

## Correspondence

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### Psychopathy

Cooke *et al* (2007) claim that there is no compelling empirical evidence to support the conclusion that antisocial behaviour is a central feature of psychopathy. However, in the same issue of the *Journal of Personality Disorders* (2007) report a common genetic component to callous-unemotional traits and antisocial tendencies. Other studies cited by Viding *et al* report similar results. Moreover, Larsson *et al* (2007) reported that the same general four factors present in our four-factor model of psychopathy (Vitacco *et al*, 2005) all loaded onto a single genetic factor. Longitudinal research (not cited by Cooke *et al*) indicates that antisocial tendencies are significantly linked to the longitudinal stability of psychopathic traits (Frick *et al*, 2003). Cooke *et al* refer to the work of Cleckley (1988) to support their position, but in Cleckley's accounts of psychopathy antisocial behaviours play an important role. As Patrick (2006: p. 608) noted, 'There is no question that Cleckley considered persistent antisocial deviance to be characteristic of psychopaths. Without exception, all the individuals represented in his case histories engage in repeated violations of the law – including truancy, vandalism, theft, fraud, forgery, fire-setting, drunkenness and disorderly conduct, assault, reckless driving, drug offences, prostitution, and escape.' As Blackburn (2007: p. 145) recently put it, 'Contra Cooke, . . . antisocial behavior, conceived broadly, is a characteristic feature of psychopathy.'

In our paper based on a very large sample (Vitacco *et al*, 2005), we demonstrated the conceptual errors and flaws in modelling that went into the development of Cooke's model and provided evidence for the four-factor model. Interestingly, Cooke *et al* did not cite this large study but rather chose to cite our small preliminary studies, although they are in line with our larger study. We do not view criminality

as central to psychopathy. Indeed, the Psychopathy Checklist – Screening Version (PCL–SV) contains two items that refer to antisocial behaviour and that can be scored without evidence of criminality. The PCL–R and PCL–SV are virtually identical psychometrically, as noted previously by Cooke *et al* (1999).

**Blackburn, R. (2007)** Personality disorder and antisocial deviance: comments on the debate on the structure of the Psychopathy Checklist – Revised. *Journal of Personality Disorders*, **21**, 142–159.

**Cleckley, H. (1988)** *The Mask of Sanity* (5th edn). Mosby.

**Cooke, D. J., Michie, C., Hart, S., et al (1999)** Evaluating the screening version of the Hare Psychopathy Checklist (PCL: SV): an item response theory analysis. *Psychological Assessment*, **11**, 3–13.

**Cooke, D. J., Michie, C. & Skeem, J. (2007)** Understanding the structure of the Psychopathy Checklist – Revised. An exploration of methodological confusion. *British Journal of Psychiatry*, **190** (suppl. 49), s39–s50.

**Frick, P. J., Kimonis, E. R., Dandreaux, D. M., et al (2003)** The 4 years stability of psychopathic traits in non-referred youth. *Behavioral Sciences and the Law*, **21**, 1–24.

**Larsson, H., Tuvblad, C., Rijdsdijk, F. V., et al (2007)** A common genetic factor explains the association between psychopathic personality and antisocial behavior. *Psychological Medicine*, **37**, 15–26.

**Patrick, C. J. (2006)** Back to the future: Cleckley as a guide to the next generation of psychopathy research. In *Handbook of Psychopathy* (ed. C. J. Patrick), pp. 605–618. Guilford.

**Viding, E., Frick, P. J. & Plomin, R. (2007)** Aetiology of the relationship between callous–unemotional traits and conduct problems in childhood. *British Journal of Psychiatry*, **190** (suppl. 49), 33–38.

**Vitacco, M., Neumann, C. S. & Jackson, R. L. (2005)** Testing of a four-factor model of psychopathy: associations with gender, ethnicity, intelligence and violence. *Journal of Consulting and Clinical Psychology*, **73**, 466–476.

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The article by Cooke *et al* (2007) contains a number of fundamental modelling errors.

First, the authors continue to present an over-factored model (i.e. hierarchical three-factor model with testlets), which results in negative variances. This 13-item model actually contains 10 factors: 6 first-order factors/testlets, 3 second-order factors and 1 third-order factor (simply count the number of circles/factors in Fig. 1). Any model can achieve good fit when it is as complex as the data it attempts to summarise. We have shown that this testlet model results in untenable parameters in four separate studies (Neumann *et al*, 2006). One author of the Cooke *et al* paper has also suggested that the testlet model is over-factored (Skeem *et al*, 2003). Cooke does not acknowledge this problem of an over-factored model, even though it is evident in his published work (see Cooke & Michie, 2001, Figs 2 and 3, which contain zero variance terms that the EQS program sets to zero when estimating negative variances). Cooke *et al* (2007) mention that we have criticised their use of testlets but they do not dispute that it creates a misspecified model with untenable parameters. Our analysis of the testlet model is available upon request.

Cooke *et al* provided a polychoric correlation matrix, ostensibly to give investigators the opportunity to replicate their findings. However, as noted in the EQS program manual, robust procedures can only be conducted with the raw items. Thus, the results reported by Cooke *et al* appear to be transparent but in reality no one will be able to unambiguously verify their analyses. When one analyses their published correlation matrix using a non-robust procedure, very different findings result. Also, Cooke *et al* relied upon a maximum likelihood procedure for estimating model parameters, despite the fact that it is well known that this procedure underestimates model parameters and model fit when used with ordinal data (Everitt & Dunn, 2001) such as the items of the Psychopathy Checklist – Revised. There was no serious discussion on why robust maximum likelihood with polychoric correlations was employed, except that it is recommended in the manual for EQS version 6. None the less, the verisimilitude of this new approach is currently unknown. A program such as Mplus, which employs a robust weighted least-squares procedure for ordinal data is an accepted approach (Neumann *et al*, 2006). Cooke *et al*'s use of Mplus was limited. Our Mplus analyses of the UK data along with our previously

published findings can be found online (<http://bjp.rcpsych.org/cgi/eletters/190/49/s39>).

Contrary to Cooke *et al*, the four-factor model clearly fits as well or better than a viable three-factor model. Moreover, our recent research indicates that the four first-order factors are explained by a cohesive superordinate factor (Neumann *et al*, 2006, 2007).

**Cooke, D. J. & Michie, C. (2001)** Refining the construct of psychopathy: towards a hierarchical model. *Psychological Assessment*, **13**, 171–188.

**Cooke, D. J., Michie, C. & Skeem, J. (2007)** Understanding the structure of the Psychopathy Checklist – Revised. An exploration of methodological confusion. *British Journal of Psychiatry*, **190** (suppl. 49), s39–s50.

**Everitt, B. & Dunn, G. (2001)** *Applied Multivariate Data Analysis*. Oxford University Press.

**Neumann, C. S., Kosson, D. S., Forth, A. E., et al (2006)** Factor structure of the Hare Psychopathy Checklist: Youth Version (PCL:YV) in incarcerated adolescents. *Psychological Assessment*, **18**, 142–154.

**Neumann, C. S., Hare, R. D. & Newman, J. P. (2007)** The super-ordinate nature of the Psychopathy Checklist-Revised. *Journal of Personality Disorders*, **21**, 102–117.

**Skeem, J. L., Mulvey, E. P. & Grisso, T. (2003)** Applicability of traditional and revised models of psychopathy to the Psychopathy Checklist-Revised. *Psychological Assessment*, **15**, 4–55.

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### Involuntary community treatment

Swanson *et al* (2000) reanalysed the results of the North Carolina trial (Swartz *et al*, 1999) and their findings are becoming increasingly influential in current debates about mental health legislation in the UK. Our recent systematic review (Churchill *et al*, 2007), which included these articles, demonstrated that there was no robust evidence to indicate that community treatment orders are associated with either significant benefit or harm. The secondary analyses performed by Swanson *et al* are, we believe, misleading for two reasons.

First, based on everyone in the trial the intention-to-treat (ITT) effect of randomisation to an involuntary out-patient commitment (OPC) was of a modest and non-significant reduction in violence (risk difference of 4.5%). This overall ITT effect of OPCs is a weighted average of the ITT effects in the two subgroups of participants

defined by their post-randomisation management (those who received short-term OPCs and those who eventually received long-term OPCs). These two subgroups would exist in the control arm had they been placed on OPCs. Assuming that there was no benefit in those who received the short-term OPCs (i.e. risk difference 0), the results of Swanson *et al* suggest that the reduction in violence in those with long-term OPCs would be 12.4%. However, even if considered clinically significant, this finding would still not be statistically significant because the overall ITT effect was not significant (assuming a zero ITT effect in those receiving short-term OPCs implies that a test of the hypothesis concerning those receiving long-term OPCs is equivalent to the test for the overall ITT effect). The only way in which there could have been a beneficial effect in those receiving long-term OPCs is if the effects in those receiving short-term OPCs were actually detrimental (i.e. increased the rate of violence). It is improbable that they would be, and in policy terms it would be unacceptable to impose OPCs in the knowledge that they would cause harm to those in whom they are only applied for a short period.

Second, a *post hoc* comparison of the outcomes in groups defined by management decisions or patient behaviour following randomisation is potentially subject to selection effects (hidden confounding). That this is in fact the case is illustrated by the results of other subgroup analyses by the same research group (Swartz *et al*, 1999: Fig. 1). The group destined to be on long-term OPC have a better clinical outcome in the first 1–2 months. In other words there is evidence that the group destined to receive long-term OPCs have a favourable clinical profile before the OPC is renewed. We believe that it is likely that long-term OPCs will only be contemplated under certain circumstances, such as when the short-term OPC has apparently made a difference. Those who have intractable problems or in whom a short-term OPC has failed to make any change might not have their OPC renewed.

The investigators responsible for the North Carolina trial accomplished one of the most extraordinary trials ever performed and as such deserve enormous praise. However, the results described in these and similar secondary analyses are, we believe, flawed and misleading, and should not be taken as evidence for a beneficial effect of OPC. We made a similar

point (Szmukler & Hotopf, 2001) following the publication of the original trial. The trial data are best interpreted using the main ITT analyses, which show no evidence of benefit or harm.

**Churchill, R., Owen, G., Singh, S., et al (2007)** *International Experiences of Using Community Treatment Orders*. Department of Health.

**Swanson, J. W., Swartz, M. S., Wagner, H. R., et al (2000)** Involuntary out-patient commitment and reduction of violent behaviour in persons with severe mental illness. *British Journal of Psychiatry*, **176**, 324–331.

**Swartz, M. S., Swanson, J. W., Wagner, H. R., et al (1999)** Can involuntary outpatient commitment reduce hospital recidivism? Findings from a randomized trial with severely mentally ill individuals. *American Journal of Psychiatry*, **156**, 1968–1975.

**Szmukler, G. & Hotopf, M. (2001)** Effectiveness of involuntary outpatient commitment. *American Journal of Psychiatry*, **158**, 653–654.

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**Authors' reply:** Hotopf *et al* make essentially the same point that we stated in the article ‘. . . the study found no significant difference in the prospective rate of violence between the two randomly assigned groups: 32.3% in the OPC group *v.* 36.8% in the control group (Fisher's exact test, one-tailed:  $P=0.292$ ; two-tailed:  $P=0.567$ )’ (Swanson *et al*, 2000).

Critics of OPC policy might wish we had left it at that, but straightforward analysis of randomised controlled trials does not tell the whole story. In this case it excluded people with a documented history of serious violence ( $n=64$ ), since the court did not permit us to randomise these to the control group. However, variability in the real-world application of OPC allowed us to examine whether longer periods of court-ordered treatment were associated with lower rates of violence over the study year. They were.

Hotopf *et al* are rightly concerned about the possibility of favourable selection bias, but we think this is an unlikely explanation for our findings. Indeed, people with a history of treatment non-adherence were more than twice as likely to receive an extended period of OPC (40.0 *v.* 18.75%). If anything, this should have stacked the deck against finding an effect for long-term OPC.