Author's reply: Amminger *et al* raise some interesting issues. I certainly agree that the estimation of premorbid IQ, particularly in patients with schizophrenia, is challenging and that further validation studies on methods for making such estimates should be pursued.

More specifically with reference to our earlier paper on the relationship of DUP to cognitive functioning (Norman et al, 2001), Amminger et al argue for the likely superiority of Bilder et al's (1992) index as a measure of cognitive deterioration in contrast to estimates based on NARTestimated premorbid IQ minus current WAIS full-scale IQ. In this respect they note that 38.1% of patients in a recent study by their group showed higher current IQ than NART-estimated premorbid IQ. This would, of course, suggest an increase in IQ after illness onset - an unlikely occurrence. I have examined this issue in our data-set and found such a pattern in 17.8% of our sample, with the average discrepancy among these individuals being 8.4 points. I can also confirm that in our sample, as in Amminger et al's sample, NART scores were correlated with age at admission (r=0.24, P<0.05), but WAIS-R full-scale scores were not.

The substantive question, of course, is whether DUP is related to cognitive deterioration. Amminger et al report that they have found DUP related to deterioration based on Bilder's index. We had reported some results using Bilder's index in our earlier paper. I will take this opportunity to report further that when we examined correlations between our two indices of DUP and Bilder's deterioration index they were non-significant (r=0.06 and r=0.04). We are currently pursuing the issue of whether DUP may be related to recovery of cognitive functioning during the first year of treatment.

The discrepancy between our earlier findings and those of Dr Amminger and colleagues does not appear to be explained on the basis of use of the NART rather than Bilder index. Other variables related to sample composition may be relevant. Also of potential importance is the method of measuring DUP, which, as has been suggested elsewhere (Norman & Malla, 2001), also needs to be more carefully considered and standardised. In this, as in many areas of psychiatric research, cumulative progress is dependent on careful and comparable measurement across studies. I endorse Amminger *et al*'s comments in this respect.

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Depression: detection and diagnosis

The October 2001 issue of the Journal reports two prevalence studies of depressive disorders (Ayuso-Mateos et al, 2001; Thompson et al, 2001). Both studies used a self-report questionnaire as an initial screening device although both avoided the sometimes reported but unjustified assertion of diagnosis based upon scores of the scales. Such scales are widely used in the manner reported by these studies and a cautionary comment is in order. There is a widespread view that the selection of instrument is unimportant so long as it is designated as a 'depression' scale; this is not true. For instance, the scales used in the above-mentioned studies were the Beck Depression Inventory (BDI) (in Ayuso-Mateos et al, 2001) and the depression sub-scale of the Hospital Anxiety and Depression scale (HAD-D) (in Thompson et al, 2001). These two instruments highlight very different aspects of depressive disorders (Snaith, 1993). The HAD-D has 86% of its variance directed to mood symptoms (depressed mood and anhedonia) but an absence of cognitive symptoms (hopelessness, low self-esteem and guilt ideation). With the BDI the reverse is the case, with 14% directed to mood and/or anhedonia but 33% focusing on the cognitive symptoms.

There is an unfortunate tendency to refute the importance of difference based upon predominant psychopathology and even, within the realm of depressive disorders, to deny the importance of diagnosis. Indeed, the first study uses the term 'prejudice' when referring to the separation of disorders and frankly advocates the conflation of disorders of major depression (for which one or other of the mood symptoms is prerequisite for diagnosis) and the other group of 'dysthymia and adjustment disorders', which are characterised by the cognitive distortion. Until diagnostic practice is based on exact psychopathology, research will remain in its present state of confusion. For instance, the oft-repeated statement that cognitive therapy and biological treatments are of equal worth in the treatment of 'depression' will continue to be made. The statement may be true if no distinction is made between different depressive disorders but non-responders to the one or other treatment will have different characteristics: the psychotherapeutic approach will be more successful in the disorders based on cognitive distortion whereas the biological treatments are likely to be more effective when major depressive disorder is present.

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Snaith, P. (1993) What do depression rating scales measure? British Journal of Psychiatry, 163, 293–298.

Thompson, C., Ostler, K., Peveler, R. C., et al (2001) Dimensional perspective on the recognition of depressive symptoms in primary care. The Hampshire Depression Project 3. *British Journal of Psychiatry*, **179**, 317–323.

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Factor structure of the Hospital Anxiety and Depression (HAD) scale

We would like to draw attention to the assertion by Mykletun *et al* (2001) that a two-factor structure best fits the Hospital Anxiety and Depression (HAD) scale, especially in individuals with mental problems. They stated that psychometric studies of this scale only involved small samples of non-psychiatric patients. However, we recently published the only factor analysis of the HAD scale based on a large population: 2669 'HAD completers' from 3002 patients (89%) with major depression, DSM–IV criteria (Friedman *et al*, 2001).

Contrary to Mykletun *et al*, we found a three-factor solution using principalcomponents analysis with factors defined by eigenvalues ≥ 1 . One of Mykletun