in ME/CFS (see both previous volumes of 'Denigration by Design?').

There is also the evidence of neurally-mediated hypotension in ME/CFS from Peter Rowe and Hugh Calkins of Johns Hopkins University, Baltimore 47, whose work approaches ME/CFS from the standpoint of autonomic dysfunction, finding that 96% of ME/CFS patients tested showed an abnormal drop in blood pressure in response to upright posture for five minutes, and that virtually all ME /CFS patients have their symptoms provoked by standing. These authors note the high prevalence of allergic disease among those with ME/CFS, observing that one would expect to find a mechanism by which allergic disease increases the activation of reflex neurally-mediated hypotension (via the discharge of mechanically sensitive fibres). More recent support comes from researchers in the Autonomic Dysfunction Center at Vanderbilt University Medical Center 48 who have identified a genetic defect in orthostatic intolerance (which the authors state bears many similarities to chronic fatigue syndrome); they have shown abnormalities and mutations in the norepinephrine transporter gene (which is the molecule that removes most of the released norepinephrine from the synapse; if it is not removed, when patients stand up, they suffer from a racing heart, nausea and dizziness).

Researchers in Phoenix, Arizona 49 have demonstrated an explanatory model of coagulation activation in ME/CFS and fibromyalgia (FM) - using five tests, including fibrinogen, prothrombin fragment 1+2, thrombin / anti-thrombin complexes, soluble fibrin monomer, and platelet activation by flow cytometry, they have shown low level coagulation activation from immunoglobulins as demonstrated by anti-B2GPI antibodies, allowing classification of ME/CFS/FM as a type of antiphospholipid antibody syndrome. This allows testing and monitoring for anticoagulation protocols in ME/CFS/FM patients.

(Author's note: Phospholipids are constituents of all tissues and organs, especially the brain. They are synthesized in the liver and small intestine and are involved in many of the body's metabolic processes. An anti-phospholipid antibody syndrome is a clinical disorder with recurrent arterial and venous thrombotic events, ie. blood clots forming in blood vessels and with decreased blood platelets. The heart, central nervous system and skin - dermal arterioles may be affected. There is a primary form, seen in patients without clinical or serological evidence of autoimmune disorder and a secondary form which is usually seen in the presence of lupus anticoagulant antibodies, ie. in association with system lupus erythematosus (known to have considerable overlap with ME/CFS/FM). This research is very relevant to the sub-set of those with ME who have vasculitic problems).

There is also the research of Leonard Jason, Professor of Psychology, DePaul University, Chicago, who has views which are distinctly different from the psychiatrists of the "Wessely School" 50. Jason specifies the need to clarify the confusion arising from the different diagnostic criteria,

which include the current UK (or Oxford) criteria which were drawn up by Wessely himself, together with other subscribers to the "Wessey School" 51 (see later).

A reasonably comprehensive source of published research articles on ME/CFS is available from The British Library (Health Care Information Service) from their Document Supply Centre at Boston Spa, Wetherby, West Yorkshire, LS23 7BQ (telephone 01937 - 546000); quarterly updates of abstracts are sent on payment of a moderate subscription. These current awareness topics (CATS) updates on ME/CFS date back to 1984.

In his Testimony before the FDA Scientific Advisory Committee on 18th February 1993, Dr Paul Cheney (Professor of Medicine at Capital University and one of the world's leading exponents on ME, which is known in the US as CFIDS, or chronic fatigue and immune dysfunction syndrome) testified as follows:

"I have evaluated over 2,500 cases .... at best, it is a prolonged post-viral syndrome with slow recovery. At worst, it is a nightmare of increasing disability with both physical and neurocognitive components. The worst cases have both an MS-like and an AIDS-like clinical appearance. We have lost five cases in the last six months. The most difficult thing to treat is the severe pain. Half have abnormal MRI scans. 80% have abnormal SPECT scans. 95% have abnormal cognitive-evoked EEG brain maps. Most have abnormal neurological examination. 40% have impaired cutaneous skin test responses to multiple antigens. Most have evidence of T-cell activation. 80% have evidence of an up-regulated 2-5A antiviral pathway. 80% of cases are unable to work or attend school. We admit regularly to hospital ... with an inability to care for self".

Comparisons have been made between the prevalence of ME/CFS and multiple sclerosis (MS) in the UK,<sup>52</sup> in the USA <sup>53</sup> and in Australia <sup>54</sup> and have been estimated to be three times, twice and equivalent respectively. Prevalence estimates for ME in Britain vary by a factor of 8: because he has broadened the case definition to include psychiatric disorders (see later) Wessely claims that there are over one million "CFS" sufferers in the UK, of which he says 75% have a psychiatric aetiology. " <sup>55</sup>

Little has been published about the cost of support services for ME/CFS in the UK because virtually none exist; the 1994 National Task Force Report on CFS/PVFS and ME <sup>56</sup> estimated direct NHS costs to be from #180 million to just over #1 billion per annum, depending on the choice of prevalence estimate, with total costs ranging from #879 million to nearly #16 billion.

Perhaps it is significant that Wessely repeatedly writes of the costs to the NHS of "medically unexplained symptoms" 57 and that he urges CBT for ME/CFS (which he claims is cost-effective 58 ie. it costs less than funding any research or performing nuclear magnetic imaging studies or providing essential respite care)

In the US (where there is no national health service), the cost to insurance services has been widely commented upon; in order of such costs, ME/CFS came second in the list of the five most expensive chronic conditions, being three places above AIDS.<sup>59</sup> The US government considers research into ME/CFS/CFIDS to be a top level priority and in 1995-6 voted \$11.8 million to this disorder, currently voting funds of about \$12 million per annum to it.

In the UK, psychiatrist Simon Wessely (adviser on ME/CFS to Government bodies 60) says ME does not exist, and sufferers only believe they are ill; he advises that state benefits should be withdrawn, and that people with this condition must be required to change the way they "perceive" their illness and must agree to exercise back to fitness and work.

Wessely pays scant regard to the impact of such profound illness, pain and relentless suffering with which those with ME have to live on a daily and even hourly basis, often with incredible but unremarked courage; he has carried out no research on the quality of life of those with ME, but others have. An American paper 61 found that the quality of life is particularly and uniquely disrupted in CFS, and that all participants related profound and multiple losses, including the loss of jobs, relationships, financial security, future plans, daily routines, hobbies, stamina and spontaneity, and even their sense of self because of CFS. Activity was reduced to basic survival needs for some subjects. The extent of the losses experienced in CFS was devastating, both in number and intensity. An Australian paper 62 found that patients with this condition had more dysfunction than those with multiple sclerosis, and that in ME/CFS the degree of impairment is more extreme than in end-stage renal disease and heart disease, and that only in terminally ill cancer and stroke patients was the sickness impact profile (SIP) greater than in ME/CFS.

Evidence that psychiatrists of the "Wessely School" are wrong is also to be found in the work of the Chronic Illness Research Foundation at Berkeley, California, where Dr Howard Urnovitz 63 has been working in the field of ME/CFS and autoimmune diseases for over 25 years and whose work has been published in the journal of the American Society for Microbiology and (in 1996) in Clinical Microbiology Reviews. Urnovitz has found that in such chronic diseases, the human genome is re-arranging itself in order to try and detoxify the plethora of chemicals to which it has been subjected and to fight off any new toxic exposures (cf. the work of Cheney below).

Urnovitz's work has demonstrated a fundamental breakthrough linking toxic exposure with these chronic diseases which manifest themselves sometimes years after the over-load of toxic exposure. This fits in with the American findings that the 2-5A RNase anti-viral pathway can be damaged by chemicals as well as by viruses.<sup>64</sup>

Significantly, Urnovitz has found segments of nucleic acid material or amplicons which are homologous to regions of a particular chromosome (22Q I 1.2), which means that these researchers have found a new mechanism of how viruses, bacteria and chemicals - and even radiation - interact with human chromosomes to create novel new proteins which have never been seen before and which are believed to form a missing link in chronic diseases such as ME, post-polio syndrome, Parkinson's Disease, multiple sclerosis and other autoimmune diseases.

Such findings are ignored by psychiatrists of the "Wessely School" but for those who claim expert knowledge of CFS, it is irrational for them to ignore the work of world-class experts on ME/CFS like Paul Cheney. In his Workshop and Case Study seminar given in February 1999 <sup>65</sup> Cheney discussed the various stages of CFS and then the clinical management, concentrating on the glutathione deficiency in ME/CFS, shown by glutathione markers in blood and urine, together with citrate elevation. Importantly, glutathione is an impressive anti-viral agent.

Cheney stated that he was constantly amazed at how complicated this disease is; he pointed out that there are three phases in this illness, and that there is an end-stage which is resistant to all therapeutic intervention, in that any intervention simply makes the patient even more ill.

He has found generally low red cell selenium values, with even lower values in white cells, especially in lymphocytes. If there is depletion of selenium, it will inevitably impact on the glutathione functional system, and knocking out glutathione is producing apoptosis (programmed cell death, linked to mitochondrial deficiencies), a finding which is well-documented in the international ME/CFS literature. <sup>66 67 68</sup> With low glutathione, chemicals or toxins can induce micro-organism replication rates, and immune-activation states also can induce the activation of endogenous microbes in the presence of glutathione deficiency. This could explain why in ME/CFS, one sees a lot of endogenous viral activation such as EBV, CMV, HHV6, mycoplasma incognitus, chlamydia pneumoniae, candida etc, as cytokines in excess stimulate these organisms, especially in the presence of glutathione deficiency.

Put simply, upon selenium depletion, glutathione synthesis is wiped out, resulting in rapid viral replication, causing energy loss and detoxification failure at the cell level. If glutathione deficiency drops low enough, the cells simply die an apoptotic death.



In the more advanced stages of ME/CFS, there is failure of the detoxification system at cellular level, so these people are vulnerable to the very lowest common denominator of the toxins to which they are exposed. If there is a glutathione defect, the patient is vulnerable to his/her own cell toxicity, especially in the portal circulation, as even normal gut ecology is too toxic when there is a glutathione defect. In this context, Cheney mentioned multiple chemical sensitivity (MCS); he described the case of one of his patients who was very sick; she had to wear a mask and was bedridden; she had to be wheeled around and had to be fed and bathed and dressed.

(Author's note: in the USA, MCS is formally recognised as a legitimate disability entitling those affected to protection under specific laws enacted to safeguard the civil rights of the disabled - it is formally accepted by the US Department of Justice, by the US Department of Housing and by the US Department of Education. MCS is also formally accepted by the FDA, who list it as an illness 69 but in the UK, Wessely denies the existence of MCS.<sup>70</sup> Indeed, Wessely states that such patients with what he calls "functional somatic symptoms" - and he includes those with ME/CFS and FM - are "generally viewed as an unavoidable, untreatable and unattractive burden" <sup>71</sup>)

Cheney stressed that it is important to recognise the three phases of ME/CFS, because each phase has to be dealt with differently. In phase one, the R-Nase L is significantly elevated (for about the first five years). After five years there is a progressive loss of this enzymatic upregulation, and by phase three, it is not seen anymore. This means that if R- Nase L activity is measured across the whole spectrum of this disease, it will be found to be high in some patients and normal or low in others. It is therefore not a diagnostic marker for the condition.

In phase two, there is a significant down-regulation of R-Nase L, so patients do not have that underlying protein synthesis disruption that R-Nase produces. However, patients in phase two cannot do as much as they could when they were in phase one (even though they were in fact more sick in phase one), but they are more limited and are still pretty sick. Phase two is primarily a toxicity issue, as the R-Nase activity knocks out the body's detoxification system, so patients start getting toxic.

In phase three, patients have no R-Nase activity but are now really locked into their boundaries and are limited by the damage done to deep brain structures, (particularly the hypothalamic region), by the loss of dynamic hormone responses necessary to meet the exigencies of life, and by damage done to the mitochondrial DNA, which Cheney believes is substantial. It is the loss of mitochondria and (most importantly) the loss of dynamic hormone response which causes the limitations so universally experienced by these patients. These patients are severely affected by their low dynamic response to any stressor, and it is this dynamic loss

(the hypothalamic injury) which is so limiting. That is the end-point of the disease.

Cheney then discussed various treatment/management approaches, noting particularly the importance of elimination diets and the problems caused by undigested food proteins coursing through the small bowel and by the permeability of the gut, resulting in undigested food antigens crossing into the blood stream and getting exposed to immune competent cells; Cheney said that at this point, "you're off to the races with this disease".

(Author's note: This entirely fits in with the findings of a urine test carried out in the UK at the University of Sunderland, which identifies IAG (indolylacroyiglycine - a tryptophan metabolise) and other small peptide peaks, which indicate a leaky gut which is associated with a compromised digestive system, leading to ensuing disruption of the central nervous and endocrine systems. This test is 95% positive in Gulf War Syndrome; it is 100% positive in low-dose organophosphate exposure, and it is positive in ME).

With regard to activity, Cheney notably advises "The most important thing about exercise is not to have them do aerobic exercise. I believe that even progressive aerobic exercise, especially in phase one and possibly in other phases is counter-productive. If you have a defect in mitochondrial function and you push the mitochondria by exercise, you kill the DNA".

(Author's note: This is the exact opposite of what psychiatrists of the "Wessely School" believe: Wessely urges patients to undergo exercise programmes, claiming that such programmes are beneficial and safe,<sup>72</sup> and that patients only have problems with their muscles because they are de-conditioned through lying around and that they should exercise back to fitness. When patients simply cannot do so, their state benefits are stopped,<sup>73</sup> as are their insurance payments,<sup>74</sup> largely because of advice from psychiatrists of the "Wessely School" to the DSS / Benefits Agency / permanent health insurance companies that if patients do not co-operate with psychotherapy aimed at changing their "dysfunctional belief" that they are ill, then they do not want to recover. Who would compel those with motor neurone disease or multiple sclerosis to "exercise back to fitness"? Who would condone the withdrawal of their state benefits and insurance policies when they simply could not do so?)

Cheney said that in phase three of ME/CFS, because of the injured brain, there will be things which these patients will never be able to do again and they will be locked into significant impairment. Psychiatrists of the "Wessely School", however, do not include for consideration such research findings, but confidently assert that ME/CFS and FM (and other syndromes for which medicine has not yet discovered the cause <sup>75</sup>) are psychiatric conditions.

For substantial illustrations of other research which psychiatrists of the "Wessely School" also ignore, trivialise or entirely dismiss, see both previous volumes of "Denigration by Design ?" 76

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Footnotes :

as footnote (23) above

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### 3. Tactics used by psychiatrists of the "Wessely School"

In their apparent desire to suppress dissemination of research into ME  
which does not accord with their own narrowly-defined parameters of  
psychiatric illness, these psychiatrists consistently use the same tactics: it  
is fair to say that by virtue of the sheer volume of his published papers,  
Wessely himself must be considered the prime proponent.

By their repeated and therefore apparently deliberate ignoring, dismissing  
or trivialising of the research evidence with which they do not agree,  
Wessely and his own psychiatric lobby misinform and mislead readers,  
both medical and lay, thereby influencing and manipulating the perception  
of ME/CFS which their readers will acquire. Such intellectual manipulation  
is achieved by outright selectivity which might even amount to deception,  
and by biased use of the available published referenced literature on  
ME/CFS, a technique of which Wessely especially is master on the grand  
scale. By using this particular tactic, these psychiatrists fail to provide a  
balanced overview of the available published evidence on the state of  
knowledge about ME/CFS and thereby seem to be attempting to remove  
discourse on the nature of ME from the scientific arena, but good science  
thrives on open and honest scientific debate.

By their insistence on the exclusion from their own (Oxford) revised case definition criteria of all physical signs which might indicate the neurological component of ME, and by their insistence that all cases of unexplained 'fatigue' of six months' duration must (since 1991) be included in the definition criteria, these physicians have diluted the critical definition of ME, because the deliberate inclusion of those psychiatric conditions which are known to be associated with prolonged "fatigue" obfuscates crucial case delineations. This is bad science. It is also bad science to focus only on the symptom of "fatigue" and on the cognitive dysfunction and altered sleeping patterns (complaints commonly found in psychiatric illness) and to ignore or dismiss as of no consequence prevalent problems found in ME such as the inability to stand unsupported, vertigo, nystagmus, dysequilibrium, double vision, ataxia, neuromuscular incoordination, a positive Romberg sign, photophobia, parasthesia, nausea, diarrhoea, frequency of micturition by night as well as by day; shortness of breath, Raynauds syndrome, hair loss, problems with thermodyregulation, cardiac arrhythmia, pancreatic exocrine insufficiency; headaches of a particular nature; problems with hypersensitivities to food and household chemicals, including medicinal drugs and anaesthetics and the cardinal features (which are almost impossible to overlook) of intense post-exertional muscle fatiguability, with vice-like myalgia and malaise, none of which equates with "tiredness" or even with "fatigue", whether chronic or not. In particular, the matter of pain management in ME/CFS needs to be urgently addressed, as it is inappropriate to convey to those with ME that merely be changing their beliefs about causation, their suffering will cease. The US Satellite (Teletraining) Physician Education Conference of September 1997 cautions doctors that they should not under-estimate the degree of pain which those with ME/CFS are experiencing.

Psychiatrists of the "Wessely School" seem to think that the standard of evidence required is different in the discipline of psychiatry: for example, they always quote extensive reference papers in supposed support of their published articles but with this particular group of psychiatrists, the impartiality of the references they cite needs to be scrutinised, because these psychiatrists often name just the lead author and perhaps two or three others and then write "et al". This is customary practice when listing medical references, but with this group, it conceals the fact that they are often simply citing themselves and their own papers. It used to be the case that editors of medical journals would permit no more than two or three self-references for an article. Seemingly, executive editors now make no stipulation about the number of self-references permitted, which automatically opens the door for bias and bad science and for those who are unashamedly self-promoters.

Psychiatrist and other subscribers to the "Wessely School" (most notably Wessely himself are highly selective in the patient cohorts they purport to

study. Wessely rarely includes in his studies and trials anyone who is too sick to get to clinics or anyone who is house - or bed bound. When patients become too sick to continue participating in studies, the authors merely claim a high drop-out rate; Wessely offers no follow-up. For illustrations, see previous volumes of *Denigration by Design?* 77

Wessely patronises, denigrates and mocks patients with ME/CFS, 78 thereby damaging them and their credibility in the eyes of others. This demeans patients' great suffering. For illustrations and examples, see previous volumes of *Denigration by Design?* 79 The harm which his views are believed to have caused to patients is incalculable.<sup>80</sup> Many patients have committed suicide, and details were put before the Chief Medical Officer in person on 11th March 1998.

Wessely rarely performs (and advises others not to perform <sup>82</sup>) laboratory or neuro-imaging tests which might reveal the very serious nature of this illness.

Wessely makes assumptions and takes for granted what still needs to be explained. He commonly generates his conclusions before he has generated the data to support those conclusions, for example, he claims that people with ME/CFS benefit from "adopting the sick role", <sup>83</sup> but adduces not a shred of evidence in support of such a claim, and he never evaluates the losses sustained by those with ME.

Psychiatrists of the "Wessely School" study patients with "fatigue" (which includes those with psychiatric illness) and then claim that their results relate to ME/CFS, when the literature plainly states that such results cannot be so interpreted. <sup>84</sup> These psychiatrists need to pay greater attention to how they use certain terms (ie. fatigue, chronic fatigue, chronic fatigue syndrome, post-viral fatigue syndrome, ME) because they are not interchangeable, and to treat them as identical or comparable misleads physicians and Government officers. Studies using mixed populations are not useful unless the researchers disaggregate their findings: research should not be summarized across studies using different populations (as was done in the Joint Royal Colleges' report CR54) because it is very misleading.

Psychiatrists of the "Wessely School" advocate the use of anti-depressants in ME/CFS <sup>85</sup> (despite the published evidence that they do not work <sup>86</sup> and might be harmful), which conveys to those who are over-eager to hear it the notion that the illness must be a psychiatric one. The evidence for psychological factors playing a role in the perpetuation of the illness is rapidly being contradicted: over-estimating the importance of psychological factors is a research error that has major implications for people's lives - for example, how other people (including their own family) treat them; how the medical system treats them; whether or not they can survive financially or even physically etc.

Wessely and his frequent co-author Michael Sharpe in particular seem relentless in their determined efforts to advise insurance companies that those with ME/CFS who are seeking payment of benefit under their policies should not qualify for such payment (on the grounds that "CFS" is not a permanent incapacity and that it is a psychiatric disorder which is amenable to psychotherapy). Moreover, some insurance policies specifically exclude payments for psychiatric conditions. One illustration of Wessely and Sharpe's activities in this field is that on 17 May 1995, both Wessely and Sharpe, together with their non-medical colleague and frequent co-author Trudie Chaider were the main speakers at a symposium held at The London Business School entitled "Occupational Health Issues for Employers", at which this trio advised employers how best to deal with employees who are on long-term sickness absence with "ME". Unsurprisingly, the advice presented consisted of informing employers and attendees that ME/CFS has also been called (quote) "the malingerer's excuse". Wessely spoke on the (quote) "myths" of ME and about the role which he believes psychology plays; Sharpe spoke about anti-depressant and cognitive behavioural therapy, and Trudie Chaider spoke about "Selling the treatment to the patient" and about increasing the sufferer's activity levels in order to achieve a graded return to work. Another speaker at this symposium was Dr John le Cascio, Vice President of UNUM, the UK's largest disability insurer. Currently, there is great concern about this insurance issue, and particularly about the enormous problems with UNUM (and with Swiss Life), to the extent that the All Party Parliamentary Group on ME has devoted entire meetings to the insurance issue, the most recent one being on 25th January 2000. Just three extracts from a copy of UNUM's Chronic Fatigue Syndrome Management Plan dated 4th April 1995 are significant:

"Diagnosis: Neurosis with a new banner"

"UNUM stands to lose millions if we do not move quickly to address this increasing problem"

"Attending Physicians - work with UNUM rehabilitation services or as an outside vendor in an effort to return the patient / claimant back to maximum functionality with or without symptoms"

Further information about this may be obtained from the Chairman of the All Party Parliamentary Group on ME, Tony Wright MP, via his parliamentary assistant Bradley Brady at the House of Commons, London SW1A 0AA, telephone 020 7219 4832. Of note is the fact that at the end of February 2000, a meeting was held at The Royal College of Physicians in London, attended by Simon Wessely and Michael Sharpe, at which Sharpe is believed to have informed Dr Charles Shepherd (medical adviser to the ME Association) that he was recommending to insurance companies that claimants with ME should be subjected to covert video surveillance.

The evidence is there - in the public domain - that Wessely makes frequent mistakes. This is not just careless research, because his errors are always in the direction of supporting his own theory about ME/CFS, ie. his errors are only in one direction. As pointed out by psychologist and research methodologist Dr Terry Hedrick from the USA in the Quarterly Journal of Medicine, 87 Wessely et al's paper in the Quarterly Journal of Medicine 88 is an example of the mischaracterization of the facts. Wessely et al summarized a wide variety of studies, drawing conclusions across seven studies which were based on different patient populations - from simple fatigue of 30 days to chronic severe fatigue of decades - and they used different diagnostic instruments and different definitions of improvement. They also used different timing of measures (eg. how the patients were prior to illness, at intake of the study, years after onset of illness and at final follow-up). Wessely et al did not assess the adequacy of the analyses performed. In some cases, they even left out findings from cited studies which were inconsistent with their own conclusions. Further, the studies cited by Wessely et al do not (as claimed by them) yield a consistent pattern between psychiatric disorder and poor prognosis. Moreover, in one cited study, an overwhelming majority of individuals categorised by the authors as "recovered" had rated themselves as only slightly more than half-way back to premorbid health levels.

As Hedrick makes plain, "Studies and review articles on psychiatric factors and CFS need to be subject to the same standards of scientific inquiry as studies investigating organic factors, lest the theoretical stance of the researchers / authors turns out to be the most powerful predictor of results ... Not only did the article fail to summarize the psychiatric literature accurately, it omitted discussion of the many avenues now being explored on the organic underpinning of CFS". (See also 3 above).

A further illustration can be found in the paper by Wessely's associate Alicia Deale.<sup>89</sup> Whilst Wessely's name does not appear as co-author in this particular paper, Alicia Deale is a behavioural psychotherapist who echoes Wessely's own views; as she cites at least thirteen references from the "Wessely School" in this short piece, Wessely's influence is unequivocal, so he may therefore be held accountable for the message conveyed by one of his own team.

Deale purports to describe how cognitive behaviour therapy helped "Clive" regain an active, fulfilled lifestyle but as so often with these particular workers, this paper is flawed at a very basic level, so Deale's conclusions fail to impress; given that this is a case study which they chose to publish as illustrative, one must assume that it is typical.



Inevitably, Deale promotes the party line: "Avoidance of exercise or activity and accommodating lifestyle to the illness are associated with poor outcome, greater functional impairment and more somatic complaints.... Catastrophic beliefs about the consequences of increasing activity have also been associated with greater disability and fatigue", but the causal direction of the relationship between avoidance of exercise and beliefs about the nature of the illness and about recovery are unknown. Even catastrophic beliefs could be due to people having had extreme relapses after trying to increase exercise.

As in Wessely's Quarterly Journal of Medicine article (where it is very clear that he was selective and inaccurate in summarizing the results of previous research on prognostic indicators of recovery), the identical problem exists here.

In this study, Deale greatly over-simplifies the results of CBT / graded exercise studies to date: the only studies finding positive effects have come from the British "Wessely School" - US and Australian researchers have not replicated the "Wessely School" results. 90

(Author's note: Fred Friedberg, Clinical Professor in the Department of Psychiatry at the State University of New York, makes the point that "Several studies of graded activity-oriented cognitive behavioural treatment for CFS, all conducted in England, have reported dramatic improvements in functioning and subsequent reductions in symptomatology. On the other hand, cognitive behavioural interventions conducted in Australia and the United States have not found significant improvements in functioning or CFS symptoms. Furthermore, descriptive studies of CFS patients in England, the US and Australia suggest that the CFS population studied in England shows substantial similarities to depression, somatization or phobia patients, while the US and Australian research samples have been clearly distinguished from depression patients and more closely resemble fatiguing neurological illnesses". Friedberg observes that because all the apparently successful CBT studies have all been conducted in England, a replication of those findings in a well-designed US study would be necessary before a general recommendation for CBT could be made).

Deale offers no explanation as to why she believed that a previously fit and active 34 year old carpenter with two children (who had been a regular runner) should become more rapidly "deconditioned" because of two weeks' flu than someone previously less fit and active - such a statement requires a credible explanation before it can be put forward as a valid reason.

Importantly, there is a major problem in this case study with the scoring of achievement of the patient's longterm goals. One of Clive's stated longterm goals was to work for 30 hours a week, but he never achieves

more than 16 hours a week, and this was working only for a friend, yet on a scale of 0-8 (with 8 representing maximum difficulty) Deale gave him a score of 1 at final follow-up (indicating almost total achievement of the longterm goal, even though this is barely over half the stated longterm goal). Not considered by Deale is that if Clive did not have a wife to look after him, he would be unlikely to achieve anything beyond taking care of his own daily living needs as a realistic ultimate goal.

Notably, whilst Deale chose to believe Clive's own statement at follow-up that he believed he would continue to improve, she chose not to believe his self-report at session 6, at which he was resigning himself to chronic illness - one has to ask what a longer follow-up time would have shown about goal achievement relating to returning to paid employment.

This article is potentially damaging, in that physicians need to be provided with a more accurate picture of what is really possible; misleading articles such as this could well result in doctors acquiring unrealistic expectations of what their patients are able to do.

The mark of good researchers is that they themselves point out the weaknesses and limitations of their studies, as well as plausible alternative explanations for their findings.

While the intent of publishing a case study such as this one is to provide a richer description of the author's advocated therapy than would be possible in other journals, if prior and current research are misrepresented in the process, clinicians will continue to be misled about the supposed efficacy of the treatment, with the real risk of causing harm to patients.

Further illustrations are plentiful, particularly in the joint Royal Colleges' Report CR54, where the authors mention a paper by Buchwald, Gallo and Komaroff et al (reference 128 in the Report) but dismiss it, stating "White matter abnormalities occur in a number of settings, and their significance remains to be determined", whereas the paper itself concludes that patients with ME/CFS "may have been experiencing a chronic, immunologically mediated inflammatory process of the central nervous system", and that the MRI scans revealed a punctate, subcortical area of high signal intensity consistent with oedema or demyelination in 78% of cases". This is a clear illustration of the biased and misleading personal interpretation of the available evidence presented by the authors of the joint Royal Colleges' Report CR54.

In that report, the authors mention a paper by Bombadier and Buchwald (reference 173 in the Report), conveying that this paper supports their own stance, whereas the paper itself actually states "The fact that the same prognostic indicators were not valid for the group with CFS

challenges the assumption that previous outcome research on chronic fatigue is generalizable to patients with chronic fatigue syndrome".

Another illustration is that the authors mention a paper by Sandman (reference 153 in the joint Royal Colleges' Report) in apparent support of their own view that the results of neuropsychological testing have been "inconsistent", but the paper in fact concludes that "the performance of the CFIDS patients was sevenfold worse than either the control or the depressed group. These results indicated that the memory deficit in CFIDS was more severe than assumed by CDC criteria. A pattern emerged of brain behaviour relationships supporting neurological compromise in CFS". One would never know this from the way the authors of the joint Royal Colleges' report deliberately downplay and manipulate their own representation of this important ME/CFS research.

Out of the many available, just one further illustration is included here: Deale, Chaider and Wessely run true to form in their paper on cognitive behavioural therapy in CFS 91, with their customary but unscientific use of terminology as interchangeable and with their failure to disaggregate their findings even though they drew those findings across studies using different populations, which produces misleading results but which supposedly support their own theory.

Yet again, these authors over-estimate the importance of psychological factors; they have produced confounded measurement leading to naive analysis. They advocate the use of anti-depressants, even though other researchers have found that patients are made worse by such medication.

Further, it is absolutely unethical to urge that a patient's belief that s/he has a physical illness be challenged as part of the treatment when there is no proof (but only the belief of this team) that patients do not have a physical illness.

Are these authors not aware (and as self-proclaimed experts on ME/CFS, they ought to be) that the US CFS Co-ordinating Committee has noted that organ donation by those with ME/CFS is not encouraged, and that in the UK, those with ME are not permitted to be blood-donors 92 and are to be considered permanently excluded from so doing? Patients with a psychiatric condition are not permanently excluded from being blood donors.

Wessely needs to be reminded again and again and again that correlation is not the same thing as causation, and that he should not over-interpret results as having more practical importance than those results warrant. To do so is not only methodologically flawed, but it contributes to the perception of the illness as one which can be cured if the patients would only try harder, when the international scientific evidence does not support such a belief.

The tragedy of poorly summarised research for ME/CFS is that it is seized upon by definers of public policy who have vested interests in reducing their costs (ie. governments, insurers, employers), and Wessely has had it pointed out to him many times that he should be more cautious about his claims, because he is doing harm, but he pays no heed, appearing to be quite certain that he is right. According to Dr Dorothy Rowe, "people who know absolutely that they are right are very dangerous".<sup>93</sup> There are case reports which indicate that a whole professional community may be unable to observe a problem with a calm professional eye, thus tending to delete or abolish the problem, even though existing professional knowledge indicates that a different professional approach is available.<sup>94</sup> Has this happened about ME/CFS? If so, is Wessely responsible in any way?

When he is confronted with his obvious errors, Wessely's first tactic is seemingly to threaten and bully and to try to intimidate those raising concerns about his published papers. In 1994 when he did not like the published criticisms <sup>95</sup> of his own published papers, Wessely threatened the UK distributors of the journal with legal action unless before distributing the copies, they physically tore out the article to which he objected. (He had no such power over the copies distributed world-wide from America). Intimidated by his threats, the UK distributors gave in to his bullying. UK subscribers to the journal were angry that a journal for which they had paid in advance had been defaced in the absence of an injunction. Another example is in a letter from Wessely dated 18th January 2000 to the Countess of Mar; this letter contains what many have interpreted as a thinly veiled threat, effectively implying that if the criticisms of his work do not stop, it will have an undesirable effect on ME/CFS sufferers by those (quote) "in high office".

When intimidation clearly cannot be used, Wessely then blames others: on one notable occasion he blamed his peer-reviewers for "allowing" his unequivocal errors to be published (which he did about the errors in his article The prognosis of chronic fatigue and chronic fatigue syndrome: a systematic review. Q J Med 1997.,90:223-233).

More recently, when his methodological improprieties were exposed <sup>96</sup> Wessely again publicly tried to deflect blame from himself for his own failure to observe rules to which he might reasonably be expected to adhere: he blamed the theft of a computer for his being apparently unable to locate the original data (author's note: there are stringent requirements by Ethics Committees about the need to ensure the keeping of data for a specified number of years, often 15 years) and he referred to being "attacked by gremlins" and then stated he found it "hard to believe how the usually infallible statistical reviewers at the BMJ could have

overlooked this"; he then went on to wonder if the blame could be transferred to "the production side" of the BMJ for his own very substantial errors in his earlier (1994) paper, about which Martin Bland, Professor of Medical Statistics at St George's Medical School, London, wrote that it "should not be allowed to remain in the literature to be cited uncritically by others".<sup>97</sup> It is known that Wessely wrote an unprofessional letter to Bland, objecting to the fact that Bland had not first contacted him. It is also believed that Bland let it be known that he would not be threatened by anyone.

In psychiatry, there is a condition known as "dissociation", which is the process whereby thoughts and ideas can be split off from consciousness. Is it conceivable that a similar process might underlie Wessely's apparent lack of awareness of the reason his work is rejected by ME/CFS sufferers and by experienced clinicians and scientists?

Apparently Wessely knows that he is hated, and this is on public record,<sup>98</sup> so on one level it would be difficult to believe that he was unaware of the impact of his work on the very people he is supposed to be helping, yet he claims to be hurt and upset by their reaction, and he purports not to understand why so many people are incensed and distressed by his views.

At a recent meeting at the National Institutes of Health in the USA in February 2000, Wessely said to an attendee (who was a former medical professional but who now has ME/CFS) that he couldn't understand why he should be criticised; the person to whom he said this then asked him a single question: "Don't you read what you write?"

Also, Wessely appears never to entertain the possibility that he might be wrong, or that his preferred explanation for ME/CFS cannot feasibly be sustained, and that if it cannot be sustained, the ramifications of his beliefs have enormously harmful consequences - medical, financial, practical, emotional - for so many severely sick people and their families and associates.

Perhaps tellingly, Wessely is now letting it be known that the wider the distribution of Denigration by Design?, the better he is pleased, because he claims to be getting so much sympathy from other doctors.

Whether or not he is able to comprehend the effects of his beliefs, there is an abundance of published evidence showing how Wessely's views have influenced others, not only in the UK but in Australasia<sup>99</sup> <sup>100</sup> <sup>101</sup> and America as well, as typified by the recent paper by Barsky and Borus from the USA.<sup>102</sup>

Published letters about this paper include the following: <sup>103</sup>.

"(Barsky and Borus) managed to omit hundreds of peer-reviewed articles documenting physiologic bases for illnesses such as the chronic fatigue syndrome ....Even the review of the psychological literature left out articles inconsistent with Barsky and Borus's speculations and sometimes inaccurately portrayed the research they included". (Terry E Hedrick PhD)

"I've never been able to determine how secondary gains that include financial hardship, social isolation and reduced quality of life can perpetuate illness behaviour" (James McSherry MB.CHB)

"Without any evidence of clinical experience treating (chronic fatigue syndrome), the authors lump it with several other syndromes and draw conclusions about treating [it] from articles written about completely different and distinct syndromes. ...They make no mention of several peer-reviewed articles that show distinct physiologic symptoms of the syndrome.....(the authors) claim that [CFS] has 'enough in common' with other syndromes for them to be lumped together. Since when was 'enough' a suitable quantification to pass peer review.?" (Kenneth Clemenger BS).

"The authors were careful to avoid many substantive articles on the chronic fatigue syndrome, even one published in Annals.....Barsky and Borus's article ignores volumes of references that refute their claims" (Alan Clemenger MD)

"The authors' discussion of the chronic fatigue syndrome was highly selective and clearly aimed at supporting the authors' hypothesis.....many of (their) arguments seemed to rely on generalizations, oversimplification and a theory-led blindness to individual differences....the authors were allowed to present opinion as facts ... and to ignore the many studies that undermined their hypothesis. Would such obvious bias be acceptable in obstetrics or oncology? Whatever the reason, the authors' lack of objectivity resulted in the publication of a poorly researched article that misrepresented the research and perpetuated myths. What happened to evidence based medicine?" (Ellen Goudsmit PhD)

Many of these valid criticisms could equally well apply to the work of psychiatrists of the "Wessely School".

Does deliberate, repeated misrepresentation of the known facts not amount to scientific misconduct? Some lawyers believe that it does, because patients are being harmed by such deception, and evidence of this was put before the Chief Medical Officer in person. 104

In a paper delivered on 26th February 1999 at the Alison Hunter Memorial Foundation lecture in Australia, Simon Molesworth AM QC delivered a passionate speech, which has been published. 105 Molesworth makes the

point -forcefully - that the generally inadequate response by the medical profession when dealing with patients with ME/CFS leaves physicians seriously vulnerable to lawsuits when it is established that those physicians were all too ready to dismiss ME/CFS as a somatisation disorder or as other psychopathology.

Molesworth has found that doctors associated with treatment for ME/CFS take the easy option of not pursuing on-going investigation of possible causes and that patients are not monitored adequately. Molesworth surmises that this response may be largely due to the influence of those encouraging a psychiatric diagnosis. The current favour for cognitive behavioural therapy biases the treatment of these patients because psychiatrists have come to dominate the ME/CFS scene.

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## Footnotes

as footnote (23) above

audio tape and transcript of Wessely's Eliot Slater Memorial Lecture: Microbes, Mental illness, the Media and ME: The Construction of Disease, London, 12th May 1994; copies available at cost price as per footnote (23) above

as footnote (23) above

as footnote (23) above

as footnote (12) above

as footnote (11) above

as footnote (23) above

Outcome and prognosis of patients with chronic fatigue vs chronic fatigue syndrome. Bombadier C, Buchwald D. Arch Intern Med 1995.-155.. 2105-2110

as footnote (23) above - copious illustrations are provided; see also footnote (11) above

as footnote (26) above

Chronic fatigue syndrome. TE Hedrick QJMed 1997..90.723-727

The prognosis of chronic fatigue and chronic fatigue syndrome: a systematic review. Joyce J, Hotopf M, Wessely S. QJMed 1997.90.-223-233

Treating Chronic Fatigue Syndrome with Cognitive Behavioural Therapy. Alicia Deale. Mental Health Care, December 1997.. vol 1:no.3..134-137

A Subgroup Analysis of Cognitive Behavioral Treatment Studies. Fred Friedberg. JCFS 1999: 5., 3-4:149-159. Also co-published simultaneously as 'Chronic Fatigue Syndrome: Advances in Epidemiologic, Clinical and Basic Science Research'. (ed) Roberto Patarca-Montero. Pub. Haworth Press Inc. 1999

Cognitive Behavioural Therapy for Chronic Fatigue Syndrome: a Randomized Controlled Trial. Alicia Deale, Trudie Chalder, Isaac Marks, Simon Wessely. Am J Psychiat 1997..154:3.406-414

Guidelines for the Blood Transfusion Services in the UK HMSO 1989:  
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 The professional historical error. A Levy. Arch Gen Psychiat  
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 'The views of Dr Simon Wessely on ME: Scientific Misconduct in the  
 Selection and Presentation of Available Evidence?' E Marshall; M Williams.  
 CFIDS Chronicle, 1994:14-18  
 Fatigue and psychological distress: statistics are improbable. Martin  
 Bland. BMJ 2000..320..515-516  
 ibid  
 Ill-defined notions. Ziauddin Sardar. New Statesman, 5th February 1999  
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 reality? Rona Moss-Moffis et al. JNNP 1996.60..474-477  
 Dr Simon Wessely: Prophet or Profit? (Dr) K.Joily. Meeting Place,  
 (ANZMES) Spring 1998: 10-12 (Journal of the Australia & New Zealand  
 ME Association)  
 Professor Simon Wessely 'Down Under'. Meeting Place, Autumn 1999  
 Functional somatic syndromes. Barsky AJ, Borus JF. Ann Intern Med.  
 1999:130..910-921  
 Letters: Functional Somatic Syndromes. Ann Intern Med. 1999..  
 132..4..327-330  
 as footnote (12) above  
 The Precautionary Principle, CFS Investigative Research and Patient  
 Support Regimes in Litigious Times. Simon Molesworth AM QC,BA,LLB,  
 FEIA,FAICD. Meeting Race Nov. 1999.58 (the journal of the Australia and  
 New Zealand ME Society)

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#### 4. Possible Misfeasance

Molesworth points out that in these litigious times, such an approach to ME/CFS is perilous and could lead to a spate of litigation for both misfeasance and nonfeasance: misfeasance could be applied where (inappropriate) treatment worsens the patients' suffering and nonfeasance applies where there is a lack of on-going monitoring and investigation of all the options, should it subsequently be proven that a more proactive approach may have brought relief earlier.

Molesworth advises that all options must be kept open so that the parameters of research are not narrowed by one particular discipline.

Simon Molesworth can be contacted on (Melbourne) 00613-9225-8571.

In conclusion, mention must be made of the February 2000 issue of The American Journal of Medicine: in an editorial commenting on a paper by



De Meirleir et al 106 in the same issue, Professor Anthony Komaroff states 107

"Many controlled studies have compared patients with chronic fatigue syndrome with aged-matched and gender-matched healthy control subjects, and with matched groups of patients with various fatiguing illnesses....several objective biological abnormalities have been found significantly more often in patients with the syndrome than in the comparison groups. The evidence indicates pathology of the central nervous system and immune system.

What is the evidence of central nervous system pathology? Magnetic resonance imaging has revealed punctate areas of high signal in the white matter. Single photon emission computed tomography (SPECT) signal abnormalities also are found more often in patients with chronic fatigue syndrome, abnormalities like those seen in patients with encephalopathy due to the acquired immunodeficiency syndrome (AIDS) and unlike the findings in patients with depression.

"Autonomic nervous system testing has revealed abnormalities of the sympathetic and para-sympathetic systems that are not explained by depression or physical deconditioning. 'Studies of hypothalamic and pituitary function have revealed neuroendocrine abnormalities not seen in healthy control subjects, and generally opposite to those found in major depression.

There is often a central down-regulation of the hypothalamic-pituitary-adrenal axis, resulting in a mild hypocortisolism, as well as disruption of both serotonergic and noradrenergic pathways."

(Author's note: see also the paper from scientists at Dundee 108 who have found evidence of an abnormality in cholinergic (muscarinic) activity in patients with ME/CFS affecting the blood vessels. Their results demonstrate the presence of a defect in peripheral cholinergic activity within the vascular endothelium; such disruption of microvascular integrity may provide an explanation for some of the vascular features such as orthostatic intolerance commonly found in ME/CFS).

"There is considerable evidence from different investigators, using different technologies and studying different groups of patients, of a state of chronic immune activation in many patients with chronic fatigue syndrome.

In this issue of The American Journal of Medicine, De Meirleir, Bisbal and their colleagues from Belgium and France report finding another immunological abnormality in these patients. Their work was prompted by a previous report from Suhadolnick and colleagues in the United States. Both the European and US teams studied an enzymatic pathway in

lymphocytes called the 2-5A pathway .... viral infection and interferon induced by viral infection turn on this enzymatic cascade, leading to increased levels of two polypeptides 2-5A synthetase and 2-5A-dependent ribonuclease L. The RNase L then selectively degrades viral RNA. Thus, viral infection elicits a compensatory antiviral effect through the 2-5A pathway.

Using a somewhat different technique, De Meirleir, Bisbal and their colleagues studied an entirely different and considerably larger group of patients ..... Like Suhadoinick et al, the European team found increased levels of the normal 80 kDa (kilodalton) and 40 kDa forms of RNase L in patients with chronic fatigue syndrome, as well as a novel low molecular weight form (weighing 37kDa). The ratio of the novel 37 kDa protein to the normal 80 kDa protein was high in 72% of the patients with chronic fatigue syndrome compared with 1 % of the healthy control subjects [Author's note: the Editorial does refer to 1%, but the paper itself states 11% of healthy controls] and none of the depression and fibromyalgia control patients, a striking and highly significant difference.

What is this research telling us? It is another piece of evidence that the immune system is affected in chronic fatigue syndrome, and it reproduces and extends the work of another investigator, lending credibility to the result.

The hypothesis is that the chronic infection leads to a chronic low-level "war", with the immune system attempting in vain to rid the body of infection. The on- going war leads to the production of various cytokines that cause the symptoms of the syndrome.

Finding aberrations in an 'antiviral' pathway....is consistent with the hypothesis that there is an underlying chronic viral infection.

In summary, there is now considerable evidence of an underlying biological process in most patients who meet the CDC case definition of chronic fatigue syndrome.

The report by De Meirleir, Bisbal and their colleagues is another strong piece of evidence that is consistent with the hypotheses that the immune system is activated and that the object of the immune system's attack could be a chronic infection.

Furthermore, the report is inconsistent with the hypothesis that chronic fatigue syndrome involves symptoms that are only imagined or amplified because of underlying psychiatric distress - symptoms that have no biological basis.

It is time to put that hypothesis to rest."

For the record, one prominent psychiatrist of the "Wessely School" stated as recently as February 2000 (ie. in the same month and just after the issue of The American Journal of Medicine mentioned above was published) that he and Wessely became involved in CFS research at the time it was attributed to a virus and when people were told to take prolonged rest, so he and Wessely were fighting the "rest is best" theory, and that they have "fought" those factors, as he and Wessely considered them to be potentially damaging to patients. This psychiatrist still thinks that illness beliefs have an important place as an obstacle to recovery in CFS.<sup>109</sup>

Also for the record, it should be recalled that Wessely himself declares (and has done so repeatedly for over a decade, despite the absence of evidence) that ME/CFS is not due to a virus: he believes that "there lies at the heart of CFS not a virus (or) immune disorder, but a distortion of the doctor-patient relationship" <sup>110</sup> and that management should focus on "perpetuating factors", which are listed as inactivity, illness beliefs, fears about symptoms, symptom focusing and emotional states. <sup>111</sup>

No matter how significant the evidence about the organic aetiology of ME/CFS, nothing seems to change in the "Wessely School": in December 1999, the second issue of a new journal called Clinical Evidence was published (by the BMJ Publishing Group with the American College of Physicians - American Society of Internal Medicine). It claims to be:

"a compendium of the best available evidence on the effects of common clinical interventions, bringing you the most reliable and up-to-date findings on important questions in clinical practice. It provides a concise account of the current state of knowledge, ignorance and uncertainty about prevention and treatment of a wide range of clinical conditions. Clinical Evidence can be used by anyone making decisions about patient care: Clinical governance leads, GPs, Hospital doctors, Hospital managers, Medical directors, Policy makers, Practice managers, Public health professionals.....It uses explicit methodology for selecting which evidence to summarise ..... [it] emphasises outcomes that matter to patients .... [and] provides guidance on applying evidence in practice".

In this major new compendium, the section on chronic fatigue syndrome (there is no mention of ME) is written by psychiatrists Steven Reid, Anthony Cleare, Matthew Hotopf and Simon Wessely, with input by Trudie Chaider (listed as a senior lecturer). There is absolutely no input by an immunologist, clinical allergist, neurologist, virologist, microbiologist, endocrinologist, paediatrician, pharmacologist, rheumatologist, molecular biologist, biochemist, biostatistician or by experts in general medicine, vascular medicine, nuclear medicine or physical rehabilitation.

The authors of the section on CFS cite eleven self-references, and other papers by subscribers to their own beliefs, one such being from the Cochrane Library (see page 9 above).

Unsurprisingly, major depression is not excluded from the stated diagnostic criteria.

The review highlights "key messages", which are presented in a style of instant impact and which include :

antidepressants may be useful (but up to 15% of participants withdrew from active treatment because of adverse drug effects)

a graded exercise programme can produce substantial improvements in physical functioning (author's note: the cited trial was complicated by a high withdrawal rate of 37%)

prolonged rest can be harmful

there is no clear evidence of benefit from magnesium injections or from oral evening primrose oil

there is no advantage in temporary immunotherapy, which can have serious adverse side effects

cognitive behaviour therapy (CBT) administered by highly skilled therapists in specialist centres is effective in CFS.  
Outcome is stated to be influenced by beliefs about causation.

Extracts from the above review in Clinical Evidence were reproduced in the BMJ of 29th January 2000:320:292-296; that version incorporates one additional intervention - located in the "unknown effectiveness" box - of oral nicotinamide adenine dinucleotide (NADH).

The omission of reference to mainstream documented neuroimmunological deficits is notable, given that the stated remit of Clinical Evidence is to "provide a concise account of the current state of knowledge" about "a wide range of clinical conditions".

Absence of consideration of effective therapeutic interventions and management other than psychiatric is explicable on the basis that currently there are no such protocols in existence for sufferers from ME/CFS. It must not be forgotten that those of the "Wessely School" advise Government and NHS commissioning officers that they see "no reason for the creation of specialist units" or for "specific guidelines on the management of CFS " to be issued to general practitioners, and advise that there is little point in looking at parameters of anti-nuclear factor, immune complexes, immune subsets or cholesterol levels. In the joint

Report CR54 112 the authors spell this out again, directing that "No investigations should be performed to confirm the diagnosis". If an investigative approach is curtailed as non-essential, one must ask what are the prospects for scientific advancement in medicine.

There are, though, signs of change: an article by Jeremy Laurant in The Independent on 16th March 2000 indicated the direction this change may take. Laurant states that he talked to a psychiatrist, "a specialist in chronic fatigue syndrome", who said that after a lifetime in the specialty, he had decided that psychiatrists were part of the problem. This "specialist in chronic fatigue syndrome" told Laurant that "A patient with chronic fatigue syndrome, who is resistant to a psychiatric explanation of his or her illness, is forced to maintain the illness in the face of psychiatric treatment to prove that it does not work".

Laurant reports that "This doctor's technique now is to act indirectly, advising the GP or neurologist on treatment but allowing them to take the lead, thereby affirming the patient's belief that they have a 'real' illness". One cannot but ask if it is ethical for such intervention to be administered 'indirectly' via a GP or neurologist without the patient's knowledge or informed consent.

It may be timely to quote from recent comments made by the Countess of Mar: 113 "I continue to pursue various avenues in the House of Lords. Gulf veterans, sheep dip victims and ME sufferers seem to take up much of my time. I continue to be concerned about the appalling way in which the Government treats these individuals and will not let go until they are treated as they should be".

Psychiatrists of the "Wessely School" (who are deeply involved in all three groups mentioned by the Countess of Mar) would do well to start practising what they claim to be practising, namely evidence-based medicine.

Despite all that has been written by these psychiatrists about the psychiatric status of ME/CFS, it is the case that neither ME, CFS nor chronic fatigue is designated as a mental disorder in the American Psychiatric Association Diagnostic and Statistical Manual of Psychiatric Disorders, 4th edition (DSM IV, Washington, 1994). Further, the criteria for somatisation disorder do not include chronic fatigue, chronic fatigue syndromes or ME.

"Functional somatic syndrome" is not a recognised category in DSM IV.

A spokesperson for the American Psychiatric Association has confirmed that there are no plans to include ME/CFS in the next edition (DSM V). If these psychiatrists of the "Wessely School" continue to ignore what is now worldwide evidence, they will be universally seen to be unscientific; they

will continue not "to see the world as it actually is", 114 and the consequences will continue to be disastrous for those with ME/CFS. In the interests of science if not of humanity, this can no longer be tolerated.

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## Footnotes

A 37kDa 2-5A binding protein as a potential biochemical marker for chronic fatigue syndrome. DeMeirier K, Bisbal C et al. Am J Med 2000.,108:99-105

The biology of chronic fatigue syndrome. Anthony L.Komaroff Am J Med 2000. 108..2.-169-171

Enhanced sensitivity of the peripheral cholinergic vascular response in patients with chronic fatigue syndrome. Vance A.Spence Faisal Khan Jill JF Belch. Am J Med (in press)

Personal communication.

Chronic fatigue syndrome: an update. Anthony J.Cleare Simon C.Wessely. Update (Recent Advances): 1,0 August 1996.61-69 ibid  
ibid

as footnote (11) above.

Chairman's Comments: Newsletter of The Environmental Medicine Foundation, March 2000: page 4

as footnote (18) above

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Click [here](#) for Appendix 1

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## Appendix 2

### Recommended Reading

Research shows clear differences between subgroups of "chronic fatigue syndrome" (CFS).

### Chronic Fatigue Syndrome

The term CFS was coined in 1988 by Dr Gary Holmes of the CDC as a replacement for the term Chronic Epstein Barr Virus Disease (the name

used by some USA physicians until it was realised that EBV was not the only virus associated with this illness). It was based on a single symptom found in those affected by the 1984 outbreak of ME at Lake Tahoe, Nevada.

"CFS" has since become an umbrella term much favoured by certain psychiatrists, particularly those of the "Wessely School" and by others who find it a less challenging option.

The term "CFS" has given rise to much confusion, especially since Wessely et al broadened the definition criteria (Oxford, 1991) to include all categories of unexplained "fatigue" in the UK, it encompasses disorders other than ME, including undiagnosed hypothyroidism, masked depression, and disorders related to lifestyle and nutrition. In America, stricter criteria select a more homogeneous population, so some US studies on CFS are undoubtedly looking at true ME.

(For a more comprehensive explanation, see Appendix V to the original Denigration by Design?).

Despite Wessely's obsession with reductionism, on scientific grounds it is helpful and appropriate to consider the differences between sub-groups.

#### Myalgic encephalomyelitis (ME)

This is one specific subgroup of the many chronic fatigue or post-viral syndromes. It is a multi-system disorder and is primarily neurological (affecting not only the central nervous system but also the autonomic and peripheral nervous systems), with variable involvement of cardiac and skeletal muscle. There is also involvement of the liver, and of the lymphoid and endocrine organs. In CFS, the focus is on "fatigue", whereas in ME the focus is on post-exertional fatiguability.

Whereas the 1988 Holmes / CDC definition placed great emphasis on symptoms such as mild fever, sore throat and tender lymph glands (ie. glandular fever), the definition criteria of true ME include the following:

muscle fatiguability following minimal exertion, with prolonged recovery time

evidence of neurological disturbances (CNS + ANS + PNS)

evidence of impaired circulation

a marked variability of symptoms (from day to day and even from hour to hour)

an extended relapsing course, with a tendency to chronicity

an increasing sensitivity to drugs (at the Dublin International Meeting on CFS presented under the auspices of The World Federation of Neurology, 18-20 May 1994, Professor Charles Poser of the Department of Neurology, Harvard Medical School, and the Neurological Unit, Beth Israel Hospital, Boston, Mass., said this is virtually pathognomonic of true ME). Additionally, in The International Classification of Diseases, the World Health Organisation officially classifies ME as a neurological disorder (ref. G.93.3) whereas it officially classifies fatigue syndromes as "other neurotic disorders" (ref: 48.0).

It is therefore not an option for Wessely et al to seek to overturn such official classification.

Much of the work on the broadly-defined CFS has failed to find the type of abnormalities found in the more strictly-defined ME; this ought not to be surprising, given that the CFS criteria definition specifically does not require evidence of central nervous system dysfunction.

For those who look and who wish to see, there are clearly discernible differences between "CFS" and ME, most notably in the pattern of cognitive impairment; in the type and pattern of immune dysfunction; in the clinically unmissable circulatory impairment and in the endocrine abnormalities -- for example, in CFS there is usually a normal to low level of cortisol (Demitrack et al, 1991: "Evidence for impaired activation of the hypothalamic-pituitary-adrenal axis in patients with chronic fatigue syndrome." J Clin Endocrinol Metab.

1991:73:1224-1234) whereas in ME, researchers report cortisol levels which are normal to high (Hilgers & Frank, 1992: "Chronic fatigue immune dysfunction syndrome in 103 patients - diagnosis, test results and therapy" Zeitschrift fur Klinische Medizin, 1992:47.4:152-166: In German); Richardson 1995: ("Disturbance of hypothalamic function and evidence for persistent enteroviral infection in patients with chronic fatigue syndrome" Journal of Chronic Fatigue Syndrome 1995.1:2:59-66).

Indeed, Wessely himself also found mean salivary cortisol concentration to be significantly higher in patients than in controls, concluding that:

"These findings are at variance with earlier reports that CFS is a hypocortisolaemic state and suggest that in CFS the symptom of fatigue is not caused by hypocortisolaemia". ("Salivary Cortisol Profiles in Chronic Fatigue Syndrome" Barbara Wood, Simon Wessely et al, Biological Psychiatry. 1998:37.1-4).

Wessely states that his patients in this study fulfilled both the UK and CDC criteria for CFS and that they had no history of neurological,



cardiovascular or endocrine disease, so one wonders about the definition of his cohort.

Black 1 puts forward the observation that the immune system is turned on (or more appropriately, is not turned off) because of a hypothalamic defect in the synthesis and / or secretion of CRF. CRF mediates the central nervous system response to environmental, physiologic or psychological stress, thus an ongoing immune response results in elevated levels of corticosteroids, catecholamines and certain endogenous opiates.

For clarification, the following suggested additional reading has been listed in various categories.

#### A. DEFINITIONS of ME

Wallis A.L. An investigation into an unusual disease in epidemic and sporadic form in general practice in Cumberland in 1955 and subsequent years. University of Edinburgh, Doctoral Thesis 1957.

Ramsay, A.M. O'Sullivan E. Encephalomyelitis simulating poliomyelitis. Lancet. 1956;1:761-766

A. Melvin Ramsay. "Myalgic Encephalomyelitis and Postviral Fatigue States" 2nd edition, Gower Medical Publishing, London 1988. (1st edition 1986 entitled "Postviral Fatigue Syndrome -- The Saga of Royal Free Disease". Ramsay always said how much he regretted not standing firm about the title of the 1st edition, which he wished to call "Myalgic Encephalomyelitis", not "Postviral Fatigue Syndrome" - in the 2nd edition, he stood firm).

Dowsett EG, Ramsay AM. Myalgic encephalomyelitis - a persistent viral infection? Postgraduate Medical Journal: 1990.,66:526-530

Hyde, BM. The Definitions of ME / CFS. In: "The Clinical and Scientific Basis of Myalgic Encephalomyelitis / Chronic Fatigue Syndrome" Ed. Hyde, BM, Goldstein J. & Levine P. Pub: The Nightingale Research Foundation, Ottawa, Canada, 1992

Dowsett EG & Weisby PD. "Conversation Piece". Postgraduate Medical Journal: 1992;68:63-65

Hyde BM. "Are myalgic encephalomyelitis and chronic fatigue syndrome synonymous ?" MPWC: 1999:3-17

#### B. EVIDENCE OF VIRAL TRIGGER

The presence of enteroviral particles has been found in a significant number of muscle biopsies taken from ME patients. This was rare in healthy controls.

Enteroviral sequences have been detected in tissue samples taken from the hypothalamus and brain stem of a patient with ME. Such sequences were not found in samples from depressed patients who had not suffered from ME.

Innes SBG. "Encephalomyelitis resembling benign myalgic encephalomyelitis". *Lancet*: 1970.1:969-971

Gow JW, Behan WMH, Clements GB, Behan PO et al. "Enteroviral sequences detected by polymerase chain reaction in muscle biopsies of patients with postviral fatigue syndrome". *BMJ*. 1991;302:692-696

Bowles NE, Lane RJM, Cunningham L & Archard LC. "Persistence of enterovirus RNA in muscle biopsy samples suggests that some cases of chronic fatigue syndrome result from a previous, inflammatory viral myopathy." *Journal of Medicine*: 1993;24:145-160

McGarry F, Gow J & Behan PO. "Enterovirus in the chronic fatigue syndrome". *Ann Int Med*: 1994;120:11:972-973

Clements GB et al "Detection of enterovirus-specific RNA in serum: the relationship to chronic fatigue." *Journal of Medical Virology* 1995;45:156-161

#### C. ABNORMALITIES IN MUSCLE TISSUE

In a study of a fairly homogeneous population, 80% of the biopsies showed evidence of structural damage to the mitochondria.

A deficiency in the levels of carnitine and serum acylcarnitine have been found; researchers believe this may be involved in the muscular symptoms of ME. Abnormalities in muscle function have been found and do not appear to be related to inactivity. In people with ME, objective tests have found prolonged recovery rates following exercise.

Behan WMH et al. "Mitochondrial abnormalities in the postviral fatigue syndrome." *Acta Neuropathologica*: 1991;83:61-65

Majeed T, Behan PO et al. "Abnormalities of carnitine metabolism in chronic fatigue syndrome." *European Journal of Neurology*, 1995;2:426-428

Kuratsune H, Evengard B et al. "Low levels of serum acylcarnitine in chronic fatigue syndrome and chronic hepatitis type C, but not seen in other diseases." International Journal of Molecular Medicine: 1998;2:1:51-56

Lane RJM, Archard LC et al. "Muscle fibre characteristics and lactate responses to exercise in chronic fatigue syndrome." JNNP, 1998;64:3:362-367

D. THE FATIGUE REPORTED BY PATIENTS WITH ME AND STRICTLY DEFINED CFS IS VERY DIFFERENT FROM THAT EXPERIENCED BY THE GENERAL POPULATION. SCORES ON FATIGUE SCALE LIKE THOSE OF PEOPLE WITH OTHER NEUROLOGICAL DISEASES SUCH AS MULTIPLE SCLEROSIS.

Krupp LB et al. "An overview of chronic fatigue syndrome." Journal of Clinical Psychiatry, 1991;52:10:403-410

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E. EVIDENCE OF ON-GOING INFECTION AND IMMUNE ACTIVATION  
Many studies have found evidence of an overactive (up-regulated) immune system. The immunological changes documented in ME and in strictly-defined CFS are related to the severity of the illness and correlate with intensity of symptom expression. These immune changes are generally more common in the severely affected. The immunological changes are not the same as those documented in depression. Some symptoms of ME may be related to an inflammatory process: findings are consistent with the view that fatigue in ME could be due to cytokine production within the central nervous system.

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#### F. EVIDENCE OF HPA DYSFUNCTION

Research has revealed a number of disturbances in the function of the hypothalamic-pituitary-adrenal axis. Some of these are different from the abnormalities documented in patients suffering from depression - indeed, some are the exact opposite.

Symptoms indicative of autonomic nervous system dysfunction are not related to psychiatric disorder. Such symptoms cannot be explained by "de-conditioning".

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#### G. EVIDENCE OF HYPOPERFUSION IN BRAINSTEM

MRI scans have revealed abnormalities in up to 80% of patients. Researchers believe that these defects are probably caused by viral encephalitis. There is a correlation between the areas involved and the symptoms experienced. The number of defects are correlated with clinical status.

Abnormalities on SPECT scans provide further objective evidence of central nervous system dysfunction. Studies published to date show patterns of reduced blood flow which are markedly different from those documented in depression.

The results on SPECT have been replicated using PET.

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#### H. EVIDENCE OF PROFOUND COGNITIVE IMPAIRMENT

Neuropsychological tests on patients with ME / PVFS and strictly-defined CFS have revealed abnormalities which are consistent with organic brain disorder.

These deficits have been found in both community and hospital samples.

The deficits were not the result of psychiatric disorders, such as depression. Exercise has an adverse effect on cognitive functioning in ME/CFS.

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I. PSYCHIATRIC STUDIES: FINDINGS DO NOT SUPPORT A PSYCHIATRIC DIAGNOSIS

It should be noted that there is no evidence of maladaptive beliefs, nor of phobic avoidance of activity in patients with ME.

In contrast to claims made by the "Wessely School", other more rigorously controlled studies have found low rates of depression.

The depression experienced by patients with ME / strictly-defined CFS is different from that found in psychiatric patients and is closely related to the severity of other symptoms.

The fatigue is not due to a lack of motivation or effort.

Evidence indicates that most patients with ME / CFS do not spend the whole day resting, and that a number of coping strategies are used.

Longitudinal studies using appropriate measures have shown that patients' attributions to a physical cause do not affect outcome; moreover, research on patients with ME indicate that a belief in a biological cause is not associated with poor mental health.

Graded exercise (where activity is increased according to a pre-set plan irrespective of symptom severity) is not appropriate for all patients with ME / CFS: over-exertion can lead to relapse.

There has been no study assessing the effectiveness of graded exercise or cognitive behavioural therapy in ME or in strictly-defined CFS.

The documented links between CFS and psychiatric disorders may reflect the overly-broad diagnostic criteria and the researchers' choice of measures for assessing psychiatric morbidity.

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Note: In her paper in ME Today (BRAME) 1999:9: pp 27-31 entitled "Research into ME / CFS 1988-1998: Too much philosophy and too little basic science", Dr E.G.Dowsett (former President of the UK ME Association) states:  
"Owing to severe problems in obtaining any adequate funding and in securing subsequent publication for ME research outside the psychiatric remit in the UK, most basic scientific work is performed with difficulty and published abroad".

Dowsett observes:

"Previously reputable medical journals concur with therapies which compound psychological manipulation. A leading proponent of this approach has ensured that the very words of a leading article on this subject are now inscribed upon a wide variety of benefit agency, insurance, retirement and other official forms which doctors must sign on behalf of their patients."

"Compared with this bludgeoning of public opinion, the 'mass hysteria' allegation at the Royal Free Hospital seems little more than the mad buzzing of a demented fly."

One must never forget that the recipients of this aptly-described bludgeoning are many extremely sick and disabled human beings.

It is worth recalling that in his address to the 1999 Sydney, Australia ME / CFS Conference, Simon Molesworth QC pointed out that "Litigation for misdiagnosis is a reality", and he asserted that doctors are legally vulnerable if they dismiss CFS as somatisation disorder or as another manifestation of psychopathology.

It is hoped that if used as a compendium, the two volumes of Denigration By Design? will help to establish the prominent role played by Simon Charles Wessely in the dismissing of ME / CFS and related syndromes as

somatisation, despite the enormous body of published research which indicates that such a view is inappropriate, unproven and harmful.

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## Footnotes

Immune system - central nervous system interactions: effect and immunomodulatory consequences of immune system mediators on the brain. Paul H. Black. Antimicrobial Agents and Chemotherapy.. 1994;38..1:7-12.

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## Appendix 3

### Suggested Contacts

Dr Byron Hyde, convenor of the First World Symposium on ME held at the University of Cambridge, UK, April 9th-12th, 1990; adviser on ME to the Canadian Government and Editor of the major 724 page text book on ME entitled The Clinical and Scientific Basis of Myalgic Encephatomyelitis / Chronic Fatigue Syndrome, pub. The Nightingale Research Foundation, Ottawa, Canada, 1992. Contact address: 121, Iona Street, Ottawa, Canada, K1Y 3 MI. Telephone: 001-613-7298995.

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Professor Anthony Kornaroff, Editor-in-Chief, Harvard Medical Publications; Contact address: 10 Shattuck Street, Suite 602, Boston, MA 02115, USA. Telephone: 001-617-432-4714.

Professor Robert Suhadolnick Contact address: Department of Biochemistry, Temple University School of Medicine, 3420 N.Broad Street, MRB 412, Philadelphia, PA 19140. Telephone: 001-215-707-4607.

Professor Kenny de Meirleir, Professor of Medicine & Physiology, Contact address: Department of Human Physiology, Vrije Universiteit, Brussels, Belgium. Telephone: 0032-24774600. Home address: Stuivenbergbaan 89, Mechien 2800, Belgium. Mobile: 0032-75468761.

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Professor Dedra Buchwald, Assistant Professor of Medicine; Director, Chronic Fatigue Clinic, University of Washington. Contact address: Harbourview Medical Centre, 325 Ninth Avenue, Box 359780, Seattle, WA 98199, USA. Telephone: 001-206-731-3160.

Professor Leonard Jason. Professor of Psychology. Contact address: Department of Psychology, DePaul University, 2219 No. Kenmore Avenue, Chicago, IL 60614-3504, USA. Telephone: 001-773-325-2018.

Professor Malcolm Hooper, Professor of Medicinal Chemistry, University of Sunderland, UK. Contact address: 2, Nursery Close, Sunderland SR3 1 PA. Telephone: 0191-528-5536.

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