of the early intervention practitioners. They describe straightforward psychosocial and pharmacological therapies that should be used by all multidisciplinary teams. The only distinguishing feature is the sub-specialists' touching faith in the effectiveness of antipsychotic medicines, which presumably arises from lack of prolonged experience with individual patients.

This inadequate experience of chronic illness is certain to lead to tragedies in the UK. The chosen remit of early intervention practitioners is to assist patients during the first 3 years of illness (Birchwood *et al*, 1998) – unless case-loads are high, when the 'critical period' can be reduced to 18 months (McGorry *et al*, 1999). When relapses occur, ordinary in-patient and community teams will, of course, have to pick up the pieces and I am in no doubt that they will be criticised for not being as attentive and caring as previous keyworkers.

Community mental health teams do not 'inevitably focus on the needs of "prevalent" rather than "incident" cases'. Those who are definitely – or probably, or possibly – in the early stages of psychosis are high in their list of priorities. Unlike Manchanda *et al*, they do not require 'controlled trials to assess the efficacy of early intervention'. These patients are unwell and they all require prompt and appropriate treatment. One of the most important tasks of consultant psychiatrists is to prioritise according to clinical need and it is frustrating when diversion of resources to highly protected teams makes difficult decisions even more painful. Your correspondents are shirking their responsibilities in depending on central planning to protect their case-loads (Milner, 2003; Owen, 2003). Valuable work has been done in this area (Kennedy & Griffiths, 2001) and training would be available for any sub-specialist who returns to mainstream practice.

The introduction of early intervention teams in the UK should now be halted. This will provide an opportunity for proper scientific evaluation by comparing the processes and outcomes of care in areas where these teams have and have not been established. It will also free up some financial and human resources for serious hospital and community psychiatry.

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Testosterone and psychosis

Increased testosterone may be the cause of the finding of Sundquist *et al* (2004) that 'A high level of urbanisation is associated with increased risk of psychosis and depression for both women and men'. Two hypotheses are required to explain this.

It is my hypothesis (Howard, 2001*a*) that human evolution is driven by testosterone. Based on this, I suggest the 'secular trend', the increase in size and early puberty of children, is actually an increase in the percentage of individuals of higher testosterone. The trend may actually be a change in percentage of individuals within our populations and their 'characteristics' may also be increasing. This phenomenon occurs when a 'feed and breed' environment occurs. In these situations, individuals of higher testosterone, both men and women, will increase more rapidly than those of lower testosterone over time. (Individuals of higher testosterone are more aggressive and sexual.) Urban areas are 'feed and breed' centres; I suggest urban centres are areas of higher testosterone.

I hypothesise that dehydroepiandrosterone (DHEA) is directly involved in growth and development, and subsequent maintenance, of all tissues, especially the brain. (The large brain of mammals may have resulted from an evolutionary increase in DHEA; Howard, 2001*b*.) Numerous reports of beneficial effects of DHEA on neurons and tissue-level structures of the brain exist in the literature. I have suggested in the past that depression and schizophrenia, among other mental disorders, result from low DHEA during growth and development, subsequently exposed by adverse circumstances during maintenance. In depression and schizophrenia DHEA is low. Two other hormones may adversely affect the function or availability of DHEA: cortisol and testosterone. Over the past few years a connection with low DHEA, along with increased cortisol, has been demonstrated regarding depression. It is known that schizophrenia is often characterised as resulting from a non-causal, but significant, stressful event (cortisol) usually beginning in the late teens or early twenties (testosterone of puberty, in men and women, along with the natural decline of DHEA which begins at around age 20). In individuals of low DHEA, increased cortisol and testosterone may expose underlying, silent pathology.

Therefore, I suggest that increased rates of psychoses and depression in urban areas may be the product of increased stress and testosterone in both men and women. As suggested above, the secular trend may be due to increasing numbers of individuals of higher testosterone. This increase in these individuals of higher testosterone, along with increasing stress of urbanisation, may account for the findings of Sundquist *et al*, as well as reports of recent increases in these mental disorders.

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Sundquist, K., Frank, G. & Sundquist, J. (2004) Urbanisation and incidence of psychosis and depression. Follow-up study of 4.4 million women and men in Sweden. *British Journal of Psychiatry*, **184**, 293–298.

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Neurosurgery for mental disorder

Dr Persaud provides an ardent but ultimately flawed argument in favour of allowing neurosurgery for mental disorder (NMD) to die out (Persaud/Crossley & Freeman, 2003).

Patients who are considered for NMD are among the most severely ill and disabled who come into contact with any branch of the medical profession, and such presentations merit conceptualisation as rather more than having 'psychological problems'.

It is also disingenuous to argue that 'psychosurgery' (*sic*) tries to locate complex psychiatric disorders in 'one so-called "abnormal" brain region'. Such hangovers

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from Cartesian dualism fail to advance clinical neuroscience or the practice of psychiatry. Dr Persaud will, of course, be aware of the compelling evidence for changes in brain function and structure in both depression and obsessive–compulsive disorder, the main indications for NMD (Drevets, 1998; Szeszko *et al*, 1999).

The argument that there is a lack of randomised controlled trial (RCT) data to support NMD applies equally to a range of 'cutting edge' medical and surgical procedures. The proportions of medical and surgical treatments based on RCT data are 53% and 24%, respectively (Ellis *et al*, 1995; Howes *et al*, 1997). In such situations, prospective clinical audit becomes the tool of choice. If Dr Persaud demands that NMD cease because of the absence of robust RCT support, then he must surely demand the same rigour from other interventions such as heart transplantation or dynamic psychotherapy.

With respect to the issue of consent, in Scotland NMD does not take place unless the patient provides informed consent and the Mental Welfare Commission for Scotland agrees both that it is an appropriate treatment and that consent is valid. Regrettably, Dr Persaud continues to trade on the outdated image of patients receiving NMD against their wishes. Indeed, he implies that chronic intractable mental illness robs patients of their capacity to provide informed consent. It is demeaning to assert that individuals are incapable of evaluating the risks and benefits of a treatment simply because they have a mental illness. Perhaps it is the failure to appreciate this perspective that leads to excessive concern for the 'stigmatised profession of psychiatry'? Believing ourselves to be persecuted perpetuates outdated views of psychiatry, and does nothing to reduce the stigma of mental illness.

Declaration of interest

K.M. has received payment for lectures on the management of depression from various pharmaceutical companies. K.M. and M.S.E. run the Dundee Neurosurgery for Mental Disorders Service.

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Author's reply: My necessarily abbreviated arguments against the continued practice of NMD are intended to be within the spirit of the debate section of the *Journal*. A debate necessarily requires two sides. Given that the title of the debate I was given included the term 'mental disorder' I am confused that an objection should be raised to my nod towards the well-recognised controversy over the modern phrenological localisation of psychiatric disorder. But I am perhaps mostly perplexed by the failure to see that the use of an irreversible surgical treatment directly applied to the brain necessarily demands much higher standards of certainty over its benefits than something like dynamic psychotherapy, particularly given the political context of a profession with obvious public image difficulties. Anyone aware of the widespread coverage that our debate received in the Scottish newspapers would be immediately impressed by this public relations context, which is precisely the area the coverage focused on.

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Stigma and somatisation

In their exhaustive review of the impact of globalisation and culture on depression, Bhugra & Mastrogianni (2004) highlight the role of somatisation in many parts of the world, where it often accounts for 'common presenting features of depression' (p. 16). Emphasising both the ubiquity and cultural aspects of somatisation, they cite an earlier characterisation of common mental disorders that refers to the 'black box of somatisation' (Bhui, 1999). In doing so, however, they miss an important explanatory feature of this process with substantial practical and clinical significance – that is, the role of stigma. Despite increasing availability of effective treatments, many people with depression (perhaps even a majority) do not seek professional help because of the stigma associated with the illness. Efforts to clarify the impact of stigma are crucial for explaining cultural aspects of illness-related experience and meaning, and highly relevant for planning interventions that are culturally appropriate and locally effective.

As one effort towards elucidating the experience of depression, in a study in Bangalore, India, we examined the role of self-perceived stigma (Raguram *et al*, 1996). We found that greater severity of depression was associated with higher stigma scores, but more somatisation was associated with less stigma. Through qualitative analysis of patients' narratives, we also demonstrated that patients viewed depressive, but not somatic, symptoms as socially disadvantageous. Somatic symptoms were considered to be less stigmatising since they resembled illness experiences that most people could expect to have from time to time. Consequently, studying the work of culture clarifies the nature of somatisation. From a Western vantage point, somatisation may appear enigmatic, but attention to stigma helps to illuminate the internal structure of the black box.

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Author's reply: Drs Raguram and Weiss are right to point out the role stigma plays in help-seeking. We agree that many people with depression will not seek help from Western medical sources. The problem

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