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TO THE EDITOR: The process of destigmatising chronic fatigue syndrome (CFS) is not advanced by either limiting enquiry to “acceptable” sciences or increasing the stigma already experienced by people with other neuropsychiatric disorders. Contrary to its intent, and in contrast to the recently published Royal Australasian College of Physicians (RACP) guidelines,¹ the recent statement by the immediate past president of the RACP and the Chairman of the ME/Chronic Fatigue Syndrome Association of Australia² is in danger of *increasing* the stigma for both people with CFS and people with other common mental disorders.

Unfortunately, key propositions in their letter (“There is no evidence that the illness is primarily psychological in origin”) are clearly at variance with the tone of the guidelines (see Box 1.5, p.S31; Box 1.7, p.S32; and, “Management” summary, p.S38). Their letter reinforces the classical “dualistic” and rather simplistic “biological” approach (eg, “There is significant evidence of a range of biological abnormalities occurring in people with CFS”). Unwittingly, it colludes with community-based beliefs that mental health problems are “not health”,³ and often imaginary or under the voluntary control of the patient.⁴

There is no doubt that people with CFS share many experiences with people with other neuropsychiatric disorders. They both have daily experiences where their credibility is challenged, their disability is minimised and their needs for appropriate medical management are not met.

Australian research and best practice have been recognised internationally for emphasising the integration of psychological, psychiatric and biological factors and respect for the experiences of persons with these debilitating disorders.⁵ Unfortunately, the major advances captured in the guidelines may now be undermined if the RACP is perceived to be backing away from supporting appropriate psychological assessment and provision of effective “psychological” treatments (such as cognitive-behavioural therapy and physical rehabilitation approaches). Similar equivocation has left clinical guideline processes in the United Kingdom in disarray.⁶

As demonstrated recently, prolonged fatigue syndromes are common in the Australian community, and the vast majority of those who seek healthcare services have concurrent depression or anxiety.⁷ Real progress towards destigmatisation,

meaningful research progress and improved health services for people with CFS will only occur when the field is mature enough to deal with the clear relevance of psychological factors. Instead of rejecting “psychological factors” and associated treatments, relevant professional and consumer bodies should now join with the broader community movement towards increased community awareness of common neuropsychiatric disorders, genuine understanding of their (genetic, “biological”, psychosocial and personal) causes and provision of effective (pharmacological and psychological) treatments.⁸

1. Chronic fatigue syndrome. Clinical practice guidelines – 2002. *Med J Aust* 2002; 176 Suppl May 6: S17-S56.
2. Larkins RG, Molesworth SR. Chronic fatigue syndrome clinical practice guidelines [letter]. *Med J Aust* 2002; 177: 51-52.
3. Highet NJ, Hickie IB, Davenport TA. Monitoring awareness of and attitudes to depression in Australia. *Med J Aust* 2002; 176 Suppl May 20: S63-S68.
4. McNair BG, Highet NJ, Hickie IB, Davenport TA. Exploring the perspectives of people whose lives have been affected by depression. *Med J Aust* 2002; 176 Suppl May 20: S69-S76.
5. Lloyd AR, Hickie IB, Loblay RH. Illness or disease? The case of chronic fatigue syndrome. *Med J Aust* 2000; 172: 471-472.
6. Eaton L. Chronic fatigue report delayed as row breaks out over content. *BMJ* 2002; 324: 7.
7. Hickie I, Davenport T, Issakidis C, Andrews G. Neurasthenia revisited. *Br J Psychiatry* 2002; 181: 56-61.
8. Hickie IB. Responding to the Australian experience of depression. *Med J Aust* 2002; 176 Suppl May 20: S61-S62. □

Donald D Beard

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TO THE EDITOR: In the recent letter from Larkins and Molesworth¹ various statements are made on which I would like to comment.

From time to time everyone becomes physically or mentally exhausted, whether or not it is related to activity.

For some people this exhaustion becomes disabling. They deserve understanding and sympathy. We must do everything we possibly can to assist them to recover and to try to find possible causes.

Larkins and Molesworth acknowledge that chronic fatigue syndrome is a serious, disabling illness. When does ordinary exhaustion become disabling?

I would agree that at this stage there is no clinical evidence that the condition is primarily psychological. Nor is there evidence that it is primarily physical. There may be a mixture.

What is the “significant evidence” of a range of biological abnormalities occurring in people with CFS? What are these biological abnormalities and what physiological evidence is there for each one of these abnormalities to produce fatigue?

Larkins and Molesworth state that treatment plans should be “within the capabilities of the patient”: is there evidence to indicate that stimulating each patient to do just that little more each day will do harm?

It was stated that scientific evidence of the aetiology, pathology and treatment is grossly deficient. It is in fact absent. There is no evidence at all. Research is certainly required.

One of the problems is that, as soon as a medical advisor informs a patient that investigations have shown no serious abnormality, the patient often goes away and says to himself or herself or family that the “doctor said there is nothing the matter with me and that it is all in my head”. Nothing could be further from the truth. Something *is* the matter and it is up to us to find it out.

1. Larkins RG, Molesworth SR. Chronic fatigue syndrome clinical practice guidelines [letter]. *Med J Aust* 2002; 177: 51-52. □

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IN REPLY: We thank the writers for their comments on the CFS guidelines¹ and our joint letter about these guidelines.²

Hundertmark remarks on the interplay between physical and psychological factors in morbidity associated with CFS. We trust that our letter in no way contradicts this. Similarly, the inferences that Hickie drew from our letter are not supported by the text of the letter. Far from undermining the guidelines, our letter had the full support of the convenor of the working party responsible for the guidelines.

As clearly discussed in the guidelines, in the absence of specific diagnostic tests it is likely that a range of factors may contribute to the pathogenesis of CFS. Assumption of a primarily “psychological” pathogenesis is as unjustified as assumption of a primary “physical” basis. There are “abnormal” test results in many people with CFS, including abnormalities of the hypothalamic–pituitary–adrenal axis and some abnormalities of immune function. As stated, it is controversial whether such abnormalities are primary or secondary.

While cognitive–behavioural therapy with graded exercise is effective in some patients, the guidelines outline the deficiencies of the evidence which “significantly limit the generalisability of the findings”. As the guidelines indicate, and as is supported by our letter, treatment should be designed in

partnership with the patient, and tailored according to the patient's capacity and response.

Finally, as implied by Beard's letter, we restate the need for further research into the aetiology, pathology and treatment of CFS.

We believe that effective progress in the management of this complex and mysterious illness will be best achieved by positive and cooperative rather than adversarial relationships between those suffering from the condition and the doctors and researchers attempting to help them.

1. Chronic fatigue syndrome. Clinical practice guidelines – 2002. *Med J Aust* 2002; 176 Suppl May 6: S17-S56.
2. Larkins RG, Molesworth SR. Chronic fatigue syndrome clinical practice guidelines [letter]. *Med J Aust* 2002; 177: 51-52. □

Colorectal cancer prevention

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TO THE EDITOR: Bolin et al,¹ in their editorial accompanying articles by Yusoff et al² and Bampton et al,³ took the opportunity to make their case for endoscopic screening for colorectal cancer. We believe that their editorial is seriously misleading.

1: It is misleading to suggest that the 27 case-control and cohort studies in the meta-analysis by Johns and Houston⁴ stratified the index case by age at diagnosis. The 2.25 risk quoted by Bolin et al refers to the overall risk of first-degree relatives in families with one affected relative. In those studies in which age was stratified in the meta-analysis, there is a spectrum of risk, with families with onset of bowel cancer at an older age having lifetime relative risks much less than the average. A subanalysis of seven studies with age stratification showed the risk to be 1.82 (95% CI, 1.47–2.25), where the index case was over 59 years at diagnosis.

Whether a 1.8-fold risk elevation warrants colonoscopic surveillance could be debated. "First do no harm" is an important axiom in well-patient screening, so one should aim for an order of magnitude of benefit over risk, which in this situation is not secured until the patient being screened is older than the suggested 40 years of age.

2: The recommendation that colonoscopic follow-up of patients with only small, tubular, distal adenomas can be at less frequent intervals is not based on the US National Polyp Study,⁵ as suggested in the editorial. It is based on the large cohort

study of Atkin et al,⁶ who reported that patients with this finding were actually at below-average risk (relative risk, 0.5) for subsequent colorectal cancer after prolonged follow-up. This occurred despite the inevitable "miss rates". The editorial by Bolin et al handles this issue unconvincingly. The main message of the US National Polyp Study⁵ was that follow-up (except in exceptional circumstances of numerous polyps, or incomplete removal of malignant polyps) is not needed at 12 months — after 3 years is adequate. The National Health and Medical Research Council guidelines extend this to 4–6 years in the low risk groups, as defined by Atkin et al.⁶ The Atkin et al data, however, are only Level 3 evidence.

3: Bolin et al¹ completely miss the point about pilot programs of screening with faecal occult blood testing (FOBT). There is no intention to confirm evidence of mortality reduction. The pilot studies are neither designed to, nor capable of, doing this. Mortality reduction from FOBT is well established on Level 1 evidence. The pilot studies are in place to answer the very practical questions of

- how to implement large-scale screening programs in Australia;
- what logistic and resource issues are involved;
- how compliance and acceptance will best be secured; and
- how to approach difficult-to-access populations (perhaps with low health insurance rates as distinct from populations well supplied by colonoscopy services).

The central issue in advocating a menu of options to individuals versus more prescriptive screening (based on FOBT) is whether the height of the scientific bar should be at Level 1 evidence or modestly robust Level 3 evidence (flexible sigmoidoscopy) or the less robust Level 3 evidence (colonoscopy), complemented by certain appeals to logic (carefully crafted in the editorial). Medical initiatives based on less than Level 1 evidence have a history of being shown to be wrong, and the concept of colonoscopic surveillance is not immune from this outcome. Where a significant outlay from the public purse is involved, the Federal Government is being appropriately prudent in acting on Level 1 evidence.

1. Bolin TD, Cowen AE, Korman MG. Colorectal cancer prevention [editorial]. *Med J Aust* 2002; 176: 145-146.
2. Yusoff IF, Hoffman NE, Ee HC. Colonoscopic surveillance for family history of colorectal cancer: are NHMRC guidelines being followed? *Med J Aust* 2002; 176: 151-154.
3. Bampton PA, Sandford JJ, Young GP. Applying evidence-based guidelines improves use of colonoscopy resources in patients with a moderate risk of colorectal neoplasia. *Med J Aust* 2002; 176: 155-157.
4. Johns LE, Houston RS. A systematic review and meta-analysis of familial colorectal cancer risk. *Am J Gastroenterol* 2001; 96: 2992-3003.

5. Winawer SJ, Zauber AG, Ho MN, et al. Prevention of colorectal cancer by colonoscopic polypectomy. The National Polyp Study Workgroup. *N Engl J Med* 1993; 329: 1977-1981.

6. Atkin WS, Morson BC, Cuzick J. Long-term risk of colorectal cancer after excision of rectosigmoid adenomas. *N Engl J Med* 1992; 326: 658-662. □

Terry Bolin, Alistair E Cowen, Melvyn G Korman

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IN REPLY: Macrae and Hebbard fail to grasp the concept that colorectal cancer is the only potentially preventable cancer in men and one of the two preventable cancers in women. In any discussion about screening options, this fact must be kept clearly in focus.

In terms of surveillance of first-degree relatives, we doubt the available evidence is of sufficient quality to be certain whether the absolute risk is 1.82 or 2.25. In either event, we would advocate colonoscopic surveillance. We would, however, agree with Macrae and Hebbard on the importance of safety issues. Elsewhere we have advocated confining the performance of colonoscopies to endoscopists with Conjoint Committee Accreditation, and ensuring that the procedures are undertaken in accredited, suitably equipped facilities.¹

We find it difficult to understand why Macrae and Hebbard believe that "the main message of the US National Polyp Study² was that follow-up . . . is not needed at 12 months". Showing a reduction in expected cancers of 90% seems to us a far more important finding. We would, however, point out that our editorial does not advocate routine colonoscopic follow-up at 12 months.

In relation to the pilot faecal occult blood testing (FOBT) studies, we believe that Macrae and Hebbard have missed the point. They advocate delaying colorectal cancer screening for a further five years to await the results of studies which, many believe, will be both outdated and probably inconclusive. Their continued inflexible stand is one that is being rejected by a rapidly increasing number of countries, including the United States, Germany, Italy and the recently formed Global Alliance for the Prevention of Digestive Cancer. Colorectal cancer is the commonest cause of mortality in both non-smoking men and women, with a death from this disease every two hours in Australia.

We suggest that introducing screening is far more urgent than Macrae and Hebbard advocate. We re-emphasise the point that