Bowel problems in ME/CFS

Margaret Williams 9th August 2014

For those battling to convince their permanent health insurers (PHI) that irritable bowel syndrome (IBS) is a known component of ME/CFS and that neither IBS nor ME/CFS is a functional somatic disorder (and thus excluded from benefit), recent evidence should help dispel any doubt about the organic nature of their disorder(s).

It is a matter of record that the basis of the Wessely School's beliefs about "CFS/ME" upon which the PACE trial was based is that, together with fibromyalgia, irritable bowel syndrome, atypical chest pain and multiple chemical sensitivity, "CFS/ME" is but one functional somatic syndrome (ie. a behavioural / somatisation disorder with no grounding in organic pathology) which, due to an *"artefact of medical specialisation"*, naïve clinicians fail to recognise and thus treat as different disorders (S Wessely, C Nimnuan, M Sharpe, Lancet 1999:354:936-939; S Wessely, Psychol Med 1990:20:35-53). This is what has been taught to medical students for the last three decades.

Furthermore, during the consultation period for the NICE Clinical Guideline CG53 on "CFS" that was published in August 2007, Professor Peter White's psychiatric unit at St Bartholomew's Hospital stated*: " '..gut anti-spasmodics..' are not treatments of CFS/ME since bowel symptoms are not part of CFS/ME"* (SH St Bartholomew's Hospital Chronic Fatigue Services 85 FULL 229 6.4.5.5).

It has been on record since 1987 that IBS is common in post-viral syndromes (ME Association Newsletter Winter 1987-88).

In a study supported by Action for ME, in 1996 it was demonstrated by MJG Farthing, Professor of Gastroenterology at St. Bartholomew's Hospital, that there was a prevalence of 63% of IBS in "CFS" sufferers (Journal of the Royal College of Physicians of London 1996:30:6:512-513). This greatly exceeds the prevalence of IBS of up to 22% in the general population.

In 1998 Hyman and Wasser showed that the gastrointestinal manifestations of "CFS" significantly affects patients' quality of life (JCFS 1998:4 (1):43-52).

In 2000, Aaron et al showed that there is a growing literature of co-morbid illnesses in "CFS", including IBS (Arch Intern Med 2000: 160(2):221-227).

In 2002 Whitehead et al published a systematic review of the co-morbidity of IBS and showed that the non-psychiatric disorders with the best-documented association are fibromyalgia and "CFS" (Gastroenterology 2002:122(4):1140-1156).

In 2003 research presented at the plenary session of the 68th Annual Scientific Meeting of the American College of Gastroenterology in Baltimore by lead investigators Professors Peter Moses and Gary Mawe identified molecular alterations in patients with IBS, showing that key elements of serotonin signalling are changed in IBS, confirming that it is not simply a psychological or social disorder but is due to altered gut biochemistry and interactions between the gut and the brain (Science Daily News Release: University of Vermont, 15th October 2003).

In 2004 Professor Michael Gershon, gastrointestinal expert and Chairman, Department of Anatomy and Cell Biology, College of Physicians & Surgeons, Columbia University, New York, stated: "*IBS has long been classified as a purely psychosomatic condition…Patients may have been treated solely for a condition that was supposedly ' all in their heads'. However, IBS is now associated with a very real abnormality in the gut and one that is as biochemical as any other"* (http://news.biocompare.com/newsstory.asp?id=40849).

Also in 2004, Burnett and Chatterton demonstrated that gastrointestinal symptoms are common in "CFS" patients and are associated with objective changes in upper GI tract motility (BMC Gastroenterology 2004:4:32).

In 2006 the BMJ published an over-view of IBS for its BMJ Learning series, the authors pointing out that: *"A number of pathological 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, dietary factors, inflammation and neurotransmitters"* (PJ Whorwell, Professor of Medicine & Gastroenterology: BMJ 2006:332:280-283).

In 2007 it was shown that regarding IBS 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).

Considerable evidence has continued to emerge of the organic nature of IBS and of its strong co-morbidity with ME/CFS, none of which can be credibly denied.

In July 2014 Hughes et al built on the known evidence of alterations in the neuro-immune axis that contribute towards viscerosensory nerve sensitivity in IBS and proved that it is due to altered immune function: "*Monocyte/macrophages are the predominant immune cell type responsible for ß-endorphin secretion in humans. IBS patients have lower monocyte derived ß-endorphin levels than healthy subjects, causing less inhibition of colonic afferent endings. Consequently, altered immune function contributes towards visceral hypersensitivity in IBS"* (Brain, Behaviour and Immunity 2014).

Here, then, is the evidence that altered immune function is present in IBS, just as has been demonstrated to occur in ME/CFS.

Given the extent of the published evidence that the Wessely School psychiatrists are simply wrong about IBS and CFS/ME being part of one functional somatic syndrome, it is not surprising that people are asking why these psychiatrists are accountable to no-one for the harm they may have done to so many sick and vulnerable people over the last three decades.

Mindful of the evidence-base of organic pathoaetiology now known to be underpinning ME/CFS (and also IBS), it is inconceivable that the interventions used in the PACE trial (ie. CBT or "cognitive restructuring" that was designed to change the way patients think about their disease), and GET (ie. incremental aerobic exercise) could possibly lead to "recovery" as claimed by the PACE trial investigators.

Indeed, in response to Professor Peter White's claims of "recovery" with CBT and GET, Friedberg has recently warned against overstating the capacity of any currently available therapy to produce recovery from (ME)CFS: "*The publicity generated by trumpeting recovery outcomes for CFS far exceeds the relatively modest results found for most patients in behavioural treatment research"* (Reports of recovery in chronic fatigue syndrome may present less than meets the eye. Fred Friedberg and Jenna Adamowicz. Evidence-Based Mental Health, August 2014).