For the attention of the Gibson Inquiry

Eileen Marshall Margaret Williams 13th July 2006

It is acknowledged that members of the Gibson Inquiry cannot continue to be inundated with emerging information about ME/CFS, but as evidence is still to be heard up to 18th July 2006, three recent items would seem to merit consideration.

Firstly, an important paper from well-respected Japanese investigators concludes that Vis-NIR analysis for sera, combined with chemometrics analysis of serum, achieves complete separation of (ME)CFS patients from healthy controls. The authors state that this technique deserves further evaluation as a diagnostic marker for the disorder and they note: "More importantly, these results suggest that unknown factor(s) in the serum are commonly present in all (ME)CFS patients".

What "unknown factors" might these be? How significant are they? It is obvious that in order to find out, more research is needed.

The authors of this study on (ME)CFS would unambiguously seem to disagree with (1) the view of Professor Anthony Pinching, who is famous for his on-the-record comment in 2000 at the CMO's Working Group Adult Sounding Board event that there is no need for research into ME/CFS before treating people who suffer from it, saying: "Our worries about names, causation, mechanisms, OK, are fun"; (2) the well-publicised beliefs of members of the Wessely School and (3) the officially recorded view of Professor Colin Blakemore (Chief Executive of the Medical Research Council) which he presented to the All Party Parliamentary Group on ME on 26th April 2006 at the House of Commons, all of whom advise that "illnesses" can be treated without knowing the causes and that there is no need to look for the cause of ME/CFS:

"Further information obtained from detailed analysis may make significant contributions not only to diagnosis, such as finding reliable biochemical markers, **but also to the understanding of (ME)CFS pathophysiology, which will lead to an effective treatment for the disease**".

In plain terms, the authors are saying that it is an understanding of the pathophysiology of the disorder that will lead to an effective treatment (ref: Spectroscopic diagnosis of chronic fatigue syndrome by visible and near-infrared spectroscopy in serum samples. A. Sakudo, H. Kuratsune et al. Biochemical and Biophysical Research Communications 2006:345:1513-1516).

Why is this basic scientific concept continually rejected by decision-makers in the UK in relation to ME/CFS?

Given the apparent intention of the State not to spend money on the chronically sick (who are economically unproductive), and the determination to remove people from incapacity

benefit and return them to gainful employment, the answer may be contained in the second item of interest.

The CFIDS Association website (http://www.cfids.org/cfidslink/2006/prevalence.asp) carries the latest estimate of the prevalence of (ME)CFS provided by the US Centres for Disease Control (CDC). It shows that CDC researchers have just completed a new study of the prevalence of (ME)CFS using improved screening methods and more sensitive and specific diagnostic criteria (http://www.cdc.gov/cfs/publications/casedef_10.htm), and that the study has "yielded a significantly higher estimate of the number of people who have (ME)CFS".

As the UK Government has apparently already decided not to spend money on supporting the chronically sick, the last thing Ministers and their advisers want to hear is that the numbers of such chronically sick who suffer from ME/CFS are significantly higher than was previously believed.

An expedient way out of this dilemma for policy-makers and Government advisers seems to be to continue to reject applications for biomedical research that would render untenable the continued denial of ME/CFS as an organic clinical identity, with the consequent corollary that increasing numbers of sufferers would need lifelong medical, social and financial support provided by the State. Therefore, Ministerial advisers have decided that there must be rigidly controlled policy-based evidence and not evidence-based policy. On 22nd June 2005 Laurie Taylor presented a programme on BBC Radio 4 called "Thinking Allowed", which Taylor ended with a momentous statement: "The last word on methodology, and the importance of valid and empirical work, must go to the anonymous political insider who recently characterised the present Government's approach to research in the following manner: **it is not, he said, so much evidence-based policy-making as policy-based evidence-making**".
(see http://www.meactionuk.org.uk/Politically-modified_Research.htm).

The third item comes from a study by gastroenterologists at the world-famous Mayo Clinic in Rochester, US, who report that they have found strong indications for a genetic basis for irritable bowel syndrome (IBS), another chronic disorder whose aetiology some psychiatrists are still wont to claim is psychosocial as opposed to organic. As many as 81% of the fibromyalgia patients studied and 63% of the (ME)CFS patients met the diagnostic criteria for IBS. The Mayo Clinic researchers found that fully 70% of diagnosed IBS patients in their study had one or more family members with symptoms characteristic of IBS.
(http://www.chronicfatiguesyndromesupport.com/library/showarticle.cfm/id/7174)

As consultant physician Dr William Weir said to the Gibson Inquiry at the fourth Oral Evidence session on 10th July 2006: there is a long history of the biopsychosocial (BPS) model of disease being discarded once the evidence is obtained that disproves it. Dr Weir emphasised that the BPS model is a default philosophical posture which some people embrace when they do not know what is going on or do not understand the science. He

pointed out that in the 1980s, the BPS model was claimed to be the cause of AIDS until virologists found the virus.

Finally, in view of the entrenched belief in the BPS model by psychiatrists Professors Simon Wessely, Michael Sharpe and Peter White et al, it may be worth drawing the Gibson Inquiry members' attention once again to the findings by researchers at Georgetown University in the US (as well as by colleagues in Italy). Although dating from December 2005, these researchers believe they have found evidence that (ME)CFS is a legitimate neurological condition. The study revealed that patients with (ME)CFS have a set of proteins in their spinal cord fluid that have not been detected in healthy individuals. Many of the proteins found in (ME)CFS patients are involved in protein folding and in various other neurological syndromes.
(see http://www.biomedcentral.com/1471-2377/5/22)

Thus the evidence continues to mount that the BPS model for ME/CFS is untenable. As Professor Malcolm Hooper noted after attending the first Oral Evidence session on 18th April 2006: "The psychiatric theories of ME, under whatever name, are dead. They have been exposed as flawed and invalid. They must be abandoned forthwith".

It is to be hoped that the Report from the Gibson Inquiry (due in the autumn) will reflect this reality and will recommend (i) that substantial funding be made available by Government agencies for urgent biomedical research to seek a reliable diagnostic marker and (ii) that the continued rejection of high quality biomedical research proposals into ME/CFS (known to have been submitted) should cease, whereupon the controversy (and the distress it causes to those least able to deal with it) would also cease.