reserving an immunological assessment for those with new-onset syndromes. The different health care system in the USA, in which patients have more direct access to specialists, gives a different bias, and may have accounted for the contradictory results. It is difficult to establish the true role of past psychiatric history in the genesis of CFS using hospital-based case-control studies.

The authors do not emphasise somatisation as a significant process in CFS, partly on the results of the Illness Behaviour Questionnaire (IBQ). Given the nature of the sample, it is perhaps unwise to put much credence on the results of a questionnaire that includes such unsubtle questions as "If a disease is brought to your attention do you worry about getting it yourself?". However, the authors make one further important clinical observation. They report that the patients firmly believed in the physical nature of their condition, and rejected any psychological contribution. Such observations are in keeping with other studies of the condition (Imboden *et al*, 1959; Wesseley, 1990), emphasised by the classic quotation on neurasthenia at the start of their paper. This suggests an additional characteristic of many chronic sufferers that may be more clinically important than the presence or absence of either immunological or psychiatric disorder.

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SIR: We enjoyed the paper by Hickie *et al* (*Journal*, April 1990, **156**, 534–540), but wish to comment on the interpretation of the results.

Selection of depressed controls should avoid unintentional overlap with CFS patients. Overlap occurs in physical markers such as the VP-1 antigen (a proposed marker of chronic enterovirus infection) with groups such as major depressives (Lynch & Seth, 1989) and those with neuromuscular disorders (Halpin & Wessely, 1989). From further studies we estimate that 30–40% of our depressed controls would show other similar physical abnormalities to CFS patients (Lynch *et al*, 1990, submitted). For these reasons, depressed controls should undergo the same assessment as for CFS and patients with significant physical abnormalities should be excluded.

Secondly, the control group should be homogeneous; in this study, patients possibly with different types and severity of depression are included. We found that in-patient depressed controls had more severe depressive symptoms and fatigue than out-patients, whom CFS patients resemble more in terms of depressive and fatigue severity. We would advocate using out-patients with major depression of milder severity (Lynch & Seth, 1990).

Assessment for both control group and CFS should be initially without medication (antidepressants have quite marked effects on depression and fatigue complaints in previously untreated depressives by the second week of treatment). Other difficulties are whether fatigue should be excluded from diagnostic criteria, as its nature is uncertain in the chronic fatigue syndrome (Wessely & Powell, 1989).

The conclusion that ". . . there is no evidence that CFS is a variant or expression of a depressive disorder . . ." is not justified. The control group used was of typical major depression and findings only hold for this group and not other depressive groups. There are also alternative explanations consistent with the findings on phenomenology and illness behaviour.

Regarding phenomenology; in the analogous situation of atypical facial pain, for example, there is one major symptom of pain, and depressive symptoms may not be obvious. This group would also differ phenomenologically from the depressed controls in Dr Hickie *et al*'s study. This study design cannot in itself refute or confirm whether CFS is an atypical depressive syndrome. The findings on illness behaviour are consistent with those of Wessely & Powell (1989) and Wessely *et al* (1990) in that the differences in attribution of symptoms explain why depressive symptoms such as self-esteem and guilt are more prominent in major depression than CFS. This can be taken to support or refute the above hypothesis concerning CFS. The only certain way of resolving this dilemma is to clarify the nature of fatigue in CFS

and its relation to psychological symptoms and/or physical symptoms over time.

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——, DOLAN, R. & POWELL, R. (1990) Attributions and self esteem in depression and the chronic fatigue syndrome. (abstract) Spring Quarterly Meeting, Royal College of Psychiatrists, University of Galway, April 1990.

SIR: In response to Dr Wessely and Drs Lynch & Seth, we would like to make the following comments. Firstly, when comparing patients with CFS with those with primary depressive disorders, cases of each disorder should be assigned strictly on the basis of published criteria and should not then be reallocated, post hoc, following the identification of some other potentially important biological parameter. Secondly, we agree that depressive controls should be homogeneous, and that they should have a depressive subtype potentially comparable with CFS. As highlighted by Dr Wessely, other researchers have noted the number of patients with CFS who have sufficient depressive symptoms to reach the criteria for 'major depression'. Appropriately, therefore, we contrasted subjects with major (non-melancholic, non-psychotic) depression with patients with CFS to test the hypothesis that the latter have unrecognised 'major depression'. We demonstrated that patients with CFS differed on key clinical variables (i.e. prevalence of pre-morbid psychiatric disorder, current depression severity and neuroticism). Further, others have demonstrated that patients with CFS do not show proposed biological markers of 'major depression' such as non-suppression on the dexamethasone suppression test (Taerk *et al*, 1987) or shortened latency of rapid-eye-movement sleep (Moldofsky, 1990). Having shown that the hypothesis of CFS being a form of 'major depression' is unlikely, some researchers now propose that such patients have 'atypical' depression, whereby they misattribute their somatic symptoms to physical rather than psychological causes and thereby avoid any personal guilt or fall in self-esteem. However, it seems more likely that CFS is not a primary depressive disorder at all, but rather an acquired neuropsychiatric condition in which depressive and other neurocognitive symptoms are prominent (Lloyd *et al*, 1988, 1990).

Dr Wessely takes issue with the selective nature of our sample. We do not see this as a deficit of the study but rather as a major strength. Surely the key research and clinical issue is to distinguish those patients with CFS from the mass of those with non-specific fatigue and other related states encountered in general medical practice, given that the latter may be inappropriately labelled, by the patients themselves or their doctors, as sufferers of CFS.

The essential psychiatric finding in our report was the low rate of *pre-morbid psychiatric disorder*. Importantly, Goldenberg *et al* (1990) have also reported pre-morbid psychiatric disorder to be infrequent. Dr Wessely is particularly concerned about our low rates of other psychiatric disorders, particularly anxiety and somatisation. It may well be that our strict selection process and the tertiary referral nature of the Immunology and Infectious Disease practices from which the patients with CFS were drawn meant that individuals who did not meet our operational criteria for CFS but had primary psychiatric disorders were treated appropriately elsewhere. Thus, the rate of pre-morbid psychiatric disturbance is likely to be influenced strongly by referral biases.

Dr Wessely raises the difficult issue of 'somatisation', a topic that requires clarification. Those with primary depressive disorders often have multiple somatic complaints, but are generally recognised by their psychiatrists as suffering from depression. As discussed, this is clearly not the case with patients with CFS. The proponents of 'somatisation' argue simply that patients with CFS have a type of communication deviance in which they express their dysphoria primarily in a somatic form. This is at best a highly speculative hypothesis. Surprisingly, Dr Wessely takes issue with our use of a well standardised instrument, the Illness Behaviour Questionnaire (IBQ) to evaluate this concept. Lipowsky (1989) has addressed the difficult issue of somatisation within CFS and has warned psychiatrists to avoid simplistic causal hypotheses. Dr Wessely's final statement that the patient's belief in the physical nature of their condition is clinically more important than either concurrent immunological or psychiatric disorder must