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.

Birchwood, M., Todd, P. & Jackson, C. (1998) Early intervention in psychosis. The critical period hypothesis. *British Journal of Psychiatry*, **172** (suppl. 33), 53–59.

Goode, E. (1999) Doctors try a bold move against schizophrenia. *New York Times*, 7 December.

Kennedy, P. & Griffiths, H. (2001) General psychiatrists discovering a new role for a new era . . . and removing work stress. *British Journal of Psychiatry*, **179**, 283–285.

McGorry, P. D., Edwards, J. & Pennell, K. (1999) Sharpening the focus: early intervention in the real world. In *The Recognition and Management of Early Psychosis* (eds P. D. McGorry & H. J. Jackson). Cambridge: Cambridge University Press.

Milner, E. C. R. (2003) Invest in CMHTs or sub-specialist teams? *British Journal of Psychiatry*, eLetter, 4 March.

Owen, A. (2003) What is early intervention? (letter) *British Journal of Psychiatry*, **183**, 562.

A. J. Pelosi Hairmyres Hospital, East Kilbride G75 8RG, UK. E-mail: anthonypelosi@compuserve.com

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.

Howard, J. (2001*a*) Androgens in human evolution. A new explanation of human evolution. *Rivista di Biologia*, **94**, 345–362.

Howard, J. (2001*b*) Hormones in mammalian evolution. *Rivista di Biologia*, **94**, 177–183.

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.

J. M. Howard 1037 North Woolsey Avenue, Fayetteville, Arkansas 72701-2046, USA. E-mail: jmhoward@anthropogeny.com

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

https://doi.org/10.1192/bjp.185.2.173 Published online by Cambridge University Press