Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

Contents
- Predicting violent offences by released prisoners
- Effectiveness of methadone treatment for heroin addiction
- Psychosis as a failure of reality testing
- Psychotherapy for severe somatoform disorder: problems with missing studies

Predicting violent offences by released prisoners

For a pejorative term without proven clinical utility, psychopathy has generated some very catchy sayings. Some bear little relationship to the research that generated them. ‘Treatment makes psychopaths worse’ is one (see Rice et al). I fear that without urgent corrective action, ‘Risk assessment doesn’t work for psychopaths’ (see Coid et al) will be another.

Coid et al compared the ability of three structured risk assessment instruments – the Violence Risk Assessment Guide (VRAG), the Historical, Clinical, Risk Management-20 (HCR-20) and the Offender Group Reconviction Scale-II (OGRS-II) – to predict violent offences by released prisoners in different diagnostic groups. They defined one such group, ‘psychopathic personality’, using a score of over 30 on the Psychopathy Checklist-Revised (PCL-R). For most instruments and groups, Coid et al found moderate levels of predictive accuracy. For the 5.7% of the sample scoring over 30 on the PCL-R, however, no risk assessment instruments performed better than flipping a coin. The authors see major implications for risk assessment. They state that new actuarial tools may be required.

A better conclusion would be that if you define a group using a high score on one instrument that predicts violence, other such instruments will struggle to predict violence in that group. Originally designed to measure a psychological construct, psychopathy, the PCL-R has proved to be one of several instruments that consistently predict violence better than chance (area under the curve (AUC) 0.65–0.75; see Singh et al). The VRAG and the HCR-20 are others. The other instruments could only have successfully predicted violence among Coid et al’s ‘psychopathic personalities’ if structured risk assessment instruments could be applied serially with increasing success.

We know that they cannot. When Seto combined the results of using instruments sequentially to predict serious offending, also in ex-prisoners, he did no better than he had using one instrument alone. These data, and others suggesting the particular items on a scale are less important than the constructs, such as past behaviour and substance use, that the items represent, have led some to suspect that a ceiling effect may apply to the prediction of violence in psychiatric and other populations. Efforts to improve the accuracy of structured risk assessment instruments are probably better directed at reducing the quantity of missing data than at adding new instruments.

I have a wager for Coid et al: try the process in reverse. Select the 5.7% of the sample with the highest HCR or VRAG scores and test whether the PCL-R is predictive in these groups. My five pounds says it will not be, and for the same reason. More is not necessarily better. Or, once you have squeezed the fruit, there usually isn’t much point squeezing it again.


Authors’ reply: In the introduction of his letter, Buchanan refers to psychopathy as a ‘pejorative term’ but later categorises it as a risk assessment instrument. It is neither. Psychopathy as a psychotic syndrome was first described by a general psychiatrist and further developed into a diagnostic construct operationalised with the PCL-R. It is retained within dissociative personality disorder in ICD-10 and as an alternative model of antisocial personality disorder in DSM-5. The PCL-R is recognised internationally as the gold standard for assessment of psychopathy. Proficiency in its use should be a core competency for clinicians who work with offenders. Sadly, many are not adequately trained and struggle to comprehend why their treatments usually fail with these individuals and sometimes make their behaviour worse.

Buchanan may have misunderstood Seto’s method. The instruments were applied simultaneously, not sequentially. However, he is right that sequential screening does not improve accuracy. We would suggest a better reference for an explanation. We would also emphasise that risk assessment instruments are no more than screening instruments. Most importantly, there is currently no evidence base to demonstrate that routine clinical use of these screens can prevent violence, despite mandatory use in some UK services.

With regard to the ‘glass ceiling’ effect that we have previously investigated, reducing missing data will achieve little to break through this. Trigger factors precede many violent events. They may occur in the context of static and dynamic risk factors which have predictive efficacy. But trigger factors are causal, can occur within seconds to trigger violence and, most importantly, are not predictable.

Finally: the wager. There is no purpose in doing this if psychopathy is a personality construct. Furthermore, we have previously shown that few PCL-R items are predictive. But we did rise to the challenge of Buchanan and tested the predictive accuracy of the VRAG, OGRS and HCR-20 in high-risk groups defined by these instruments. Using 32 as the HCR-20 cut-off and 27 for VRAG to be as close as possible to Buchanan’s 5.7%, we estimated AUCs for VRAG and OGRS in the same HCR-20 high-risk group, and AUCs for HCR-20 and OGRS in the corresponding VRAG high-risk group. In the VRAG high-risk group, the HCR-20 showed a low AUC of 0.44 (95% CI 0.37–0.41), whereas the VRAG had an AUC of 0.63 (95% CI 0.57–0.69). We do not think this is evidence that the VRAG is no better than chance.
0.28–0.61). The OGRS was a more respectable 0.69 (95% CI 0.53–0.85). In the HCR-20 high-risk group, AUC for VRAG was 0.67 (95% CI 0.54–0.81) and OGRS 0.68 (95% CI 0.64–0.81).

Perhaps there would be mileage in squeezing the fruit again in Buchanan’s next study?


Effectiveness of methadone treatment for heroin addiction

Regarding Byford et al’s paper,¹ the authors present an analysis of the results of the Randomised Injectable Opiate Treatment Trial (RIOTT).² Participants of RIOTT were very few in number – fewer than 45 individuals in each of the three arms of the study (injectable heroin, injectable methadone and ‘optimised’ oral methadone). It required 3 full years at 3 sites to screen 301 volunteers, of whom 127 (40%) began the trial and only 89 completed the 26-week treatment protocol.

All of the participants had been receiving ‘conventional’ methadone treatment for more than 6 months and continued ‘to inject ’street’ heroin regularly’. On average, they had had over four prior treatment episodes. Accordingly, it is reasonable to assume that the overriding motivation of those who volunteered was the hope of receiving injectable opiates, and it is likely that participant bias may have had a substantial impact on outcomes. Indeed, it is revealing that among those assigned to receive optimised oral methadone, 7 (17%) never began the trial and of the remaining 35 only 24 were still enrolled 26 weeks later.

Some of the reported findings seem to underscore the severe limitations that must be kept in mind in drawing even the most tentative conclusions. For example, although the oral methadone group claimed to have committed roughly three times as many crimes as the intravenous methadone group (mean 21 v. 7 crimes), the latter group spent 15 times more nights in prison (mean 6.1 v. 0.4). Surely provision of oral methadone did not somehow make patients more successful in their criminal pursuits.

Perhaps inevitably, the limited ability to extrapolate has been ignored in the wider distribution of the findings. Thus, one report (which refers readers seeking more information to the Press Officer of King’s College London, with which the principal author and five of the seven co-authors are affiliated) had the unqualified headline: ‘Injectable opioid treatment for chronic heroin addiction more cost-effective than oral methadone’, and claimed that ‘total cost savings of providing injectable opiate treatment for this chronic group in England could be between £29 and £59 million per year.’³

The criticisms noted above must not detract from the bottom-line, common sense, conclusion with regard to injectable opioid treatment: in the interests of addicts as well as the general community, it is essential that those who respond poorly to treatment (any treatment) be provided information on and referral to the broadest possible array of alternative services.


Authors’ reply: Newman rightly draws attention to the effectiveness of appropriately delivered methadone treatment for many people with heroin addiction worldwide over the past half-century. Our economic evaluation¹ and the preceding report on the main findings from the RIOTT trial² should not be considered an attack on the value of oral methadone to the majority who show substantial benefit from this treatment.³,⁴ Rather the RIOTT trial needs to be recognised for what it was – an investigation of effectiveness and cost-effectiveness of alternative treatments in a subgroup of the treatment population with severe and chronic addiction who were not responding to oral methadone maintenance treatment.

It is also appropriate to inject a note of caution about the potential influence of expectations on trial participants. This limitation is inherent in any trial where the patient has a preference for which treatment arm they may be assigned to, and Newman is right that this has the potential to be a pronounced influence in the addiction treatment field. In fact, aware of this potential, we gathered some data from patients on their expectations and experiences of treatment within the trial, and this has recently been reported separately.⁵

Newman notes the modest sample size in this trial (total of 127 participants). This is a particular challenge in a field where treatment is intensive and expensive, and in countries which do not have a tradition of funding large treatment trials in the addictions field. We would nevertheless point out that the sample size was calculated in advance by the applicants for the original research award and was judged to be adequate to detect the expected effect size as defined in the protocol.²

Newman highlights a further limitation of sample size in this highly variable population, using the example of criminal activity. Although the oral methadone group reported committing a much higher number of crimes than the injectable methadone group, the latter group spent more nights in prison. However, the total number of participants spending any time in prison (n = 6; 5%) is extremely small relative to the number reporting any criminal activity (n = 50; 42%), so it would be inappropriate to try and come to any comparative conclusions.
Correspondence

In conclusion, we acknowledge the limitations of research in this complex subgroup with chronic heroin addiction and also the evidence of benefit from oral methadone in the broader population of people addicted to the drug. However, we consider the important findings reported in the paper are that, for this subgroup doing persistently badly on oral methadone treatment, it is important for clinicians to work with their patients to explore alternative options, such as injectable treatments, which may achieve health benefits not being achieved in the expected manner with the orthodox first-line treatment, and which may achieve this health benefit in a more cost-effective manner. Such personalisation of treatment plans is important but is currently being hindered by the cost implications of providing injectable alternatives and a previous lack of evidence of cost-effectiveness.


Sarah Byford, Centre for the Economics of Mental and Physical Health, Box P024, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK. Email: s.byford@kcl.ac.uk; Barbara Barrett, Centre for the Economics of Mental and Physical Health, Nicola Metrebian, Addictions Department, National Addiction Centre, Institute of Psychiatry, King’s College London, UK; Teodora Groshkova, European Monitoring Centre for Drugs and Drug Addiction, Lisbon, Portugal; Maria Cary, Centre for the Economics of Mental and Physical Health, Vikki Charles, Addictions Department, National Addiction Centre, Institute of Psychiatry, King’s College London, UK; Nicholas Linzeris, The Langton Centre, South Eastern Sydney Local Health District, NSW Health, Australia; John Strang, Addictions Department, National Addiction Centre, Institute of Psychiatry, King’s College London, UK.

doi: 10.1192/bjp.204.3.242

Psychosis as a failure of reality testing

Garety & Freeman present a timely review on the nature of delusional experience.1 Their conclusion regarding the need to focus on individual features of psychosis seems apt. The presented overview of cognitive and affective mechanisms influencing delusion development seems, however, to overlook an essential component of delusional experience; that psychotic symptoms, including delusions, at their heart represent a failure of reality testing.

The description of jumping to conclusions, together with the probabilistic reasoning task methodology, appear to rely on a logical chain of thought progression and conclusion – what Campbell has referred to as an empiricist understanding.2 This approach, however, does not take into account the nature of conclusions reached in delusional belief. Conclusions reached on seeing two, or fewer, coloured counters seem quite distinct from classic examples of delusional perception: ‘I saw the traffic lights turn green and realised that the world would end’.3 Campbell’s classic examples of delusional perception: ‘I saw the traffic lights turn green and realised that the world would end’.3 Campbell’s classic examples of delusional perception:

We do not agree that world beliefs are fundamentally rearranged in people with delusions. Rather, the person’s delusions can be shown to build on the pre-existing thoughts about self and world, and are actually typically preceded by periods of anxious worry.4 Traditional views of sudden dramatic changes are not in general supported by the evidence. Although we show that there is clear evidence of the importance of emotional processes – and in some cases this can be linked to childhood trauma – we do not conclude that the delusion represents a defence. The psychoanalytic defence accounts are not supported by the evidence. Rather, anxiety and depression – and negative views of self and others – are risk factors for and commonly expressed by patients with delusions.5 We consider that these research findings render delusions explicable, and may have implications for the way all clinicians engage with people with delusions.

We advocate that there is now enough certainty in the evidence base for concerted efforts to translate them into targeted treatments for delusions. It is through further trials, drawing on the evidence base which identifies mechanisms underpinning

Authors’ reply: We differ in our approach from that taken by Shepherd, in that we are advocating an empirical approach which posits hypotheses that can be and are tested. Our review of over 200 studies demonstrates how much has been learned by testing hypotheses, amassing evidence and replicating findings.1 Thus there is now strong and consistent evidence that delusions are associated with biases in reasoning, such as are assessed by experimental tasks and reliable interviews. These findings are important and provide an explanation of the failure to take on board all the evidence – or a failure of reality testing, as Shepherd puts it. We now therefore have secure knowledge of specific reasoning processes which may be targeted in treatment.2

We do not agree that world beliefs are fundamentally rearranged in people with delusions. Rather, the person’s delusions can be shown to build on the pre-existing thoughts about self and world, and are actually typically preceded by periods of anxious worry.4 Traditional views of sudden dramatic changes are not in general supported by the evidence. Although we show that there is clear evidence of the importance of emotional processes – and in some cases this can be linked to childhood trauma – we do not conclude that the delusion represents a defence. The psychoanalytic defence accounts are not supported by the evidence. Rather, anxiety and depression – and negative views of self and others – are risk factors for and commonly expressed by patients with delusions.5 We consider that these research findings render delusions explicable, and may have implications for the way all clinicians engage with people with delusions.

We advocate that there is now enough certainty in the evidence base for concerted efforts to translate them into targeted treatments for delusions. It is through further trials, drawing on the evidence base which identifies mechanisms underpinning

3 Bayne T, Pacherie E. Bottom-up or top-down: Campbell’s rationalist account of monothematic delusions. Philos Psychiatr Psychol 2004; 11: 1–11.

Andrew Shepherd, Doctor, University of Manchester, UK. Email: andrew.shepherd3@nhs.net

doi: 10.1192/bjp.204.3.242

doi: 10.1192/bjp.204.3.241a

Sarah Byford, Centre for the Economics of Mental and Physical Health, Box P024, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK. Email: s.byford@kcl.ac.uk; Barbara Barrett, Centre for the Economics of Mental and Physical Health, Nicola Metrebian, Addictions Department, National Addiction Centre, Institute of Psychiatry, King’s College London, UK; Teodora Groshkova, European Monitoring Centre for Drugs and Drug Addiction, Lisbon, Portugal; Maria Cary, Centre for the Economics of Mental and Physical Health, Vikki Charles, Addictions Department, National Addiction Centre, Institute of Psychiatry, King’s College London, UK; Nicholas Linzeris, The Langton Centre, South Eastern Sydney Local Health District, NSW Health, Australia; John Strang, Addictions Department, National Addiction Centre, Institute of Psychiatry, King’s College London, UK.

Psychosis as a failure of reality testing

Garety & Freeman present a timely review on the nature of delusional experience.1 Their conclusion regarding the need to focus on individual features of psychosis seems apt. The presented overview of cognitive and affective mechanisms influencing delusion development seems, however, to overlook an essential component of delusional experience; that psychotic symptoms, including delusions, at their heart represent a failure of reality testing.

The description of jumping to conclusions, together with the probabilistic reasoning task methodology, appear to rely on a logical chain of thought progression and conclusion – what Campbell has referred to as an empiricist understanding.2 This approach, however, does not take into account the nature of conclusions reached in delusional belief. Conclusions reached on seeing two, or fewer, coloured counters seem quite distinct from classic examples of delusional perception: ‘I saw the traffic lights turn green and realised that the world would end’.3 Campbell’s classic examples of delusional perception:

We do not agree that world beliefs are fundamentally rearranged in people with delusions. Rather, the person’s delusions can be shown to build on the pre-existing thoughts about self and world, and are actually typically preceded by periods of anxious worry.4 Traditional views of sudden dramatic changes are not in general supported by the evidence. Although we show that there is clear evidence of the importance of emotional processes – and in some cases this can be linked to childhood trauma – we do not conclude that the delusion represents a defence. The psychoanalytic defence accounts are not supported by the evidence. Rather, anxiety and depression – and negative views of self and others – are risk factors for and commonly expressed by patients with delusions.5 We consider that these research findings render delusions explicable, and may have implications for the way all clinicians engage with people with delusions.

We advocate that there is now enough certainty in the evidence base for concerted efforts to translate them into targeted treatments for delusions. It is through further trials, drawing on the evidence base which identifies mechanisms underpinning
delusions, and with change in delusions as the primary outcome, that we will make progress towards alleviating the distress at the heart of delusional experience.


P. A. Garety
Max PO77, Institute of Psychiatry, King’s College London, De Crespigny Park, London SE5 BAF, UK. Email: philippa.garety@kcl.ac.uk; D. Freeman
Department of Psychiatry, University of Oxford, Warneford Hospital, Oxford, UK
doi: 10.1192/bjp.204.3.242a

Psychotherapy for severe somatoform disorder: problems with missing studies

The recent review by Koelen and colleagues of psychotherapy for severe somatoform disorder is welcome in highlighting the need for better evidence in this area. It has unfortunately omitted a number of relevant studies, especially relating to conversion disorder. One major reason for this is that the index date on which studies were searched for, March 2010, was nearly 4 years prior to publication. It is a pity that the authors did not update their analysis at the time of their last revision in June 2013, as they would, at this time, have been able to include a number of relevant studies, including a randomised trial of cognitive-behavioural therapy for non-epileptic seizures (n = 66) and a randomised controlled trial of guided self-help for functional neurological symptoms (i.e. conversion disorder) (n = 127). These two studies were published before one of the studies included in the analysis, the study by Sattel et al published in 2012.

There are further studies of psychotherapy in conversion disorder which were published before March 2010: a study of psychotherapy for non-epileptic seizures (n = 20); a study of psychotherapy for conversion disorder (n = 91); a study of psychotherapy for psychogenic movement disorders (n = 10); and a large controlled and negative trial of psychotherapy for patients with somatofor disorder in a general hospital (n = 91). The authors may have excluded them but they did not present a list of the 64 excluded studies as a supplemental file.

Other types of study that could arguably have been included using the authors’ own criteria are some randomised trials in functional dysphonia, a form of conversion disorder treated in secondary care with voice therapy and sometimes psychotherapy. There are also treatment studies of children with conversion disorder which have not been included and would not have been excluded by the authors’ inclusion criteria.

Further studies in conversion disorder have followed in the past 2 years which describe outcomes from multidisciplinary treatment including psychotherapy. Journal articles cannot always be up to date, but the number of omissions here make this meta-analysis immediately in need of updating.

Two included studies were of hypnosis for motor conversion disorder. Hypnosis is arguably a form of psychotherapy, but also arguably not. In addition, the inclusion of studies which randomised bioenergetic exercise against gym exercise in a setting where all patients received psychotherapy and a study of in-patient multidisciplinary rehabilitation in chronic pain (n = 298) graded as ‘extremely poor’ and then included in a ‘treatment as usual arm’ is debatable.

The authors could have done more to highlight one of the obvious drawbacks of their review. There is a paradox in reporting on treatment for patients who had been defined as having somatoform disorder (often needing only to have three symptoms e.g. pain, fatigue, dizziness or irritable bowel syndrome) while ignoring studies on psychotherapy for individual functional somatic disorders such as irritable bowel syndrome and fibromyalgia. Most patients with functional somatoform disorder also have other symptoms such as fatigue and pain, and probably would, for example, meet criteria for multisomatoform disorder.

It is at times highly arbitrary whether authors decide, for example, to use the term somatoform pain disorder or chronic pain disorder. A broader overview of studies in all these fields or at least greater acknowledgement of the overlap would have been helpful for the reader.


Authors’ reply: Stone commented that more studies should have been included in our meta-analysis. In particular, he suggests that six studies (references 2, 3, 12–15 cited by Stone) were published after the index date of our literature search (March 2010) and four other studies that were published before 2010 (references 5–8 cited by Stone) could have been included, and that one review that contains randomised trials in functional dysphonia might have met our inclusion criteria (reference 9 cited by Stone). Stone’s concern is that these omissions make our meta-analysis out of date.

Apparently, the rationale behind our inclusion criteria requires further clarification. Most importantly, while previous reviews were restricted to psychodynamic psychotherapy only, or included medically unexplained physical symptoms according to divergent criteria, our meta-analysis examined the effectiveness of psychotherapy for severe somatoform disorder. As mentioned in our publication, ‘severe’ was defined as a diagnosis of somatoform disorder according to established criteria and treatment offered in secondary or tertiary care settings.

We chose to utilise established criteria for somatoform disorder from the psychiatric nomenclature (ICD and DSM in particular). Our main rationale was that psychiatric diagnoses contain explicit criteria about impaired daily functioning in main areas of life (social, interpersonal and occupational), and about psychological factors implicated in the disorder. We opted for these criteria because in our view these would best capture the impairments and the complicated aetiology of these disorders. For this reason, we disregarded medical diagnoses that do not always consider psychological factors implicated in the disorder, and that often use less stringent criteria regarding the duration and severity of the disorder.

The search terms we used simply would not have yielded most of the studies mentioned by Stone, because for the reasons outlined above we did not search for dissociative seizures, pseudo-seizures, psychogenic non-epileptic seizures and psychogenic movement disorders. One study was excluded because Escobar et al’s less stringent criteria for somatisation were used (reference 8 cited by Stone). Stone also mentions a review of randomised trials for functional dysphonia (reference 9 cited by Stone), but for the same reasons, these studies do not meet our inclusion criteria. Two studies mentioned in Stone’s letter meet our inclusion criteria, one of which should probably have been included after revision in June 2013 (reference 12 cited by Stone), while the other was published in October 2013, and could not have been included (reference 13 cited by Stone).

We agree with Stone that there is a paradox in including somatoform disorder while excluding individual somatic syndromes, especially given the arbitrary cut-offs and the high overlap between seemingly distinct somatic syndromes. This is at least in part a reflection of the problematic nomenclature for these disorders, which is divided between psychiatry and the remainder of medicine. We concur with Stone that most patients with functional somatic disorders also have other symptoms, and may even meet criteria for somatoform disorder. Yet, we cannot be sure that all patients with individual somatic syndromes do meet criteria for somatoform disorder. For this reason, and for the reasons outlined above, we did not include individual somatic syndromes. At the same time, other reviews have already summarised the effectiveness of psychotherapy for medically unexplained physical symptoms using less stringent criteria, also including some – although not all – conversion disorders.

To conclude, we consider it a strength of the current meta-analysis that it has a narrow and therefore specific focus on a precise diagnostic entity, because this clearly defines the boundaries for generalisation of the findings. We acknowledge that our findings cannot be extrapolated to all fields of medicine and somatic symptom disorders. The results from our meta-analysis specifically apply to patients with somatoform disorder according to established (psychiatric) diagnostic criteria that received psychotherapy in secondary and tertiary care.

Cost-effectiveness of cognitive–behavioural therapy as an adjunct to pharmacotherapy for treatment-resistant depression in primary care: economic evaluation of the CoBalT Trial. *BJP*, 204, 69–76. The funding declaration (p.75) should read: This research was funded by the National Institute for Health Research Health Technology Assessment (NIHR HTA) programme (project number: 06/404/02). The views expressed in this publication are those of the authors and do not necessarily reflect those of the HTA programme, NIHR, NHS, or the Department of Health.

Corpus callosum changes in euthymic bipolar affective disorder. *BJP*, 204, 129–136. In Fig. 2 (p.133) part (a) was incorrectly shown. The compete figure is correctly reproduced below. The online article has been corrected post-publication in deviation from print and in accordance with this correction.

doi: 10.1192/bjp.204.3.245
Effectiveness of methadone treatment for heroin addiction
Robert Newman
BJP 2014, 204:241.
Access the most recent version at DOI: 10.1192/bjp.204.3.241

References
This article cites 2 articles, 1 of which you can access for free at:
http://bjp.rcpsych.org/content/204/3/241.1#BIBL

Reprints/permissions
To obtain reprints or permission to reproduce material from this paper, please write to permissions@rcpsych.ac.uk

You can respond to this article at
/letters/submit/bjprcpsych;204/3/241

Downloaded from
http://bjp.rcpsych.org/ on June 27, 2017
Published by The Royal College of Psychiatrists

To subscribe to The British Journal of Psychiatry go to:
http://bjp.rcpsych.org/site/subscriptions/