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

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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

surely represent his own point of view and one which, like all others, requires empirical evaluation. Whenever patients with non-specific physical complaints fail to demonstrate evidence of a known disorder, psychiatric evaluation is likely to follow. Often this is appropriate. As has been demonstrated in patients with myasthenia gravis (Nicholson *et al*, 1986), however, such patients are often mislabelled by psychiatrists as 'somatisers' until the actual nature of the disorder is later revealed. To avoid that pitfall, psychiatric diagnoses should be restricted to the identification of 'typical' disorders in patients with positive features of psychological disturbance. More doubtful notions such as 'somatisation' should not be invoked haphazardly to conceal a lack of basic knowledge.

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Life events and the onset of mania

SIR: The paper by Sclare & Creed (*Journal*, April 1990, **156**, 508–514) makes some extraordinary claims in terms of its methodological virtues over earlier studies, and particularly selects our early work for comparisons. This would, of course, be quite legitimate, but for the fact that they almost systematically misrepresent our work. I quote from my paper: "The checklist was shortened from earlier versions by the omission of events most subjective in quality". In fact, all events referring to deterioration in relationships were omitted; yet Drs Sclare & Creed state: "whereas Ambelas examined events only during the 28 days before admission (interpersonal conflicts, difficulties at work). It is likely that the events collected were the result of the illness rather than contributing to its cause". Had they really read the paper they could not have failed to notice that most of our events were deaths, examinations, births, and physical illness, and only 25% could possibly be related to manic illness. To avoid misclassification of events as independent, whenever even a faint chance of this occurring existed, patients and, of course, all controls were interviewed; yet Drs Sclare & Creed insist on describing our work as a case-note study. They consider our four-week pre-event period insufficient. Logically, however, the further away one moves from the onset, the more over-inclusive the observations become in terms of existence of events, and the weaker the argument for temporal connections. While the longer period is almost unavoidable for studies of depression, where onset tends to be more insidious, this is hardly the case with the loud onset of mania. They also pointed out that admissions in their cohort did not follow onset very quickly; there was a lag of 22 days. In our series, admissions had taken place in most cases within less than a week. This is certainly the result of the fact that their cohort consisted of well established manic–depressive patients, many already on lithium and presumably in environments well used to their problems, while our patients were in their first ever episode when the impact is very different indeed.

This selection of patients was unfortunate in other ways since it was already known by Stern (1944), and was confirmed by our study, that the longer the duration of the illness, the less likely for life events to be associated with further episodes. Drs Sclare & Creed devoted a whole table and paragraph to the argument that patients in their cohort with less than five years of illness were somehow comparable to our first-admission cases. They are not of course; anything after the first episode is qualitatively different. In our study, patients who had more than one episode were handled as old patients in the follow-up. Furthermore, the selection of a sample of quite late manic repeat episodes cannot be used to argue any points about the onset of mania, which is their chosen title; it only tells us about events and their relationship to onset of late manic episodes. With such selection of index cases their results would have been predicted by most of the existing literature,