That there is a concerted campaign by members of the Wessely School to re-classify as a single somatoform disorder various disparate syndromes whose aetiology remains undetermined cannot be disputed.

It is worth noting that the British Medical Journal recently carried a well-structured Clinical Review of interstitial cystitis, a condition associated with gross bladder wall changes, and painful bladder syndrome, which exhibits the same symptoms but lacks gross cystoscopic findings (Serge Marinkovic et al; BMJ 8th August 2009:339:337-342). The authors stated that patients with IC are 100 times more likely to have irritable bowel syndrome and are 30 times more likely to have systemic lupus erythematosus, and that other associated chronic illnesses include fibromyalgia and chronic fatigue syndrome. The authors provided a compelling but unconfirmed theory – based on evidence that the authors say represents the majority opinion of researchers actively involved in the field – of likely autoimmune causation: “The pathological features of bladder epithelial damage and related blood vessel transitions in the absence of infection have been recognised for more than 100 years… One theory is that increased permeability of the protective glycosaminoglycan lining of the bladder epithelium causes potassium (and) toxins to leak into the mucosal interstitium, activating mast cells and generating an autoimmune response. Mast cells produce immune reactive chemicals, which in turn cause generalised bladder inflammation and bladder mucosal damage through the presence of tachykinins and cytokines. These further mediate the release of histamine, tumour necrosis factor, chymase, tryptase, and prostaglandins. Finally, inflammatory agents sensitise bladder neurones, producing pelvic and bladder pain…..Some patients have exacerbations of their symptoms after ingesting certain food or dinks….Urothelial cell cultures express abnormal gene variants. When urothelial biopsies…were subjected to stretch…they released significantly higher concentrations of ATP than control biopsies, suggesting that ATP plays an important role in this syndrome. An investigation of cultured bladder urothelial cells…showed that such cells had higher than normal concentrations of ATP, which decreases the ability of the bladder wall to conduct potassium ions…which again indicates that impaired potassium conduction is involved in the pathophysiology of interstitial cystitis’.

Professor Simon Wessely, champion of cognitive behavioural therapy and proponent of the psychosocial model of ME/CFS, seems to reject outright any autoimmune or allergic component: “The article…details associations with fibromyalgia, chronic fatigue syndrome and, strikingly, a 100-fold increased risk of irritable bowel syndrome – all of which have good evidence for the role, at least in part, of psychological factors in the their aetiology or maintenance…It is highly possible that psychological factors have an aetiological contribution to conditions such as painful bladder syndrome. Such disorders, where physical pathology cannot fully account for symptoms, are known as ‘medically unexplained’ or ‘functional’ (somatic) syndromes…It has been proposed (citing his own Lancet paper 1999:354:936-939) that they may be the same underlying disorder manifesting itself in different bodily systems…Dr Marinkovic, however, despite drawing out the evidence for such a description, seems to resist the inference, making no mention of psychological factors even as possible contributors to the aetiology…The experience of other functional somatic syndromes…is that a biopsychosocial approach is the foundation of successful cognitive behavioural therapy. This…surely deserves a place in any review of painful bladder syndrome” (http://www.bmj.com/cgi/eletters/339/jul31_2/b2707#218935).

People must decide for themselves whether or not, based on the evidence, Dr Marinkovic did “draw out the evidence” that IC is a functional somatic disorder, and which of the two theories is the more convincing.