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,

and therefore they do not counter the arguments of previous research. Finally, using patients as their own self-controls is not really the best method of controlling. With such methodological differences, the only paper with which theirs is comparable is that of Kennedy *et al* (1983). The only remaining argument to support their claims of superior methodology is that a good instrument (the LEDS) was used. Unfortunately, good tools are never a substitute for correct selection, critical judgment, and a fair design which risks erring on the side of caution rather than drive to a pre-selected outcome.

However, the publication in its current form raises an even more important issue. The paper was presented at a College meeting, and from the floor I had a chance to point out to the presenter how he was consistently misrepresenting my work and what I considered the faults in sampling to be. It obviously made no difference. It is a sad state of affairs when publishability is allowed to over-ride what one might call etiquette if nothing else, and it becomes even sadder when our academic teachers undersign the deed.

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SIR: Our study was essentially a methodological one. Only one previous paper (Chung *et al* 1986) had used the LEDS to collect events and had carefully dated the onset of illness. These authors also failed to find an increased rate of severe events before the onset of mania.

Notwithstanding the above information, Ambelas' study (*Journal*, February 1987, **150**, 235–240) remains a case-note study. He relied on patients' hospital records to elicit life events and to date the onset of mania. We have previously pointed out the drawbacks in this method (*Journal*, June 1987, **150**, 875).

From past studies we had expected to find a relationship between life events and the onset of mania. However, our results indicated that illness episodes in a group of patients with established bipolar disorder did not appear to be preceded by events. Further research using the LEDS in first manic episodes is indicated.

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Racism, diagnosis and treatment

SIR: In their not unuseful analysis of black and white patients admitted to hospital under Section 136 (*Journal*, March 1990, **156**, 373–378), Dunn & Fahy make the surprising statement that it "challenges the findings of Littlewood & Lipsedge (1981), who suggest that treatment is independent of diagnosis in this [black] group". If they are talking about the paper I think they are (for they have got the title wrong in their references), I should point out that not only did Lipsedge and I not make the statement they claim but the study itself is not concerned with psychiatric treatment at all. More importantly, they are somewhat cavalier about dismissing the possibility of racism in their own data. Selective discrimination on the grounds of perceived ethnic status, whether in medicine or in other institutions, or in everyday encounters, is not something best seen as located in individual attitudes, but in the whole way social action in a society may operate. In the review which they cite (Littlewood, 1986), I was at pains to point out that racial bias in psychiatry is hardly likely to be a conscious and overt penalisation of certain patients. The alternative, however, is not to associate differences in diagnosis and treatment simply with differences located in the patients themselves. We need, painstakingly, to reconstruct how certain assumptions, behaviours and actions of individuals, both the putative patients and the professionals, interact in certain contexts. Such studies require surveys of theoretical assumptions, attitudes to the other, and perceived dangerousness: studies using video material of actual instances, besides an examination of the political economy of psychiatry itself.

To take an instance. One possibility is that doctors, whether through their training or through