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 putative causes with gene mapping.6

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.


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) would have clinical or research expertise rather than just an opinion (100).

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 0.59 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.


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Authors’ reply: We are in full agreement with Carroll about the limited utility of clinical symptoms for ‘diagnostic tests’ and the consequent importance of efforts to discover biomarkers, endophenotypes or genetic markers. In fact, the main focus of our research is molecular genetic epidemiological investigation of mood disorders and psychoses that has precisely this aim.\(^1\)\(^-\)\(^4\) Further, we have a keen interest in using findings to provide biological validators for psychiatric nosology, classification and clinical diagnosis.\(^5\)

However, for the moment, psychiatrists have to make do with the clinical tools available and be alert to diagnostic clues that can help in the delivery of optimal care to their patients. We stand by the statements in our paper: ‘It is commonly, but wrongly, assumed that there are no important differences in the clinical presentation of unipolar and bipolar depression . . . The clinical features of depression are not, of course, a definitive guide to diagnosis but can help alert the clinician to a possible bipolar course . . . This is important because optimal management varies between bipolar and unipolar depression.’


3 Wellcome Trust Case Control Consortium. Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls. Nature 2007; 447: 661–78.


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