

Wessely's Way: Rhetoric or Reason?

Malcolm Hooper Margaret Williams

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malcolm.hooper@virgin.net

On 22nd March 2008 the Financial Times carried an item by Glasgow GP Dr Margaret McCartney (“If it’s in the mind, it’s still the real thing”) in which she stated that neither ME – to which she referred as “myalgic encephalitis” instead of the correct term myalgic encephalomyelitis – nor fibromyalgia (FM), nor repetitive strain injury (RSI) nor irritable bowel syndrome (IBS) “*has a clear pathological or biochemical abnormality*”. She went on to confirm: “*It’s certainly true that many doctors see these kinds of symptoms as an irritating and time-consuming diversion from ‘real’ pathology*”.

Unfortunately for ME patients in the UK, such comments are nothing new.

The person whose work has had most impact on their lives is psychiatrist Professor Simon Wessely, whose twenty-year published record on ME patients underpins such ill-informed comments, for example:

- “*The description given at the Mayo Clinic remains accurate: ‘The average doctor will see they are neurotic and he will often be disgusted with them’ ” (In: Psychological Disorders in General Medical Settings, ed: Sartorius et al; Hogrefe & Huber, 1990)*
- “*Blaming symptoms on a viral infection conveys certain advantages, irrespective of its validity (and) is beneficial to self-esteem by protecting the individual from guilt and blame” (In: Post-Viral Fatigue Syndrome. ed: James Mowbray and Rachel Jenkins. John Wiley & Sons, 1991)*
- “*It seems that ME sufferers prefer to feel they have a ‘real’ disease – it is better for their self-esteem” (Pfizer Invicta Pharmaceuticals 1992:4-5)*
- “*Patients with inexplicable physical symptoms are generally viewed as an unavoidable, untreatable and unattractive burden” (Brit J Hosp Med 1994:51:8:421-427)*
- “*Somatisation sufferers consume vast amounts of health resources for little benefit” (Clin Exp Allergy 1995:25:503-514)*

- *“The term ME may mislead patients into believing they have a serious and specific pathological process. Several studies suggest that poor outcome is associated with social, psychological and cultural factors”* (Joint Royal Colleges Report on CFS, October 1996)
- *“ME has never been fully accepted as a real condition, says Simon Wessely”* (The Guardian, 21st April 1998). Note that the World Health Organisation fully accepted ME as a real condition in 1969 and continues to do so
- *“It is only human for doctors to view the public as foolish, uncomprehending, hysterical or malingering”* (BMJ 2003:326:595-597)
- *“Science is indeed socially controlled, and so it should be”* (The Guardian, 1st March 2003)
- *“Functional somatic syndromes include chronic fatigue syndrome”* (Rev Bras Psiquiatr 2005:27:3). This is noteworthy, given that Wessely is on public record as stating: *“I don’t classify CFS as a somatoform disorder”* (Wessely Answers Questions. 10th April 2002: CAME).

From the above quotations, it seems there may be an explanation why doctors such as Dr McCartney are so misinformed.

However, not only does it seem that Dr McCartney has been careless over her terminology but it also seems she has not kept abreast of the medical science that has revealed the pathological and biochemical abnormalities now known to underpin these disorders.

Moreover, she claims that the recommendation for cognitive behavioural therapy (CBT) in the NICE Guideline on “CFS/ME” does not imply a psychological cause because *“behavioural treatments can be used to improve the quality of life of people who have diabetes, asthma, or cancer”*. This is undoubtedly so, but the key differences that seem to have been overlooked by Dr McCartney are that in those disorders, appropriate investigations and effective interventions are not ignored or proscribed and, importantly, behavioural therapy is an adjunctive and not the primary – indeed the sole – management recommendation as it is in ME/CFS.

Dr McCartney harks back to the much-criticised 1999 paper by psychiatrists Simon Wessely and Michael Sharpe in The Lancet (“Functional somatic syndromes: one or many?”: Lancet 1999:354:936-939) and she quotes with seeming approval Professors Wessely and Sharpe: *“The existence of specific somatic syndromes is largely an artefact of medical specialisation”*.

Apart from the Lancet article to which she refers, Dr McCartney will doubtless be aware of Wessely’s views on ME/CFS, fibromyalgia (FM), Gulf War Syndrome (GWS), the Camelford water poisoning catastrophe, the effects of chronic low-dose organophosphate

(OP) poisoning and the adverse effects of mobile phones, since Wessely has not been reticent in publicising his views. He is certain that such disorders do not exist and that people who claim to suffer from them are deluding themselves because, he says, they are actually suffering from a mental (somatisation) disorder which, to quote Dr McCartney, is “*the phenomenon of translating mental distress into physical symptoms*”. Wessely is certain that such symptoms are merely “*the modern preoccupation with the state of our environment*” and that they occur in “*a few individuals with pre-existing somatisation disorders (and are) then diverted to fall in line with the prevailing (“disease”)*. *Future investigations of environmental incidents should recall that social and cultural factors are as important as medical ones*” (The Legend of Camelford. Anthony S David and Simon C Wessely. Journal of Psychosomatic Research 1995:39:1:1-9 --- see below).

The denial of the very existence of such disorders has become Wessely’s trade-mark.

It was captured in the New Statesman almost a decade ago when in February 1999 Ziauddin Sardar wrote “Ill-defined notions”: “*When is someone sick, really sick? Who decides? By what criteria? The only thing that is certain is that you are only ill when someone says you are ill. Consider syndromes. Once this was a name for a collection of symptoms for which no clear cause had yet been found. Now it stands for a bunch of symptoms lacking even the security of certainty that they are actually there. Most notorious is ‘chronic fatigue syndrome’, known as ‘ME’. Horror stories abound of people whom the psychiatric experts considered just to be faking. The same can be said of Gulf War syndrome. Even though 400 veterans have actually died and some 5,000 are suffering from illnesses related to Gulf War syndrome, the syndrome does not officially exist. Wessely has been arguing that ME is a largely self-induced ailment that can be cured by the exercise programme on offer at his clinic. Recently he published the results of ‘the most definitive study’ of Gulf War syndrome in the Lancet. It concluded – surprise, surprise – that there is no such thing as Gulf War syndrome. Clearly, Wessely is a follower of Groucho Marx: ‘Whatever it is, I deny it’*”.

These are profoundly serious issues in which Professor Wessely seems to have been shown to be completely wrong, yet no-where has it been possible to find a retraction of, let alone an apology for, the incalculable damage that many people believe his misinformed opinions and policies have caused.

Although psychiatric disorders are diagnosed on opinion and not on a definitive diagnostic test, Professor Wessely demands “evidence-based medicine” supported by a definitive test and specific biomarkers before he will accept the reality of ME/CFS. Whilst there is as yet no specific diagnostic test, there is an abundance of biomarkers which support the diagnosis, but Professor Wessely continues his determined and sustained denial and dismissal of this scientific evidence that clearly proves him to be wrong.

As Philip Steer, Emeritus Professor, Imperial College, London, asks in the current issue of the British Medical Journal: “*Could strict adherence to evidence-based practice be*

*harmful to patients?” and he notes that: “**Conviction politicians’ may be popular, but conviction doctors are potentially dangerous**” (BMJ 2008:336:673).*

Of even more concern is the fact that, despite having been shown to be so wrong about, for example, the Camelford disaster, Gulf War syndrome, the dangers of mobile phones, the nature of IBS, the nature of fibromyalgia and the nature of ME/CFS (for evidence, see below), Professor Wessely’s influence over Government policy continues unabated.

The influence of his team in the NICE Guideline on “CFS/ME” featured in the 2007 R&D (Research & Development) annual reports by NHS organisations in England, in which the South London and Maudsley NHS Trust stated in section 2A (“Examples of impact on health and social care”): ***“We begin by summarising key achievements and follow with six examples that illustrate the impact of our research”***.

The section on “Chronic Fatigue Syndrome” boasts: ***“In October 2006 NHS Plus published Occupational Aspects of the Management of Chronic Fatigue Syndrome: a National Guideline. It was accompanied by two additional leaflets, one for Health Care professionals and one for employers. This report was heavily influenced by research carried out at our Chronic Fatigue (sic) Unit. The NICE CFS/ME guideline also includes priority recommendations to which our research, led by Trudie Chalder and colleagues, has contributed: ‘When the adult or child’s main goal is to return to normal activities, then the therapies of first choice should be CBT or GET because there is good evidence of benefit for this condition in mild to moderately affected adults and some evidence in mild to moderately affected children’. As a result of our research we have developed our chronic fatigue syndrome service to include treatment at home. In addition we now offer telephone treatment routinely after demonstrating its effectiveness”***.

ME/CFS

On 18th March 2008 The Daily Telegraph carried an item entitled “ME: ‘Invisible disease’ is now easier to read” by Bob Ward, who reported on the work of Dr Jonathan Kerr of St George’s University of London (published in the Journal of Clinical Pathology and to be presented at an ME Research UK [MERUK] biomedical conference at the University of Cambridge on 6th May 2008). The article pointed out that Kerr’s team has identified 88 genes that produce different levels of proteins and other molecules in ME/CFS compared with controls. In 2005 Kerr carried out a complex analysis and found that patients with ME/CFS can be divided into seven clinical sub-types according to specific gene combinations and the severity of symptoms. The most severely affected patients had 71 of the 88 gene abnormalities. In his follow-up paper to which the Telegraph article referred, Kerr’s earlier work was confirmed: (J Clin Pathol 2007: doi:10.1136/jcp.2007.053553): ***“In this study, for each CFS/ME subtype, we determined those genes whose expression differed significantly from that of normal blood donors. Genomic analysis was then related to clinical data for each CFS/ME subtype. Genomic analysis revealed some common (neurological, haematological, cancer) and some***

distinct (metabolic, endocrine, cardiovascular, immunological, inflammatory) disease associations among the subtypes. It is particularly interesting that in these genomically derived subtypes, there were distinct clinical syndromes, as would be expected in a disease with a biological basis”.

Other researchers have noted that patients with ME/CFS can have “*a genetic predisposition to an immunomodulatory response of an inflammatory nature, probably secondary to one or more environmental insults*” (N Carlo-Stella et al. Clin Exp Rheumatol 2006;24(2):179-182).

One would think that such evidence would lead to a change in attitude by Wessely School psychiatrists towards ME/CFS, but as has been noted countless times by many people, nothing seems to stop Wessely’s influence on Government policy: a current example is the forthcoming conference on “CFS” to be held at The Royal Society of Medicine on 28th April 2008, about which Dr Derek Enlander from New York wrote on 21st March 2008 to the Editor of the Daily Telegraph: “*Your article on gene research in ME was a breath of fresh air in the stale atmosphere of UK Government funded research. Over the years it has been shown to be a physical disease. The cause is obscure (and) this obscurity has been masterfully used by psychiatrists to claim that the disease is a manifestation of a psychiatric condition. What arrogance! The Royal Society of Medicine plays to this theme by running a conference on ME/CFS. The speakers are dwelling mainly on psychiatry – rather peculiar for a Society of Medicine. As far as I know the RSM has not noted these physical aspects. The Government through NICE continues to waste money on proven bad methods of treatment which, in a large number of cases, cause relapse. Surely, by now, the Government should be embarrassed*”.

That ME/CFS is not a somatisation disorder is now beyond doubt because there is overwhelming evidence confirming it to be a multi-system organic disorder in which there is disruption of virtually every system in the body (for evidence, see <http://www.mereseearch.org.uk/information/researchdbase/index.html> and <http://www.meactionuk.org.uk> -- between them, these sites contain over **3,000** published papers demonstrating that ME/CFS is **not** a psychiatric disorder). The item published on 18th March 2008 in The Daily Telegraph to which Dr Enlander referred above was indeed a breath of fresh air. As noted by Dr John Greensmith in his response: “*There has been ample research evidence for M.E. as a discrete illness since 1956 and it has been endorsed by the WHO as a neurological illness since 1969, yet the Government’s advisers, who are dominated by psychiatrists, have tampered with the M.E. entry in the British version of the WHO handbook (though it remains untouched in other countries) and have recommended two treatments on the basis of questionable research evidence, one of which, cognitive behavioural therapy (CBT) has no lasting benefit for people with M.E. and the other, graded exercise therapy (GET) may leave some patients irrecoverably worse. They say that they do not believe that M.E. is ‘all in the mind’ (but) since most patients are treated by psychiatrists, using treatments developed for psychiatric illnesses, most often in psychiatric units of hospitals, it is hard to think how otherwise they would treat them if they did believe it was of psychiatric origin. The situation does not look set to change. Indeed, a Royal Society of Medicine conference to*

be held on 28th April 2008, to which selected delegates have been invited and others told that they should not attend, is expected to recommend that this unproven service should be expanded” (drjohngreensmith@mefreeforall.org).

Nancy Klimas, Professor of Medicine at the University of Miami and an international expert on ME/CFS, affirmed: “*Our patients are terribly ill, misunderstood, and suffer at the hands of a poorly informed medical establishment and society*” (AACFS In-coming Presidential Address: Co-Cure 21st March 2005).

In January 2008, Klimas went on record: “*As an immunologist, I once would have said (ME)CFS is clearly an immune dysfunction state, while an endocrinologist would have called attention to the adrenal gland irregularities, and a specialist in the autonomic nervous system would be convinced (ME)CFS is all about blood pressure abnormalities. Given what we’ve discovered about the illness, I now tell people (ME)CFS is all of these things. We know that (ME)CFS has identifiable biologic underpinnings because we now have research documenting a number of pathophysiological processes involving the brain, the immune system, the neuroendocrine system and the autonomic nervous system*” (Historical perspective. Nancy Klimas. In: “Defining Moments – 20 years of making CFS History”, published by the CFIDS Association of America, January 2008).

It is regrettable that such pronouncements do not receive anything like the publicity that Professor Wessely’s pronouncements receive.

The latest evidence demonstrating the key finding that there is a low-grade inflammatory response in ME/CFS was published on 21st March 2008 in Clinical Science (VA Spence et al: Clinical Science 2008:114(8):561-566); this important paper adds to the existing body of scientific knowledge about ME/CFS that shows excessive cytokine production, disruption of the HPA axis and dysfunction of the autonomic nervous system, none of which can credibly be attributed to a behavioural disorder that is amenable to psychotherapy.

Professor Wessely and other members of the “Wessely School” simply ignore all this scientific evidence that proves them wrong and they remain committed to their own unshakable beliefs, which many people believe have resulted in unnecessary suffering of innumerable sick people.

Fibromyalgia

Just as he dismisses ME/CFS as a somatisation disorder, Professor Wessely likewise asserts that fibromyalgia (FM) also is a somatisation disorder – indeed, he asserts that it is the same somatisation disorder (Lancet 1999:354:936-939). He clearly believes this, but where is his evidence? There is none.

The scientific evidence, especially the more recent evidence, continues to mount and it does not support Professor Wessely’s beliefs. He, however, rejects this substantial body of evidence that he is wrong.

The WHO classifies FM as a discrete disorder in ICD-10 at M79 under soft tissue disorders, not as a somatisation disorder.

The Mayo Clinic recently published “Fibromyalgia myths: The truth about 9 common myths”, which stated “*Fibromyalgia is a specific diagnosis*” (<http://www.mayoclinic.com/health/fibromyalgia/AR00056>).

Illustrations of research findings in FM include the following:

In 1997 it was shown that levels of somatomedin C are lower in FM patients (AL Bennett et al. J Psychiat Res 1997:31:1:91-96).

In 1998 researchers showed that levels of Substance P are elevated in FM patients (Evengard B et al. Pain 1998:78:2:153-155).

In 2003 it was shown that endothelin-1 is raised in FM patients (Pache M et al. Rheumatology 2003:42:493-494).

Research in 2005 indicated that FM is the result of internal biochemical imbalances that cause the physical symptoms (Co-Cure MED: 2nd January 2005: Fibromyalgia: new insights into a Misunderstood Ailment).

Different research in 2005 found elevated N(epsilon)-carboxymethyllysine levels in muscular tissue and in serum of patients with FM, with more intensive staining in the interstitial connective tissue of fibromyalgic muscles (Ruster M et al. Scand J Rheumatol 2005:34(6):460-463).

Again in 2005, more serious abnormalities were demonstrated by histologic studies particularly on electron microscopy, revealing disorganisation of Z bands and abnormalities in the number and shape of mitochondria: biochemical studies and P31 magnetic resonance spectroscopy showed inconstant abnormalities of ATP and phosphocreatine levels. The authors noted that “*Mitochondrial abnormalities, reduced capillary circulation and thickened capillary endothelium may result in decreased availability of oxygen and impaired oxidative phosphorylation as well as ATP synthesis*” and commented that these abnormalities do not seem to be the consequences of de-conditioning (Le Goff P. Joint Bone Spine 2005, November 9th).

In 2006, an important review in the Annals of the New York Academy of Sciences (Sarzi-Puttini P et al, Ann N Y Accad Sci 2006:1069:109-117) demonstrated orthostatic intolerance in FM, suggesting underlying abnormalities in cardiovascular neural regulation: “*Research suggests that various components of the central nervous system are involved, including the HPA axis, pain-processing pathways, and the autonomic nervous system*”.

Again in 2006, research showed a greater prevalence of FM in HTLV-1 (human T cell lymphotropic virus) infected individuals, suggesting that FM may be associated with this viral infection (Cruz BA et al: J Rheumatol: 2006:33(11):2300-2303).

In 2007, researchers at Yale University School of Medicine showed muscle hypoperfusion induced by regional vasomotor dysregulation in FM, noting that this vasoconstriction in muscle would lead to low-level ischaemia and its metabolic sequelae (Katz DL et al. Med Hypotheses 2007: March 19th).

More research into FM in 2007 demonstrated bladder symptomatology (Brand K et al. Clin Rheumatol 2007: May 3rd).

Further research in 2007 showed that autoimmune thyroiditis is present in an elevated percentage of FM patients and that patients with thyroid autoimmunity showed a higher percentage of dry eyes, burning or pain with urination, allodynia, blurred vision and sore throat (Bazzichi L et al. Clin Rheumatol 2007: May 9th).

In 2007, Bazzichi et al also showed evidence of abnormal levels of cytokines in FM: *“The higher levels of cytokines found in FM patients suggest the presence of an inflammatory response system (IRS) and highlight a parallel between the clinical symptoms and biochemical data”* (Clin Exp Rheumatol 2007:25(2):225-230).

Another paper in 2007 revealed a conspicuous pattern of altered brain morphology, suggesting that FM is associated with structural changes in the central nervous system of patients (Schmidt-Wilcke T et al. Pain: 2007: June 21st).

In January 2008 researchers provided compelling evidence of a demyelinating polyneuropathy in FM, with electrodiagnostic (EDX) evidence of both polyneuropathy and demyelination. The authors concluded that 33% of FM patients have clinical and EDX findings of chronic inflammatory demyelinating polyneuropathy / CIDP. (Caro XJ et al. Rheumatology (Oxford) 2008:47(2):208-211).

In February 2008 researchers from McGill University, Montreal, Canada, presented evidence that *“neurotransmitter studies show that FM patients have abnormalities in dopaminergic, opioidergic, and serotonergic systems”* and that *“studies of brain anatomy show structural differences between the brains of FM patients and healthy individuals”* (Schweinhardt P et al. Neuroscientist 2008: February 12th).

Also in 2008, in a blinded study, skin biopsy samples showed electron microscopic evidence of unusual patterns of unmyelinated nerve fibres as well as associated Schwann cells, which the researchers considered may contribute to the lower pain threshold seen in FM patients (Kim SH et al. Clin Rheumatol 2008:27(3):407-411).

In a study published in March 2008, US researchers noted that previously, functional magnetic resonance imaging (fMRI) had shown that the insula displays augmented activity in FM, which means that neurons in FM patients are more active in this part of

the brain. This linked to their own findings that pain decreased when levels of the brain molecule glutamate went down, glutamate being a neurotransmitter that conveys information between neurons in the nervous system (Clauw D et al. Arthritis and Rheumatism 2008:58:3).

Such research findings cannot rationally be dismissed, yet Wessely et al still insist that fibromyalgia is a somatisation disorder and they have deliberately included FM patients in the Medical Research Council's behavioural intervention trials on patients with "CFS/ME" in which "Wessely School" psychiatrists are the investigators, a diagnostic inaccuracy that would seem to make a mockery of the MRC's claim that it funds only studies of the highest scientific calibre, especially as in July 2004 a Minister of State (Dr Stephen Ladyman MP) made it known at a House of Commons All Party Parliamentary Group on FM that doctors were to be offered financial incentives to persuade patients with fibromyalgia to enter these MRC trials.

Gulf War Syndrome

From even before 1996, the time when he and fellow psychiatrist Anthony David were awarded \$1million (£666,000) by the US Department of Defence in a Pentagon-funded study to investigate Gulf War illness among UK veterans (BMJ1997:314:95), Wessely continually denied the existence of Gulf War Syndrome.

In their official report on GWS published in the Lancet in January 1999 (Catherine Unwin et al. Lancet 1999:353:169-178), Wessely et al concluded that there is no such thing as Gulf War Syndrome and that the pathway of such illness could be the "perceived" risk of chemical attack, and that it was this "psychological" effect that might be contributing to the ill-health of Gulf War veterans.

In October that same year a study carried out by the well-respected Rand Corporation for the US Defense Department did not support Wessely's conclusions. As a result of this two-year study by Dr Beatrice Golomb, the Pentagon changed its policy and admitted that there could be a link with GWS and the use of pyridostigmine bromide (PB, or anti-nerve gas) tablets which the UK, US and Canadian troops were forced to take during the first (1991) conflict in the Gulf.

In his testimony to the Gulf War Illnesses Public Inquiry held at the Palace of Westminster in 2004 and chaired by The Rt Hon The Lord Lloyd of Berwick, Robert Haley, Professor and Director of the Division of Epidemiology and Preventative Medicine at the University of Texas South Western Medical Centre, Dallas, an acknowledged world authority on the nature and causes of neurological disease in Gulf War veterans, said of Wessely et al:

"Studies using nonspecific definitions of Gulf War neurological syndrome are biased toward finding negative results. Early in the history of Gulf War illness research, around 1993, a decision was made in the government to the effect that 'there is no Gulf

War syndrome', and this led to pressure on researchers who wanted government funding not to use a case definition of the illness in their research. Without at least a provisional case definition, however, it is virtually impossible to design studies that will elucidate the nature of the illness, or illnesses, and connect them with causes.

"The most important example of the unproductive use of a nonspecific case definition concocted was the series of studies from the Kings College London group. In place of a case definition describing the disease that veterans were complaining of, they defined Gulf War illness as having a score of greater than 72.2 on the SF-36 questionnaire, which measures functional impairment regardless of the cause. This case definition essentially counted veterans as having Gulf War illness if they had any condition that caused them to feel bad. Consequently, many veterans with diseases other than Gulf War neurological syndrome that made them feel bad were mistakenly counted as cases, and conversely, many with typical symptoms of Gulf War neurological syndrome but who were not very ill with it were not counted as cases. This severe degree of bidirectional misclassification has caused all studies from the Kings College London group to reach spuriously negative conclusions".

Professor Haley also provided evidence (against Professor Wessely's studies) that: ***"Studies using nonspecific measures of nerve agent exposure are biased toward finding negative results"***.

Wessely told the Inquiry: *"The Gulf war syndrome debate is really just of academic importance"* but Lord Lloyd (a former law lord) said there was *"every reason"* to accept the existence of a *"Gulf War Syndrome"* (The Independent Public Inquiry on Gulf War Illness. Report published on 17th November 2004).

In March 2008, The US National Academy of Sciences published another report by Dr Beatrice Golomb (of the University of California, San Diego, and Chief Scientist to the US Congress-appointed Committee on Gulf War Illnesses); this report found evidence linking the symptoms experienced by the Gulf War Veterans – including muscle and joint pain, rashes and breathing problems – to a particular class of chemicals, specifically to the anti-nerve gas agent given to the troops, to the pesticides used to control sand-flies, and to the nerve gas sarin. Dr Golomb told Reuters that: *"Convergent evidence now strongly links a class of chemicals – acetylcholinesterase inhibitors – to illness in Gulf War veterans"*. She said that a lot of attention had been given to psychological factors, but that *"psychological stressors are inadequate to account for the excess illness seen"* (<http://www.bbc.co.uk/1/hi/health/7288902.stm>). The Proceedings of the National Academy of Sciences is specific: *"Increasing evidence suggests excessive illness in Persian Gulf War veterans can be explained partly by exposure to organophosphate and carbamate acetylcholinesterase inhibitors, including pyridostigmine bromide (PB), pesticides and nerve agents (and) this exposure may be causally linked to excess health problems in Gulf War veterans"* (Proc. Natl. Acad. Sci. USA, 10.1073/pnas.0711986105).

This study was reported in *The Economist* (War of nerves. 13th March 2008), which also reported Professor Wessely's comments about these irrefutable findings: *"This may encourage sick veterans that a cause of their suffering could finally be found, but Simon Wessely, a professor at the Institute of Psychiatry's centre for military health research, is sceptical. He says that the review is 'an opinion piece that continues a line of argument Dr Golomb has put forwards for some time'"*.

In a response to *The Economist*, Malcolm Hooper (Emeritus Professor of Medicinal Chemistry and Chief Scientific Adviser to the UK Gulf War Veterans) wrote: *"The casual and dismissive comments by Professor Simon Wessely about the recent review by Professor Beatrice Golomb that makes clear the link with chemicals used in the first Gulf War are unacceptable. (They are) indicative of the resistance to extensive American research studies that have identified serious damage to the brains of sick soldiers, major heart and cardiovascular disorders, as well as immune, respiratory and neuromuscular disorders, including an excess of motor neurone disease. Despite no official funding, UK research has found excess osteoporosis and severe endocrine damage in UK veterans. The neglect of these veterans is shameful. Golomb's paper challenges us to seek and speak the truth and to act accordingly"*.

It seems strange that Professor Wessely should reject the science reported in the Proceedings of the New York Academy of Sciences (which has an impressive impact factor rating) in favour of his own speculation.

Moreover, it seems that he fails to see that he is doing exactly that of which he accuses Dr Golomb – i.e. his own view is nothing more than *"an opinion piece that continues a line of argument"* that he has *"put forward for some time"*. The big difference that Professor Wessely seems to have missed -- either by accident or by design -- is that Dr Golomb has got actual evidence to support her findings, whereas he has none.

Toxicity of organophosphate and organochlorine compounds

Professor Wessely has a long published record of rejecting the validity of environmental illness (for example: *BMJ* 1993;307:747-748; *Clin & Exp Allergy* 1995;25:503-514), particularly illness arising from exposure to chemicals, and he has apparently commented with seeming satisfaction that in the modern world it is impossible to avoid daily contact with a multiplicity of chemicals.

In numerous publications, he has seemed to disparage and denigrate patients with symptoms of environmental illness, repeating the same message time and again, both in medical journals and in the media:

"These total allergy syndromes are akin to culture-bound syndromes afflicting modern developed societies where sufferers from unexplained symptoms no longer see themselves as possessed by devils or spirits but instead by gases, toxins and viruses" (*Clin Exp Allergy* 1995;25:503-514).

*“In a previous era, spirits and demons oppressed us. Although they have been replaced by our contemporary concern about invisible viruses, chemicals and toxins, the mechanisms of contagious fear remain the same. **To the majority of observers, including most professionals, these symptoms are indeed all in the mind**” (NEJM 2000:342:2:129-130).*

“The release of poison gas into a crowded Tokyo subway killed 12 people. Since then there have been several reports of sudden episodes of panic among crowds of Japanese commuters. These were probably examples of mass hysteria. Mass hysteria is far from new. A classic book on the subject has just been reissued. It is an account of the follies of mass behaviour throughout the ages. In previous times, mass hysteria would be blamed on demons, spirits and diabolical possession. Nowadays we are oppressed by equally invisible gases, viruses and toxins” (“Have you heard? We are being poisoned”. The Times, 4th July 1995, page 14).

“Like many hospital specialists, I have seen a steady stream of patients with many mysterious symptoms. The sufferers usually blame their ill health on factors such as solvents, pesticides, pollution, food additives or dental amalgam. Many report exquisite sensitivity to such everyday substances as perfumes, deodorants, tap water and hairspray. Such people are sometimes labelled as suffering from ‘total allergy syndrome’. All explanations have much in common. First, there is no personal blame. Second, all appear to be modern worries. Third, all are linked by another modern theme – the immune system in trouble. I doubt it is a coincidence that multiple chemical sensitivity, and total allergy, rose to prominence in parallel with the rise of HIV. The idea that the immune system might give way because of an invisible external agent is now embedded in popular consciousness. But just how new are these modern illnesses? The things that we blame for making us feel ill change over the years. Medieval man was oppressed by spirits and demons. Nowadays we blame similar ills on mysterious viruses and allergies (which are) an ever-changing parody of scientific advances of the day. ‘Modern’ illness is far from modern” (“Sickness of the century. Simon Wessely sees a connection to fears of the past”. The Guardian 28th May 1996, page 13).

“ ‘People always believe they are oppressed. They seize on explanations that are credible and make sense within their world view: 300 years ago, people believed in possession by demons’. These days, he writes in an editorial (in the New England Journal of Medicine), those demons have been replaced by our ‘concern about invisible viruses, chemicals and toxins’. So how do you deal with a mass psychogenic / sociogenic illness? ‘The challenge is to convey the scientific reality without being seen as blaming the victims’, writes Wessely (The Guardian 25th January 2000, pp8-9).

“The threat of chemical and biological weapons could have serious long-term social and psychological consequences, leading to outbreaks of panic-induced illness, according to a leading psychiatrist, Simon Wessely. Outbreaks of mass sociogenic illness are already appearing, (with) worries about reproductive outcomes, such as impaired fertility or damaged babies” (“Panic could be biggest illness”. The Guardian, 19th October 2001).

It is indeed impossible to avoid daily contact with chemicals, over 30,000 of which have not been fully evaluated toxicologically, so their combined effects on humans are unknown. Lindane, an organochlorine pesticide (OCP), was widely used as an insecticide in the farming industry because of the need for ever-increasing food production. The nation (and indeed the world) has been deluged with ever more complex agrochemicals, some of which have now been banned. DDT was found in the food chain and was banned in the 1970s, but OCPs can still be found in environmental and biological matrices due to their persistence (“Man-Made Chemicals in Food Products”. TNO Report, 2006: Netherlands Organisation for Applied Scientific Research). These products are not effectively metabolised so they just accumulate in the body.

There have been innumerable items in the press about falling sperm counts and rising cancer levels, as well as the fact that the UK now has the highest incidence of asthma in Europe.

In June 2003 the Royal Commission on Environmental Pollution, chaired by Sir Tom Blundell FRS, FMed Sci, presented its 24th Report “Chemicals in Products” to Parliament by Command of Her Majesty. It caused a media frenzy. Some illustrations include the following:

“Thousands of chemicals are being used every day without proper safety tests, exposing the public to a ‘gigantic experiment’ experts warned yesterday. The potential dangers posed by flame retardants, plastics, glues and even some toothpastes are uncertain, because only 40 of 30,000 chemicals in large-scale use have been tested fully, says the Royal Commission on Environmental Pollution. Because of this, ‘the chemical disasters of the past are likely to be repeated in the future’” (“Chemical timebomb”. Daily Express, 27th June 2003).

“The government is experimenting with people’s lives by failing to test properly tens of thousands of man-made chemicals used in everyday life, according to a leading biochemist who chairs the Royal Commission on environmental pollution” (“Failure to test chemicals ‘puts lives at risk’ ”. The Guardian, 27th June 2003).

On 22nd April 2004 the Daily Mail carried an item by Robin Yapp, Science Reporter (“Revealed, the toxic chemicals invading our bodies”) in which he wrote: *“A huge cocktail of toxic chemicals can be found in every adult’s blood, research revealed yesterday. Scientists say the chemicals – found in everything from TVs to sofas, cosmetics, to computer screens – are now so widespread in the environment that no-one is likely to escape contamination”*.

Concern was expressed that most testing of chemicals is done on individual compounds, but possible synergistic effects of the compounds in multiple formulated products are generally not tested at all.

In 2005, the Pesticide Action Network UK published “The alternative pesticide residues report: What the Government doesn’t tell us”. This report provides striking examples of where the current regulatory system does not appear to protect consumers, particularly in relation to pesticide residues in food, and notes the uncertainties about the impact of pesticides on human health, particularly chronic illnesses, endocrine disruptors and the effect of a ‘cocktail’ of pesticides.

It cannot have been overlooked by Wessely et al that the work of Dr Jonathan Kerr of St Georges, London, has linked ME/CFS to OPs (J Clin Path 2005;58:826-832). **Kerr et al suggest that patients with (ME)CFS “have reproducible alterations in gene regulation”, noting that “sixteen genes were confirmed as having an expression profile associated with (ME)CFS. These genes can be grouped according to immune, neuronal, mitochondrial and other functions. These findings are consistent with previous work showing that patients with (ME)CFS have evidence of immune activation, such as increased number of activated T cells and cytotoxic T cells, and raised circulating cytokine concentrations. NTE (neuropathy target esterase) is a target for organophosphates and chemical warfare agents, both of which may precipitate (ME)CFS. EIF2B4 is a mitochondrial translation initiation factor and one of the EIFB2 family, within which mutations have been shown to be associated with central nervous system hypomyelination and encephalopathy. The involvement of genes from several disparate pathways suggests a complex pathogenesis involving T cell activation and abnormalities of neuronal and mitochondrial function, and suggests possible molecular bases for the recognised contributions of organophosphate exposure and virus infection”.**

In his subsequent paper referred to above (J Clin Pathol 2007), Kerr stated: “*We have previously documented upregulation of NTE in (ME)CFS. NTE is the primary site of action of organophosphate (OP) compounds. Exposure to OP compounds may trigger CFS/ME and Gulf War Illness*”.

Neuropathy target esterase (NTE) is inhibited by several OP pesticides, chemical warfare agents, lubricants, and plasticisers, leading to OP-induced delayed neuropathy in humans, with over 30,000 cases of human paralysis (Gary Quistad et al. PNAS June 24, 2003;100:13:7983-7987).

Although ostensibly not personally involved in the report of the joint working party of the Royal Colleges of Physicians and Psychiatrists (“Organophosphate sheep dip: clinical aspects of long-term low-dose exposure”; November 1998), Professor Wessely’s influence shines through and a large number of the 85 references are his or those of his close colleagues who share his views. His frequent co-author -- psychiatrist Anthony David -- was a member of the working party.

Commenting on the composition of the Royal Colleges’ working party, Dr Richard Horton, editor of The Lancet, said: “*All together there are ten members, including six professors. A committee with such a distinguished provenance would seem immune from criticism. Far from it. Not one of its members has direct experience of looking after*

patients exposed to OPs. The committee's conclusions are bound to be based on wholly incomplete evidence. Pompous and complacent scientists are seen to be pompous and complacent" (Observer Life 3rd August 1997:41).

It is a matter of note that the 1996 findings of neurologist Professor Peter Behan from the University of Glasgow linking ME/CFS to chronic low-dose OP exposure were excluded from the Report of the Royal Colleges, given that Behan found ME/CFS to be clinically identical to chronic low-dose OP exposure and that such OP exposure "*in some way prepared the patients for the later development of (ME)CFS*". Behan reported that the abnormalities found in both ME/CFS and in OP poisoning were "*compatible with a decreased responsiveness of CNS type II glucocorticoid receptors, (confirming) the hypothesis of brain steroid receptor resistance in patients with the delayed response to OPs and in (ME)CFS*" (J Nutrition & Environmental Medicine 1996:6:341-350).

The Royal Colleges' Report on OPs predictably recommended that treatment for those who have been exposed to OPs should be cognitive behavioural therapy and anti-depressants and it claimed that a "*vicious circle*" of self-maintaining symptoms, including "*illness beliefs and fears about the meaning of symptoms*" perpetuate ill-health. Again predictably, the Report urged against what many would regard as appropriate investigation, claiming that investigations "*may bias the consultation towards a narrow physical orientation*".

The Report barely mentioned the problems of anaesthesia for those with OP exposure, an omission which might well have given rise to a charge of scientific misconduct, given that in 1987 the Stationary Office had published a Guidance Note MS17 which unambiguously warned about the dangers of anaesthesia, especially the commonly-used muscle relaxant succinyl choline, in people who have been exposed to OPs. Further, in 1995 The Royal College of Anaesthetists had warned members about the dangers of OP compounds and anaesthesia.

Neither document was mentioned in the Report of the Royal Colleges on OPs.

Despite the large number of papers from both US and UK researchers that show clear links between neurotoxicity and organophosphate pesticides – effects exacerbated by synergistic action with other pesticides – Professor Wessely continues to insist, without any convincing evidence, that there is no link.

This is not science, but opinion wedded to fanciful postulates of somatic illness which are rejected by other psychiatrists.

There is well-established evidence of the neurological toxicity that is well-recognised in the literature, including work from the US National Institutes of Health, from the MRC Toxicology Unit at the University of Leicester, UK and from prestigious institutions such as The Scripps Research Institute, La Jolla, California.

The target enzyme systems involved in the toxicity of these compounds include not only acetylcholinesterase and butyrylcholinesterase but also much more sensitive brain enzymes and neuropathy target esterase which play a role in nerve function and in the development of motor neurone disease (MND).

It is well-known that OPs affect brain esterase enzymes at much lower dosages than those producing significant inhibition of acetylcholinesterase commonly regarded as the target enzymes for OPs.

OP compounds have traditionally been associated with the inhibition of esterase activity (Paul G Richards et al. *Molecular Pharmacology* 2000;58:3:577-583).

It is widely known that cholinesterase inhibitors such as OPs are commonly used as insecticides and pesticides and the chemically closely-related (and more toxic) organophosphonates are used (and may be stored) in biological warfare agents.

Researchers have demonstrated that the time and exposure levels of these agents have considerable relevance in determining possible brain injury (Lola Roldan-Tapia et al. *Neurotoxicology and Teratology* 2005;27:259-266).

Low dose exposure to both pesticides and nerve agents gives rise to delayed chronic neurotoxicity (Abou-Donia et al. *Archives of Environmental Health* 2003;58:484-497).

Abou-Donia and Garrettson have identified auto-antibodies to neuronal proteins as a marker for OP neurotoxicity (*Environmental Epidemiology and Toxicology* 2000;2:27-41).

It is a matter of public record that the incidence and prevalence of Alzheimer's disease are increasing rapidly (Pritchard et al. *Public Health* 2004;118:268-283).

As well as providing a target for the action of pesticides, the cholinergic system plays an important role in the progression of Alzheimer's disease and there is strong correlation between the severity of the dementia and the cholinergic deficits (Paul G Richards et al. *Molecular Pharmacology* 2000;58:3:577-583).

In a paper looking at the neurotoxicity of chronic exposure to moderate levels of pesticides, Kamel et al analysed cross-sectional data from 18,782 individuals over a four year period in relation to 23 neurological symptoms. Among chemical classes of insecticides, associations were strongest for organophosphates and organochlorines. Results suggest that neurological symptoms are associated with cumulative exposure to moderate levels of organophosphate and organochlorine insecticides (Freya Kamel et al. *Environmental Health Perspectives* 2005;113:7:877-882).

Changes in erythrocyte enzymes in humans have been reported after exposure to different pesticides, including OPs, one of which appears to be an important biological indicator of pesticide exposure (Antonio F Hernandez et al. Toxicology Letters 2005:159:13-21).

From just these few illustrations, it is clearly untenable for anyone to claim that symptoms of low-dose OP poisoning are a somatisation disorder.

The Camelford catastrophe

Wessely is equally dismissive of the Camelford drinking water contamination, where in July 1988 twenty tonnes of aluminium sulphate were pumped into the drinking water supplies of the Cornish town, resulting in the death of seven people, with 25,000 people suffering serious health effects and with 40,000 animals affected (The Ecologist 1999:20:6:228-233). The death toll has since risen – see The Daily Telegraph, 20th April 2006: “Alzheimer’s fear grips poisoned water town” by Medical Editor Celia Hall. Bone biopsies carried out over six months later showed stainable aluminium. Although noting that some peoples’ hair, skin and nails turned blue, in their paper in the Journal of Psychosomatic Research (The Legend of Camelford: 1995:39:1:1-9) Wessely and his co-author Anthony David were not to be moved: they claimed that it was all mass hysteria (BMJ 1995:311:395) and that the “somatic” symptoms were the result of heightened perception of normal and benign symptoms and irresponsible reporting by the press, though they have not explained by what mechanism hysteria affects animals.

In 1999 it was conclusively shown by Paul Altmann et al that there was objective evidence of considerable organic brain damage compatible with the known effects of exposure to aluminium and that it was this exposure, not anxiety or hysteria, which was the cause of the symptoms exhibited by those who had been exposed to the contaminated water (BMJ 1999:319:807-811).

More recently, Exley and Esiri described severe cerebral congophilic angiopathy coincident with increased brain aluminium in a resident of Camelford (JNNP 2006: doi:10.1136/jnnp.2005.086553), causing Walter Lukiw, Associate Professor of Neuroscience at Louisiana State University Health Sciences Centre, to note that as over-expression of stress-sensing, pro-inflammatory and pro-apoptotic genes have been observed in aluminium sulphate-induced neurotoxicity, “*careful attention should be paid to the neurological status and neuropathological outcome of the thousands of unfortunate victims at Camelford*” (eBMJ, 21st April 2006).

In December 2007, the West Somerset Coroner Michael Rose ordered the police to re-open the Camelford pollution case following allegations of a cover-up (Guardian, 13th December 2007).

Responding to this announcement, Sue Waddle, spokesperson for the charity ME Research UK, a magistrate and the mother of a daughter severely affected by ME wrote

to The Guardian on 16th December 2007: *“I and many others await with interest the outcome of any police inquiry. A 1995 paper by two psychiatrists asserted that mass hysteria and / or anxiety were responsible for the supposed suffering of those in the Camelford area at the time. (One of these ‘experts’) has also given his expert opinion on many other ‘non-illnesses’ and ‘unfounded health worries’. He happens to be the Government expert on electricity pylons, mobile phone masts, Gulf War Syndrome and myalgic encephalomyelitis”*.

The Coroner’s conclusions are still awaited, but clearly the existing evidence does not support Professor Wessely’s beliefs that the Camelford disaster was merely contagious mass hysteria.

Irritable bowel syndrome

Another of Professor Wessely’s targets for somatisation disorder is irritable bowel syndrome or IBS (The Lancet 1999:354:936-939) but the evidence does not support such a model.

The following are illustrative of a wide body of evidence:

At the 68th Annual Scientific Meeting of the American College of Gastroenterology held in 2003 at Baltimore, important findings were presented by lead investigators from the University of Vermont (Peter Moses, Associated Professor of Medicine and Director of Clinical Research in the Digestive Diseases, and Gary Mawe, Professor of Anatomy and Neurobiology): *“Serotonin is a critical signalling molecule necessary for normal gut function. Our finding that key elements of serotonin signalling are changed in IBS lends credibility to the notion that IBS is not simply a psychological or social disorder as was once thought, but instead due to altered gut biochemistry and interactions between the gut and the brain. Now we have a perspective on molecular changes in the intestines of individuals with IBS that we did not have before. We identified a significant decrease in the serotonin transporter in cells that form the inner lining of the bowel. Because the transporter is diminished in IBS, serotonin stays around longer, and this can lead to changes in motility, secretion, and sensitivity”* (Ecotoxicology 2003:12 (1-4):345-363).

In 2006, the BMJ Learning programme by a Clinical Research Fellow and a Professor of Medicine and Gastroenterology featured IBS (BMJ 2006:332:280-283). This programme pointed out that a number of pathophysiological abnormalities can often be identified: ***“IBS is now clearly understood to be a multifactorial condition, rather than its just being due to psychopathology. These include motility, visceral sensation, central processing, genetics, inflammation and neurotransmitters”***.

At the American Academy of Neurology 59th Annual General Meeting held in Boston in April / May 2007, researchers from Brazil showed that people with inflammatory bowel disease were at risk for subsequent neurological disorders and presented convincing evidence of the link between IBD and peripheral neuropathy: *“Based on these results, we*

believe IBD itself is directly related to the neuropathy and that neuropathy in these patients is much more common than previously thought”.

In ME/CFS specifically, there is evidence that the disorder is accompanied by an increased translocation of endotoxins of gram-negative enterobacteria through the gut wall, with signs of activation of the inflammatory response system and IgG3 subclass deficiency (Maes M et al. Neuro Endocrinol Lett 2007:28:6).

Clearly, the out-dated hypothesis that IBS is a psychosomatic disorder has been abandoned by those who fulfil their contractual obligations to keep up-to-date with medical science, yet Professor Wessely et al seem unaware of this progress in medicine.

Mobile phone sensitivity

In 2003 Professor Wessely’s team was awarded a research grant of £405,000 to investigate the psychological and biological effects of mobile phone radiation in healthy subjects and subjects with self-reported mobile phone hypersensitivity. Professor Wessely was Principal Investigator. The study was expected to last until April 2006.

When this was announced, one astute ME sufferer observed: *“That’s one more negative result, then!”*.

On 2nd September 2003 the Countess of Mar wrote to Professor George Szumukler, Dean of Psychiatry at the Institute of Psychiatry about Professor Wessely’s involvement in this study:

“As Principal Investigator of the (new) Mobile Phone Research Unit at Kings College Hospital, doubtless (Professor Wessely) is soon to ‘discover’ mobile phones have no biological consequences for human health other than the aberrant beliefs of those using them”.

Perhaps importantly, the study was jointly funded by the Programme Management Committee of the MTHR (Mobile Telecommunications and Health Research programme), which itself is jointly funded by the UK Department of Health and the mobile telecommunications industry.

The study was published on 15th April 2006 in the BMJ (2006:332:886-891).

As widely anticipated, Professor Wessely's study concluded: *"We found no evidence that self-reported sensitivity to mobile phone signals has a biological basis"*. However, the study also noted: *"That symptom severity did increase during exposure is interesting. These symptoms were not trivial. Indeed, for some they were so severe that exposures had to be stopped early or the participants withdrew from the study"*.

Undeterred, the authors still advised: *"In terms of their clinical implications, these results do not suggest that attempting to reduce exposure to mobile phone signals will be a useful strategy for patients who report sensitivity to them. Although such interventions might be actively sought by patients, in the longer term a danger exists that they will reinforce a patient's view of himself or herself as being sensitive to electromagnetic fields. Instead it may be better to encourage such patients to test alternative explanations for their symptoms by using cognitive behavioural therapy. The symptoms reported by 'sensitive' people may be primarily psychological in origin"*.

It is notable that a study from Finland that was published the same year as Professor Wessely's study came to interesting conclusions, namely, that mobile phones affect brain blood flow: *"Mobile phones create a radio-frequency electromagnetic field around them when in use. We studied the effects of a commercial mobile phone on regional cerebral blood flow (rCBF) in healthy humans using positron emission tomography (PET) imaging (in) a double blind, counterbalanced study. Explorative and voxel-based statistical analysis revealed that a mobile phone in operation induces a local decrease in rCBF beneath the antenna in the inferior temporal cortex and an increase in the prefrontal cortex, suggesting that the electromagnetic field (EMF) emitted by a commercial mobile phone affects rCBF in humans. These results are consistent with the postulation that EMF induces changes in neuronal activity"* (Sargo Aalto et al. *Journal of Cerebral Blood Flow & Metabolism* 2006;26:885-890).

Whilst the Finnish study did not seek to identify hypersensitivity to mobile phones, it did provide actual evidence that they affect brain blood flow.

Arthur Firstenberg is unequivocal: *"The most basic fact about cell phones and cell towers is that they emit microwave radiation; so do wireless computers, cordless phones and their base units. A cell phone that is on but not in use is also radiating. It is a fact that we are all being bombarded, day in and day out, whether we use a cell phone or not, by an amount of radiation that is some ten million times as strong as the average natural background. A cell phone, like a microwave oven, heats you from the inside out, not from the outside in. The presence of albumin in the brain is always a sign that blood vessels have been damaged and that the brain has lost some of its protection. Researchers have found, consistently for 18 years, (that) microwave radiation, at doses equal to a cell phone's emissions, causes albumin to be found in brain tissue. In research published in 2003, a single two-hour exposure to a cell phone just once permanently damaged the blood brain barrier. Two minutes on a cell phone disrupts the blood brain barrier; two hours on a cell phone causes permanent brain damage"* (Leif G Salford et al. *Environmental Health Perspectives*: 2003;111:7:881-883).

Firstenberg continues: *“Diseases that have increased remarkably in the last couple of decades (which) there is good reason to connect with the massive increase in radiation in our environment, include asthma, sleep disorders, multiple sclerosis, ALS, Alzheimer’s disease, **fibromyalgia, chronic fatigue syndrome**, hypothyroidism, diabetes, malignant melanoma, testicular cancer, and heart attacks and strokes in young people. The literature showing biological effects of microwave radiation is truly enormous, running to tens of thousands of documents. I am amazed that industry spokespersons are getting away with saying that wireless technology has been proved safe or – just as ridiculous – that there is no evidence of harm. A 1998 survey by the California Department of Health Services indicated that at that time 120,000 Californians – and by implication one million Americans – were unable to work due to electromagnetic pollution”* (California EMF Program: The Risk Evaluation. 2002).

Firstenberg is clear: *“**The ranks of these so-called electrically sensitive are swelling in almost every country in the world, marginalized, stigmatised and ignored**”*.

The full paper can be found at http://www.eldoradosun.com/Archives/01-06_issue/Firstenberg.htm .

Wessely et al apparently do not accept such findings, preferring instead to endorse findings of a three-year study at the University of Essex for the UK Health Protection Agency (HPA), which found “Phone mast allergy ‘in the mind’ ”. Perhaps it is relevant that, as in the case of Professor Wessely’s study, this study was funded by the Mobile Telecommunications and Health Research programme, a body which itself is funded by industry and Government.

Conclusion

In defiance of the extensive published evidence that ME/CFS and other disorders mentioned above are not psychosomatic, Professor Wessely’s unremitting insistence that they are in reality but one single behavioural disorder seems indefensible.

In April 2000 an Opinion from a leading Queen’s Counsel (who is a member of the House of Lords) was obtained about Professor Wessely’s dogma on ME/CFS. That Opinion is concise:

“On the document you have sent me there is an overwhelming case for the setting up of an immediate independent investigation as to whether the nature, cause and treatment of ME as considered by the Wessely School is acceptable or consistent with good and safe medical practice. There is substantial doubt as to whether such could be the case. It is, of course, open to patients (and) their parents to seek Judicial Review”.

In her letter of 2nd September 2003 to Professor Szmukler referred to above, the Countess of Mar wrote:

“Through his prolific output Professor Wessely has introduced his personal beliefs into the UK medical literature and those beliefs are aimed at changing the perception of

ME/CFS held by both medical and lay people. Through the shortcomings of the peer-review system, his personal beliefs have become medical doctrine, effectively turning patients into victims”.

Without doubt, there is substantial evidence in the public domain that Professor Wessely himself has carried out an unremitting campaign of denigration of ME sufferers. One of the most notorious was his involvement with a poll run by the British Medical Journal in 2002 in which doctors were asked to vote on what they considered to be “non-diseases”. It is understood that it was Professor Wessely himself who nominated ME. Along with big ears and freckles, ME was duly voted a “non-disease” that should be left medically untreated.

It must be due in large part to such disgraceful antics and to the fact that Professor Wessely and other members of the Wessely School are Government advisers on “CFS” that people with ME/CFS are suffering politically-driven health discrimination which is contrary to the Disabled Discrimination Act.

There is a broad body of informed opinion – national and international -- that Professor Wessely belittles other peoples’ work without addressing the issues.

For a detailed exposition of the tactics of dismissal used by the Wessely School, see “The Mental Health Movement: Persecution of Patients? A Consideration of the Role of Professor Simon Wessely and Other Members of the ‘Wessely School’ in the Perception of Myalgic Encephalomyelitis (ME) in the UK. Briefing Paper for the House of Commons Health Select Committee” by Malcolm Hooper et al http://www.meactionuk.org.uk/SELECT_CTTEE_FINAL_VERSION.htm

Apart from the Wessely School’s own studies, there is little published evidence to support the notion that CBT actually *works* in ME/CFS, and their own studies have been the subject of criticism on the grounds that many of their studies are deemed to be methodologically flawed, principally because of the authors’ selection bias (i.e. they are not studying cases of true ME/CFS, but are then claiming that their results relate to ME/CFS).

For many years Professor Wessely has achieved considerable coverage of his views in the UK media on topics ranging from dental amalgam, “blaming mummy for a bad tummy” “the power of the placebo”, “how long should a sick leave last?”, bogus miracle cures, and total allergy syndrome to RSI (repetitive strain injury), so the national press coverage of the apparently exponential increase in rates of psychosomatic disorder and the alleged efficacy of CBT is substantial, with Professor Wessely being frequently quoted in the broadsheet newspapers.

Also, due in no small measure to Professor Wessely’s apparent control over what gets publicly funded on ME/CFS (perhaps due his previous positions on three MRC Boards and to the fact that “Wessely School” members hold influential positions at the MRC) and what gets published on ME/CFS in the UK (perhaps exercised through his position as a member of the Scientific Advisory Panel to the Science Media Centre which was founded in 1999; it is funded by pharmaceutical companies and operates like a newsroom

to promote the views of industry and to launch fierce attacks against those who question them), the medical journals frequently publish highly uncritical assessments of CBT which focus on the few studies which support its use, whilst ignoring those controlled trials which did not find CBT to be effective (and which warned about the dangers of exercising beyond fatigue).

This matter is the subject of an article entitled “A Subgroup Analysis of Cognitive-Behavioral Treatment Studies” by Fred Friedberg (JCFS: 1999:5: 3-4:149-159; co-published simultaneously as “Chronic Fatigue Syndrome: Advances in Epidemiologic, Clinical and Basic Science Research (ed) Roberto Patarca-Montero, Haworth Press Inc. 1999).

Friedberg, clinical professor in the Department of Psychiatry at the State University of New York, made the following cardinal points:

“Several studies of graded activity-oriented cognitive behavioural treatment for (ME)CFS, all conducted in England, have reported dramatic improvements in functioning and substantial 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 (ME)CFS symptoms.

“Furthermore, descriptive studies of CF (chronic fatigue) patients in England, the US and Australia suggest that the (ME)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 primary depression patients and more closely resemble fatiguing neurological illnesses”.

Professor Friedberg notes the “widely divergent clinical presentations” and he notes specifically that because all the apparently successful CBT studies have all been conducted in England, a replication of these findings in a well-designed US study would be necessary before a general recommendation for CBT could be made.

Professor Friedberg’s paper was published almost a decade ago, yet Professor Wessely’s influence in the UK remains undiminished.

In a paper dated 8th March 2008 entitled “The Year of No Compromise” Greg Crowhurst, a health care professional whose wife is one of the most severely affected ME/CFS sufferers in the UK, said the following:

“This is a simple summary of the inferred messages underpinning the psychiatric paradigm, currently being heavily promoted in the UK”.

Although written specifically in relation to ME/CFS, the summary applies equally to all disorders designated by Wessely et al as being “medically unexplained” which these

psychiatrists assert are Functional Somatic Syndromes (FSS), including the disorders outlined above. These “Wessely School” psychiatrists in fact believe that ME/CFS, FM, IBS, non-ulcer dyspepsia, pre-menstrual syndrome, chronic pelvic pain, atypical chest pain, “hyperventilation syndrome”, tension headache, temporomandibular joint pain, globus syndrome and multiple chemical sensitivity are but one single psychiatric disorder (Lancet 1999:354:936-939).

Crowhurst’s summary exactly captures the situation in the UK:

“The recommendations:

- *do not investigate ME/CFS patients*
- *do not provide special facilities for ME/CFS patients other than psychiatric clinics*
- *do not offer special training to doctors about the disorder*
- *do not offer appropriate medical care for ME/CFS patients*
- *do not offer respite care for ME/CFS patients*
- *do not offer State benefits for those with ME/CFS*
- *do not conduct biomedical research into the disorder*

The tactics:

- *the wreaking of havoc in the lives of ME/CFS patients and their families by the arrogant pursuit of a psychiatric construct of the disorder*
- *the attempts to subvert the international classification of this disorder from neurological to behavioural*
- *the propagation of untruths and falsehoods about the disorder*
- *the building of affiliations with corporate industry*
- *the insidious infiltration of all the major institutions*
- *the denigration of those with ME*

The practices:

- *the attempt to make "ME" disappear in a sea of chronic fatigue*
- *the refusal to see or acknowledge the multiplicity of symptoms*
- *the ignoring and misinterpretation of the biomedical evidence*
- *the suppression of published findings*
- *the vested interests*

The impact:

- *the arresting and sectioning of protestors*
- *the silencing of ME patients, through being given a psychiatric label*

- *the suppression of dissent*
- *the labelling of ME patients as the "undeserving sick", as malingerers*
- *the forcible removal of sick children and adults from their homes.*

“It is poignant how an institutionally supported prejudice against people with ME has arisen, based on nothing more substantial than supposition and opinion, carefully disseminated.

“You have to be very careful how you discern the truth; it is an important issue in the corporate wall of collusion surrounding the physically sick people who have ME.

“We have to be very clear about what is the truth about ME and what is either deliberate, naive or ignorant misinterpretation or misrepresentation. The impact of the above strategy on peoples’ lives is catastrophic”.

Crowhurst’s article can be accessed at www.metrotrainingco.org.uk

As noted by Hooper et al, the malign influence of Wessely School dogma extends throughout Government departments, throughout the NHS, and even extends to the Judiciary, with one Claimant being told at a High Court Hearing that *“Judges regard ME as psychological self-indulgence”*. One Local Health Board will only fund treatment for ME/CFS where the focus is CBT/GET. A spokesman for Grampian NHS Trust is on record in 2003 (disturbingly, this was a year after the publication of the UK Chief Medical Officer’s Working Group Report) as stating *“ME is not a condition we recognise or treat”* (see *“Illustrations of Clinical Observations and International Research Findings from 1955 to 2005 that demonstrate the organic aetiology of ME/CFS”* http://www.meactionuk.org.uk/Organic_evidence_for_Gibson.htm).

The damage perpetrated on those with ME/CFS by Wessely School adherents cannot be quantified. The Wessely School argument that syndromes like ME/CFS cause *“unnecessary expenditure of medical resources”* has been criticised by a leading US researcher for its pernicious public policy implications (Lancet, 11th December 1999:354: number 9195).

In the UK, patients with ME/CFS, particularly children, have suffered gross and barbaric abuse and persistent denigration as a consequence of the beliefs of Wessely School psychiatrists who are attempting to control the national agenda for this complex and severe neuro-immunological disorder and who by their words and deeds have wreaked havoc in the lives of many ME/CFS patients and their families by their arrogant pursuit of a psychiatric construct of the disorder in clear defiance of the clinical and scientific evidence of the organic nature of ME/CFS.

There have been persistent and frequently covert attempts by these psychiatrists to subvert the international classification of ME/CFS, with destructive consequences for those affected.

It seems that Professor Wessely is accountable to no-one for his role in determining UK Government policy that the disorders mentioned above do not exist as discrete entities and that such patients should be “managed” by psychotherapy.

Instead, in return for his decades of denigration of patients (for actual quotations from his work see “Quotable Quotes about ME/CFS” available from the charity Invest in ME at 01603 – 701980) and for his denial and dismissal of the published evidence that he is wrong, and for all the seemingly consequential suffering and despair arising from his personal beliefs, Professor Wessely has been lauded and honoured.

On 27 August 2003, Dr George Szukler, Dean of Psychiatry, Institute of Psychiatry, King’s College Hospital, London, wrote to the Countess of Mar about Professor Simon Wessely: **“Professor Wessely must be judged one of the most outstanding researchers in the UK, and indeed internationally. Professor Wessely has been awarded a Research Medal by the Royal College of Physicians specifically for his work on CFS and he has served on many prestigious scientific committees, further attesting to the high regard in which he is held by the scientific community”**.

Not everyone – including doctors and medical scientists from around the world -- shares that view.