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CORRESPONDENCE

Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Immunity and depression

SIR,—I am concerned that Dr A M Denman concludes that there is little evidence of appreciably altered immunity in depressed subjects (23 August, p 464).

Physicians and psychiatrists who suspect that the mind and body may be more closely linked than we have yet been able to show will no doubt find this interesting. It is therefore surprising that there are only three studies of cellular immunity in depressed subjects. Of these, Dr Denman has quoted the work of Schieifer *et al* and Albrecht *et al*[1,2] but not the findings of Kronfol *et al*.[3] This is especially important as Kronfol and workers did find decreased proliferative responses in subjects who were less depressed than the patients of both Albrecht *et al* and Schieifer *et al*. This would argue that severity of depression alone cannot explain the findings. Any inference that might be drawn from the negative findings of Albrecht *et al* should be tempered by an awareness that their subjects were drug free for only one week.

A further criticism levelled at these studies was that they did not take into account diet, treatment, and life style. However, as with the earlier work of Bartrop *et al* and Schieifer *et al* on bereaved subjects,[4,5] they did consider drug intake. The part that diet, sleep disturbance, and exercise play in immunocompetence has been poorly tested. When done it was concentrated on extremes,[6-8] which are bound to cause physiological responses quite different from the disturbances that might be expected in a moderately depressed subject. The problem is undoubtedly complex, but when found, lymphocyte depression has not correlated with dexamethasone treatment.[2] Recent work on the relation of immune function to sleep in man describes changes in natural killer cell and interleukin activity,[13] both of which seem to be independent of the diurnal cortisol rhythm. We clearly need to know a great deal more about the impact that sleep disturbance would have on subjects with morbid mental states.

Dr Denman's insistence that future study should analyse the responses more closely linked to relevant host defence is justified. To date no one has looked at more specific antimicrobial measures of immunity. In depressed subjects there has been only one study of subset ratios,[2] and its findings were negative. However, several studies have shown alteration in natural killer cell activity and altered ratios of helper to suppressor cells in times of stress.[9-12] Although anxiety and depression were not measured in these studies, one might well speculate about the Hamilton rating scale scores of such subjects.

It is clearly correct to question the pathophysiological relevance of depressed proliferative responses to mitogens. To imply that "there are those who believe that altered immunity predisposes to depression," however, is to misrepresent the views of those working in this subject. The work of Murphy and Brown[14] amply demonstrates the caution with which psychiatrists approach the subject, and few of them suggest specious explanations for psychosomatic illness.

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