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.

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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.

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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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