Moreover, data on some 788 subjects (ITT 762), or about 37% of the meta-analysis population, come from studies published only in abstract form (Salinas *et al*, 1997; Rudolph *et al*, 1998), and the results of each must be placed in perspective. The 8-week study with some 323 patients (15% of the meta-analysis pool) by Salinas *et al* (1997) comparing venlafaxine extended release, paroxetine and placebo found no significant difference between drugs and placebo. In addition, there was a markedly greater discontinuation rate in the paroxetine group than in the venlafaxine 75 mg group (35% *v.* 20%). In an ITT last-observation-carried-forward analysis, such a difference in discontinuation rates could significantly affect the rates of response and remission.

Another paper published only as an abstract (Rudolph *et al*, 1998) was a 6-week study with some 460 patients (22% of the meta-analysis subjects) designed to compare speed of response to venlafaxine, fluoxetine and placebo. Can data from such a brief study accurately reflect remission rates at 10 or 12 weeks? Recent work by Quitkin *et al* (2003) suggests otherwise, as a significant number of non-responders to fluoxetine at 6 weeks may show remission at 12 weeks. Thase *et al* themselves acknowledge that differences in times to response between venlafaxine and SSRIs may have contributed to their findings.

In addition, Clerc *et al* (1994) likewise reported a 6-week study, wherein almost twice as many patients taking fluoxetine as those taking venlafaxine (35% *v.* 18%) dropped out of treatment. Finally, in their study of 301 out-patients (approximately 15% of subjects in meta-analysis), Rudolph & Feiger (1999) reported an almost 50% greater drop-out rate in the fluoxetine group compared with the venlafaxine group (29% *v.* 19%).

Thus, although the meta-analysis raises the interesting possibility of differential remission rates, one should bear in mind the limitations of the component studies.

Clerc, G. E., Ruimy, P., Verdeau-Pailles, J., *et al* (1994) A double-blind comparison of venlafaxine and fluoxetine in patients hospitalized for major depression and melancholia. *International Clinical Psychopharmacology*, **9**, 139–143.

Quitkin, F. M., Petkova, E., McGrath, P. J., *et al* (2003) When should a trial of fluoxetine for major depression be declared failed? *American Journal of Psychiatry*, **160**, 734–740.

Rudolph, R. L. & Feiger, A. D. (1999) A double-blind, randomized, placebo-controlled trial of once-daily venlafaxine extended release (XR) and fluoxetine for the treatment of depression. *Journal of Affective Disorders*, **56**, 171–181.

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Thase, M. E., Entsuah, A. R. & Rudolph, R. L. (2001) Remission rates during treatment with venlafaxine or selective serotonin reuptake inhibitors. *British Journal of Psychiatry*, **178**, 234–241.

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Meanings and causes in ADHD

Eric Taylor dismisses Sami Timimi's critique of attention-deficit hyperactivity disorder (ADHD) as an oversimplified polemic (Timimi/Taylor, 2004). He admits he may have been biased because he viewed it as an antipsychiatry tract. I find it unfortunate that the threat of 'antipsychiatry' means that a serious attempt does not appear to have been made to resolve the controversy surrounding ADHD (Double, 2002*a*). Is there a dispute about the facts as well as their interpretation? For example, it is not clear whether brain differences have been shown in unmedicated children, with the protagonists stating opposite views. From the article, it is difficult to see who is correct because Professor Taylor merely quotes the chapter on ADHD from his co-edited textbook (viz. Schachar & Tannock, 2002).

Furthermore, Professor Taylor makes various statements, again with the authority of this textbook chapter, which seem to need further clarification. For example, he says there are known physical counterparts of hyperactivity in brain structure and function, and then does not say what these abnormalities are. If we know what they are, they should be stated and we can then debate their role in aetiology. Similarly, he says that some molecular genetic variations have been robustly replicated, but then does not name the genes, except to say that they especially affect dopamine systems.

There is surely an onus on Professor Taylor to justify his response to Dr Timimi's challenge that the medical model of ADHD 'offers a decontextualised and simplistic idea that leads to all of us – parents, teachers and doctors – disengaging from our social responsibility to raise well-behaved children'. Instead, Taylor proposes increased recognition of the disorder, at least in the UK, 'because there are several good ways of supporting children with severe hyperactivity'. If the central issue is the role of medication in treatment, this is clearly a matter of values (Double, 2002*b*). The recently published collection edited by Fulford *et al* (2003) argues that meanings as well as causes are essential to good psychiatric care. One way of viewing the ADHD controversy is that Dr Timimi is more concerned about the meaning rather than the physical cause of the disorder. Such a position should not be dismissed as antipsychiatry, but acknowledged as a valuable contribution to the debate about the extent to which the use of medication exploits people's emotional problems.

Declaration of interest

D.B.D. is a member of the Critical Psychiatry Network.

Double, D. B. (2002*a*) The history of anti-psychiatry (an essay review). *History of Psychiatry*, **13**, 231–236.

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Fulford, B., Morris, K., Sadler, J., *et al* (2003) *Nature and Narrative. An Introduction to the New Philosophy of Psychiatry*. Oxford: Oxford University Press.

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Timimi, S./Taylor, E. (2004) ADHD is best understood as a cultural construct (debate). *British Journal of Psychiatry*, **184**, 8–9.

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Author's reply: I am grateful to Dr Double for giving me the opportunity to cite more references than are allowed in a debate; but the biological basis of hyperactivity is one of the most researched questions in psychiatry and a letter cannot do justice to it. The chapter I cited previously gives references, and interested readers might also like to consult the recent reviews cited below.

The best-established findings are probably the associations with DNA variations in genes coding for the dopamine receptor (DiMaio *et al*, 2003) and

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dopamine transporter, and neuroimaging findings of altered brain structures including frontal lobe and striatum.

Castellanos *et al* (2002), for example, report the altered neuroanatomy of ADHD, with the brains of those who have never been medicated being more abnormal than those of children who have received stimulants.

Dr Double extends the debate to the question of the use of medication. A large controlled trial (MTA Collaborative Group, 1999) has shown significant advantages of medication over psychological therapy (although I believe that psychological treatment still has an important place). I should therefore like to emphasise that there are dangers in being too reluctant to diagnose and treat ADHD. Children then often receive more destructive labels. Treatment can restore normal function, so it seems to me unacceptable to withhold its benefits from individual children for the sake of a preference for a different form of society.

Declaration of interest

E.T. has an honorary National Health Service contract, and lectures at conferences receiving sponsorship from pharmaceutical companies.

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Commissioning conundrum for custodial care

Simon Wilson presents an editorial (2004) that questions the traditional role of the prison hospital wing. I have also questioned this over the years (Gannon, 2002). However, a factual inaccuracy in his introduction flaws his conclusion.

The Health Secretary for England announced that there would be a transfer of responsibility whereby the NHS *in England* would become responsible for *commissioning* health care in prisons from April 2003. It is very different to announce 'commissioning', as distinct from 'provision' – as Dr Wilson claims. It is, I fear, less of a take-over than a make-over by the Department of Health. Primary care trusts can commission provision from a range of providers – including the current prison provider. The governor will continue to maintain control over the 'cells' in the hospital wing.

Once the reader understands the distinction between commissioning and providing, it provokes thought about the appropriate allocation of health care spending. Why spend the commissioning money twice, on the same citizen, in two different places? Why construct a parallel health care system?

Choosing to highlight capital investment on prisoners may be a public relations disaster. The general public is easily swayed by popular media headlines. Health care spending on special-care baby cots is more palatable than making the prison experience more decent for citizens.

There are hundreds of people in the secure hospitals who have been assessed as no longer requiring that level of security. Capital investment is required urgently at the lower end of the security scale – it is an illusion that more high security is required – thus creating remand beds (not cells) made directly available to courts. This is the only way to seek equivalence. Our mentally ill citizens should not be in prisons at all – we should argue for nothing less.

Eroding this principle, however well intended, just sanitises society's tolerance of this essential injustice. It is all too collusive to believe that we are somehow caring more appropriately if we allow an expansion of common law – lest it just become common lore.

Gannon, S. (2002) A reflective view. In *Prison Nursing* (eds A. E. Norman & A. Parrish), pp. 178–189. Oxford: Blackwell Science.

Wilson, S. (2004) The principle of equivalence and the future of mental health care in prisons. *British Journal of Psychiatry*, **184**, 5–7.

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Author's reply: I am pleased that my editorial has encouraged some discussion about how best to care for the mentally ill in prisons. Mr Gannon is right to point out that it is commissioning rather than providing that has moved to the primary care trusts. The reason for commissioning twice is perhaps to do with geography – people do not necessarily remain in the borough that is responsible for commissioning their health care. Prisoners are not as free to move around as other citizens and one can hardly expect a Leeds general practitioner to attend to her patient in Brixton prison, or vice versa. Otherwise, Mr Gannon and I appear to be in broad agreement – the status quo is unacceptable, and that is why I argued against any expansion of medical treatment under common law (*contra* Mr Gannon's assertion, and *contra* an earlier paper of mine (Wilson & Forrester, 2002)). I advocated an extension of the Mental Health Act 1983 to prisons precisely because that would include openness, accountability and scrutiny in a way that more use of the common law would not. I think that it is the current system that is collusive and dishonest: the championing of equivalence (a noble idea) enables us to feel better about the reality of a failing system of hospital transfers for mentally ill prisoners. I do not, however, share Mr Gannon's optimism that more secure beds (at whatever level of security) are the solution, and it seems to me that history is on my side. At the moment we cannot even make provision within the National Health Service for the most severely mentally ill prisoners, let alone Mr Gannon's suggestion that there should be no mentally ill citizens in prison at all. I wonder whether that includes adjustment disorders, mild depression, treated schizophrenia, substance dependence and personality disorder? Peter Scott, a predecessor of mine at HMP Brixton, suggested that the nature of the walls (prison or hospital) were an irrelevant distraction as the people inside were the same in both types of institution and the treatment needed was broadly similar (Scott, 1970). I have a great deal of sympathy with this view.

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