We hope shortly to be able to address the issue of the Italian psychiatric system more directly, as for two years now it has been the subject of a large-scale evaluative research program by the National Research Council, in which this Laboratory is taking part.

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References

MOSHER, L. R. (1982) Italy's Revolutionary Mental Health Law: an assessment. *American Journal of Psychiatry*, **139**, 199–203.

TOGNONI, G. & BIGNAMI, G. (1981) The confounding role of institution in scientific evaluation. In *Epidemiological Impact of Psychotropic Drugs*, (eds. G. Tognoni, C. Bellantuono and M. Lader). Amsterdam: Elsevier.

REVERSIBLE DEMENTIA AND DEPRESSION

DEAR SIR,

The paper by Rabins *et al* (*Journal*, May 1984, **144**, 488–92) emphasizes depression as a treatable cause of dementia and is a welcome move away from the illogical and somewhat misleading concept of 'pseudodementia'.

In their discussion the authors have acknowledged that depression can lead to cognitive change amounting to dementia; that primary dementing illness can produce depressive symptoms; and that a depressive illness may produce cognitive disturbance that is superimposed upon a dementia.

There is a further possible explanation for the co-existence of depression and dementia *viz.* brain dysfunction that gives rise to both dementia and depression. Rabins *et al* have noted that three of their patients remained cognitively impaired even after the depression was treated. Several of the series they have referred to (Nott and Fleminger, 1975; Ron *et al*, 1979; Wells, 1979) have suggested that the incidence of brain damage or dysfunction in terms of psychometry, EEG and PEG in patients with reversible or non-progressive dementia was greater than might be found in uncomplicated depression or other 'functional' illness. There is no evidence that these reverse with the resolution of the depressive or other illness. Unfortunately, we have no knowledge of the histopathological status of patients with depressive dementia. In showing that elderly depressives did not show significantly greater neuropathology than the normal elderly, Tomlinson *et al* (1968), of course referred to those depressives who did not show significant cognitive change.

Furthermore, several of the series referred to by the authors (e.g. Kiloh, 1961; Marsden and Harrison, 1972; Wells, 1979) have also indicated that dementia may occur at least in association with other 'functional' psychiatric illness, depression merely being the commonest such disorder. The explanation for dementia following depression may not be applicable to all those instances.

Hence it would be useful to bear in mind the possibility that some instances of dementia with depression may well be cases displaying dementia *and* depression as common phenomena of underlying pathology. Alzheimer's disease and Parkinson's disease are two conditions where dementia and depression possibly reflect brain pathology, with, of course, a more progressive course.

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References

KILOH, L. G. (1961) Pseudo-dementia. *Acta Psychiatrica Scandinavica*, **37**, 336–51.

MARSDEN, C. D. & HARRISON, M. J. G. (1972) Outcome of investigation of patients with presenile dementia. *British Medical Journal*, **2**, 249–52.

NOTT, P. N. & FLEMINGER, J. J. (1975) Presenile dementia: the difficulties of early diagnosis. *Acta Psychiatrica Scandinavica*, **41**, 210–17.

RON, M. A., TOONE, B. K., GARRALDA, M. E. & LISHMAN, W. A. (1979) Diagnostic accuracy in presenile dementia. *British Journal of Psychiatry*, **134**, 161–8.

TOMLINSON, B. E., BLESSED, G. & ROTH, M. (1968) Observations on the brains of non-demented old people. *Journal of the Neurological Sciences*, **7**, 331–56.

WELLS, C. E. (1979) Pseudodementia. *American Journal of Psychiatry*, **136**, 895–900.

DEAR SIR,

The longitudinal study by Rabin *et al* (*Journal*, May 1984, **144**, 488–92) is of importance for demonstrating that the cognitive deficits associated with depression in the elderly are, in general, likely to improve with resolution of the depression, which emphasises the need to avoid the misdiagnosis of depression as dementia in elderly patients. However, it seems unlikely that the patients in the 'demented—depressed' group of this study would have been diagnosed as demented by a clinician taking a full history and performing a competent mental state examination. Around two-thirds of this group had a previous history of affective disorder, four-fifths had delusions with a depressive content, one-fifth had a family history of affective disorder and symptoms of depressed mood, appetite and sleep disturbance were