

Correspondence

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Treatment of antisocial personality disorder

Although Professor Maden's article was only 100 words long,¹ it contained some profound and, I think, unfair and unsubstantiated, statements. Where is the evidence that patients with severe antisocial personality disorders who do not want to be treated, like many of those detained in the dangerous and severe personality disorder (DSPD) unit at Broadmoor Hospital, where Professor Maden is the clinical director, can be effectively treated?

Professor Maden criticises lawyers and independent experts but both he and others researching in the field have not produced independently verified evidence of efficacy. Professors Coid² and Duggan,^{3,4} with others, have carried out meta-analyses and concluded that there was no evidence or that the evidence was very weak. Professor Duggan went as far as to suggest that this situation was ultimately unsustainable and would inevitably lead to legal challenges by those detained on the basis of their 'treatability'.

The Canadians and Americans are concerned that psychological therapy with individuals with high scores on the Hare Psychopathy Checklist – Revised is making the situation worse and leading to increases in recidivism.⁵ They appear to have moved on and are investigating biological treatments such as hormone treatment for sex offenders or even addressing the putative causes with gene mapping.⁶

Is it really justifiable to blame lawyers and independent experts for pointing out this lack of evidence and that the DSPD project might not only be an expensive waste of time but it could be making the situation worse?

Declaration of interest

M.P.L. provides independent psychiatric reports to solicitors of patients in DSPD units. He is also a member of the Mental Health Review Tribunal and sometimes sits on DSPD units.

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- 3 Binks CA, Fenton M, McCarthy L, Lee T, Adams CE, Duggan C. Pharmacological interventions for people with borderline personality disorder. *Cochrane Database Syst Rev* 2006; **1**: CD005653.
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- 5 Seto MC, Barbaree HE. Psychopathy, treatment behaviour, and sex offender recidivism. *J Interpers Violence* 1999; **14**: 1235–48.
- 6 Seto MC, Quinsey VL. Toward the future: translating basic research into prevention and strategies. In *Handbook of Psychopathy* (ed CJ Patrick): 589–601. Guilford Press, 2006.

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Author's reply: Article, 100; response, 300+. Is this a metaphor for the medico-legal contribution to psychiatric knowledge, i.e. more words? The 'evidence' for treatability of detained patients is the case law: tribunals don't discharge them and stretch the definition of treatment in so far it makes sense only to lawyers. I don't criticise the players for making a living, but the waste of public money from asking the wrong people the wrong question. Still, the profession could do more by ensuring independent experts in psychiatry (as in other branches of medicine) have clinical or research expertise rather than just an opinion (100).

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Symptoms do not helpfully distinguish unipolar and bipolar depression

Forty *et al's* study¹ revisits a familiar question and reports some statistically significant differences in the frequency of clinical features between unipolar and bipolar depression. The report then moves beyond description to emphasise the clinical importance of these differences. The three symptoms most predictive of bipolar depression were presence of psychosis, diurnal mood variation and hypersomnia. To be considered important, these symptoms when present would need to influence clinical decisions.

The sensitivity of these three symptoms for bipolar depression ranged from 0.3 to 0.59. The specificity ranged from 0.5 to 0.9. The positive predictive value (PPV) ranged from 0.55 to 0.69. As the prevalence of bipolar disorder in the sample was 0.43, these PPV results do not greatly increase the probability of bipolar disorder above the base rate. Likewise, the negative predictive value ranged from 0.63 to 0.69, while the base rate of unipolar depression was 0.57. Again, there is little gain of information.

Because this differential diagnosis relies on pattern recognition rather than on discrete, pathognomonic symptoms, it may be more helpful to examine cases in which all three 'important clinical differences' were present. When all three features are required, beginning with the highest PPV (psychotic features), then the middle PPV (hypersomnia), then the lowest PPV (diurnal variation), and assuming the three symptoms are independent, then 34 bipolar cases and 7 unipolar cases would stand out. From this result, the sensitivity of the triune pattern would be 0.08 and the specificity 0.99. The PPV is 0.83 and the negative predictive value is 0.59. How 'important' is a clinical symptom pattern that detects only 8% of *bona fide* bipolar cases and that does not positively rule in unipolar cases? Moreover, there is no guarantee that latent bipolar depression has the same symptom profile as fully expressed bipolar depression.

All in all, these results underscore the limitations of parsing clinical symptoms for the purpose of classification. The 'important clinical differences' give little added information to clinicians for treatment planning. That is why efforts continue to discover biomarkers or endophenotypes or genetic markers.

1. Forty L, Smith D, Jones L, Jones I, Caesar S, Cooper C, Fraser C, Gordon-Smith K, Hyde S, Farmer A, McGuffin P, Craddock N. Clinical differences between bipolar and unipolar depression. *Br J Psychiatry* 2008; **192**: 388–9.

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