et al, 1998). The prevalence of affective disorders (major depression, dysthymia, mania, hypomania and bipolar disorder) was reported as only 1.7% among those aged 65 years and over, while it was 5.8% among the total adult population. Because mental disorders were reported in this survey to have a much higher prevalence among young people (over 20% at age 18–44 years, compared with 6.1% after age 65 years), the Minister for Health emphasised the need for a national focus on mental health services for young people. The New South Wales Department of Health referred to the same survey and commented on the lower mental health morbidity of older people, in a document describing plans for the distribution of resources.

Survey reports can be misinterpreted. Clinicians and administrators may need to be reminded that treatment for depression may be of benefit to far more older people than just those with a diagnosis of major depression. In this context, I agree that some prevalence studies should be recognised (at least by budget-holders) as "not that interesting".

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Stereotypes of ageing

Sir: Copeland *et al* (1999) concluded that depressive symptoms in older people do not uphold the stereotypes of old age. However, the study itself seems to preconceive the stereotype that 'older people are asexual'. This is contrary to the evidence that sex continues to play an important part in the lives of both men and women, at least until the mid-seventies, with little if any decline in enjoyment and satisfaction (Kinsey *et al*, 1953; Kellett, 1996). Sexual dysfunction is well recognised as a symptom of depression in the ICD–10 and in the DSM–IV as well as in community studies of the depressed elderly (Kivela & Pahkala, 1988). We regret to note that the new EURO–D scale (Prince *et al*, 1999) also accepts this stereotype by ignoring sexual dysfunction as a symptom of depression in the elderly.

Copeland, J. R. M., Beekman, A. T. F., Dewey, M. E., *et al* (1999) Cross-cultural comparison of depressive symptoms in Europe does not support stereotypes of ageing. *British Journal of Psychiatry*, **174**, 322–329.

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Kinsey, A. C., Pomeroy, W. B., Martin, C. E., *et al* (1953) *Sexual Behaviour in the Human Female*. Philadelphia, PA: W. B. Saunders.

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Prince, M. J., Reischies, F., Beekman, A. T. F., *et al* (1999) Development of the EURO–D scale – a European Union initiative to compare symptoms of depression in 14 European centres. *British Journal of Psychiatry*, **174**, 330–338.

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Author's reply: I will start by trying to answer the last point raised by Drs Praseedom, Tube, Vourdas, Rafnar and Woodfield. The purpose of the EURO–D Scale is to harmonise some existing scales of depression. Rightly or wrongly, such scales for older people tend not to include a question about sexual activity, which is why EURO–D itself could not include it. Similarly, the fact that sexual activity was not discussed in the article should not be interpreted to mean that the authors do not regard it as important in older people. The items discussed were limited to those which had been recorded by sufficient centres to make comparison possible. Sexual activity was not one of these. In a longitudinal study in which it is proposed to burden the respondent with later interviews, the great fear of research workers is that of refusal to proceed after the first interview. Questions on sexual activity were removed from the community versions of the Geriatric Mental State when it was found to upset a small proportion of respondents. They can be included if wished. Also, although intrinsically interesting, it is not a symptom essential for diagnosis.

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Old age forensic psychiatry

Sir: The editorial by Yorston (1999), while thought-provoking in that it highlights the potential creation of a sub-speciality, is not backed by robust arguments. Simply quoting that crime is rarely associated with the elderly is not an argument in favour of creating a sub-speciality within forensic psychiatry. In our experience, there is enough evidence to support the view that resources, if available, should be concentrated on other subgroups of patients (i.e. young offenders, women) among whom there is a well-established lack of resources to meet an increasing demand.

The author seeks to create an impression of a distinct lack of inter-speciality collaboration in respect of forensic psychiatry issues pertaining to the elderly. This surprises us as it has not been our experience, certainly at local level. When appropriate, there is close and open communication between the old age and regional forensic psychiatry services. This inter-speciality collaboration helps to identify potential problems and allows for little delay when intervention is required to respond and manage such cases as they present, particularly in the community. This has, in the past, allowed for ease of passage through the medium secure unit.

One might consider that medium secure units are not ideal environments to meet the needs of the elderly, but to extend this, as the author suggests, to a declaration of unmet needs is too sweeping a statement.

From a training viewpoint, we would expect those in higher forensic training to have appropriate experience, in both assessing and treating elderly offenders. Current higher training programmes in forensic psychiatry should offer experience in sub-specialities such as child and adolescent, learning disability, drug and alcohol, prison, and old age psychiatry.

To conclude, the notion that elderly offenders are missing out in terms of specialist assessment and treatment does not, we believe, hold true.

Yorston, G. (1999) Aged and dangerous. Old age forensic psychiatry. *British Journal of Psychiatry*, **174**, 193–195.

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Cytosolic phospholipase A_2 gene in schizophrenia

Sir: Walker *et al* (1999) present an excellent, and much needed, overview of altered lipid

metabolism and schizophrenia. In their effort to summarise a complicated field some detail was lost, which leads to the false impression that the original cytosolic phospholipase A_2 gene study (Hudson *et al*, 1996) was not replicated. A brief review of the literature supports cytosolic phospholipase A_2 as a candidate gene for schizophrenia.

Cytosolic phospholipase A_2, unlike many other phospholipase A_2 enzymes, possesses a number of properties which suggest an important role in cellular signal transduction in schizophrenia: migration to the membrane when activated by a variety of signals such as changes in intracellular calcium concentration; specificity as to the fatty acid at the second carbon of the phospholipid moiety that initiates production of prostaglandins and other lipid-based messengers; regulation by dopamine and glutamate (neurotransmitters implicated in schizophrenia).

Our original genetic study into cytosolic phospholipase A_2 in schizophrenia was in fact two separate analyses (Hudson *et al*, 1996). Initially, an association-type study compared 65 patients with schizophrenia with matched healthy controls and found an association between a marker near the cytosolic phospholipase A_2 gene and schizophrenia. Spurious results may arise with association-type studies and, therefore, we undertook a haplotype relative risk study of 44 families including a proband with schizophrenia, which resulted in the same positive association. A more recent study employing a second marker actually within the intron of the cytosolic phospholipase A_2 gene also found an association between cytosolic phospholipase A_2 and schizophrenia (Peet *et al*, 1998). Again, a haplotype relative risk study of 50 families replicated this finding (Wei *et al*, 1998). Other studies (Price *et al*, 1997) employing association strategies on smaller sample sizes have not replicated our findings.

The majority of genetic data and biochemical data to date suggest the cytosolic phospholipase A_2 gene on chromosome 1 plays a role in increased vulnerability to schizophrenia. The precise determination of the specific phospholipase A_2 enzyme(s) involved in schizophrenia may well prove critical in the development of lipid-based strategies for improved treatment of schizophrenia.

Hudson, C. J., Kennedy, J. L., Gotowiec, A., *et al* (1996) Genetic variant near cytosolic phospholipase A_2 associated with schizophrenia. *Schizophrenia Research*, **21**, 111–116.

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Price, S., Fox, H., St Clair, *et al* (1997) Lack of association between schizophrenia and a polymorphism close to cytosolic phospholipase A2 gene. *Psychiatric Genetics*, **7**, 111–140.

Walker, N. P., Fox, H. C. & Whalley, L. J. (1999) Lipids and schizophrenia. *British Journal of Psychiatry*, **174**, 101–104.

Wei, J., Lee, K. H. & Hemmings, G. P. (1998) Is the cPLA2 gene associated with schizophrenia? *Molecular Psychiatry*, **3**, 480–481.

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Hallucinatory assumptions

Sir: Feinberg & Guazelli (1999) constructed an elaborate theory of subcortical motor system dysfunction to explain schizophrenia. Whether or not it explains some symptoms, it certainly cannot explain schizophrenic auditory hallucinations for two fundamental reasons: non-verbal auditory hallucinations occur in many disparate non-psychotic states, and complex verbal auditory hallucinations occur in all psychoses.

Feinberg & Guazelli divided auditory hallucinations into four broad classes, despite conceding that there can be phenomenological overlap, and that such a classification was oversimplified. If Occam is right, as he usually is, then this scheme is overcomplicated. The literature on musical auditory hallucinations (Gordon, 1997) is quite clear that their basic phenomenology is similar, whether these occur in psychosis, delirium, ear disease, mysticism, intoxication, fever, etc. A simple common explanation was offered, namely inner-ear hyperirritability. However, one does not have to accept this explanation to realise that there must be some unitary mechanism for the production of auditory hallucinations, although of course their interpretation will vary enormously depending on psychiatric, religious or medical context. Anyone arguing that voices have nothing to do with music needs to show that these auditory hallucinations have different causes. If anyone knows of any diseases, risk makers or risk markers associated with verbal but not non-verbal auditory hallucinations (music, noises, tinnitus), please could they let me know, as I cannot find any. Feinberg & Guazelli cite Kraepelin's 1919 book which has detailed descriptions of all sorts of simple and complex auditory hallucinations in schizophrenia.

Verbal auditory hallucinations can become particularly complex in schizophrenia when associated with loss of insight or incorporated in delusional systems. Peralta & Cuesta (1999) convincingly showed that first-rank symptoms, particularly voices, are just as common in six other psychoses as in schizophrenia. Hence, Feinberg & Guazelli's theory, however plausible for schizophrenia, is a non-starter for explaining auditory hallucinations, since it cannot explain first-rank symptoms in non-schizophrenic psychoses. All other psychological theories specific to schizophrenia are equally suspect, especially linguistic ones which, in addition, make no attempt to explain non-verbal auditory hallucinations.

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Gordon, A. G. (1997) Do musical hallucinations always arise from the inner ear? *Medical Hypotheses*, **49**, 111–122.

Peralta, V. & Cuesta, M. J. (1999) Diagnostic significance of Schneider's first-rank symptoms in schizophrenia. Comparative study between schizophrenic and non-schizophrenic psychotic disorders. *British Journal of Psychiatry*, **174**, 243–248.

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Amiodarone and psychiatric symptoms

Sir: The pharmacokinetics of the class III antiarrhythmic amiodarone make it unlikely that its withdrawal was the reason for the dramatic clinical improvement observed within one week in the patient described by Ambrose & Salib (1999).

Amiodarone, a highly lipophilic drug, is extensively distributed into tissues, with a half-life as long as 100 days. Its therapeutic effect can last in excess of one month after withdrawal of long-term oral therapy (Latini *et al*, 1984).

If amiodarone is implicated in triggering psychiatric symptoms, then I would propose that the benefits of stopping this agent might not be observed until many weeks or even months after withdrawal of therapy.

Ambrose, A. & Salib, E. (1999) Amiodarone-induced depression. *British Journal of Psychiatry*, **174**, 366–367.

https://doi.org/10.1017/S0007125000153516 Published online by Cambridge University Press