Correspondence

EDITED BY KIRIAKOS XENITIDIS and COLIN CAMPBELL

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Devaluation of PTSD

The item from the Editor’s desk on the devaluation of post-traumatic stress disorder (PTSD) (Tyrer, 2005) is to be welcomed, and in my view this issue is indeed ‘highly significant’. Post-traumatic stress disorder is surely not a true medical diagnosis, rather it is best seen as a medico-legal, benefit-linked criterion. It is a bureaucratic hurdle for a claimant to surmount, not a medical diagnosis with implications for treatment and cure. Hence its ‘interference in care’ for the clinician who unwittingly misuses it.

The vague and emotive idea of ‘post-Vietnam syndrome’ was explicitly introduced in the USA to ensure that returning Vietnam veterans, with various and often non-specific mental reaction symptoms which may or may not have followed experience of ordeal, were able to have a quasi-diagnostic ‘label’ attached to them, and thus receive care under the Veterans’ Administration Medical Service, rather than being bereft of help. A core of largely anti-war psychiatrists and veterans worked for years to create the PTSD concept, and put it in DSM-III in 1980 (Scott, 1993).

The PTSD category may include probably virtually anyone suffering an unpleasant experience of which they have disagreeable memories. It simply lacks precision in distinguishing between claimed subjective distress and objective disorder. The core question is often whether the claimed sufferer is impaired in their capacity to function. If they are, conventional symptoms of mental reaction following trauma, such as anxiety, depression, phobia and addiction, in their various manifestations are overwhelmingly more likely to be the cause rather than any putative PTSD derivative.

Post-traumatic stress disorder is in my view virtually useless as a medical diagnosis. Its use does more harm than good, it carries no useful treatment implications, it is liable to lead to needless chronicity and worry, and it is irredeemably contaminated by litigation. Those whose clinical practice leads them to such conclusions should recognise that ICD is a ‘menu’, with some items best avoided, and that a psychiatric diagnosis is not necessarily a disease (Summerfield, 2001). Our patients’ needs and interests are invariably most fully met through such an approach, and with us being alert to a compensation agenda.

Declaration of interest

J.N.S. served in a field mental health team during the 2003 Gulf War.


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Time for a broad phenotype in schizophrenia?

Weiser et al (2005) suggested that it is now time for a shift from narrow to broad phenotypes in schizophrenia research. They proposed deconstructing the schizophrenia syndrome and focusing on cognitive impairment. Although we agree that there is a need for alternative phenotypes in schizophrenia, we do not believe that cognitive impairment is the best candidate. Weiser et al claim that the lack of specificity is one of the main problems of biological studies. In spite of thousands of studies of the cognitive impairment in schizophrenia, no single cognitive function has shown clinical diagnostic value.

In order to understand the pathophysiology of schizophrenia we need to understand the neurobiology of the most specific symptoms. Although auditory hallucinations may appear in other mental disorders or even in the general population, ‘voices’ remain the hallmark of psychoses, and particularly schizophrenia-spectrum disorders. Therefore auditory hallucinations are, in our opinion, a good alternative phenotype.

Although neuroimaging techniques have allowed a much better understanding of the pathophysiology of auditory hallucinations, there are few studies of genetic vulnerability to such hallucinations. Our research has focused on the molecular genetics of auditory hallucinations and supports the possible role of the CCK-AR gene in their development and persistence (Sanjuan et al, 2004). We also found a relationship between allelic variation of the serotonin transporter gene and emotional response to auditory hallucinations (Sanjuan et al, 2005). These are just some examples of how deconstructing the syndrome could help to identify alternative phenotypes.

Advances in neuroscience have been made by focusing on a small area and trying to understand it using all possible approaches. Applying this principle in psychiatry could constitute the ‘core symptom approach’. We would like to remember Rosenthal & Quinn’s (1977) advice in a beautiful study of a unique case of monozygous quadruplets concordant for schizophrenia and hallucinations: ‘If we listen intently to what these voices are telling us, we may achieve a better understanding of the mechanisms that underlie them’.


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Cancer and schizophrenia

The negative finding of the recent paper by Goldacre et al (2005) is an important addition to studies attempting to confirm or disprove the 'epidemiologic puzzle' (Jablensky & Lawrence, 2001), but the evidence remains ambiguous regarding the overall risk of cancer in people with schizophrenia. In three of five comparisons with reference populations conducted between 1992 and 2003 (see Grinspoon et al, 2005), males with schizophrenia had a reduced risk of cancer. No reduction was found among females in four comparisons but decreased risk was reported in one of two comparisons of both males and females. Two recent studies (Dalton et al, 2005; Grinspoon et al, 2005) mostly found reduced risk. When evaluating these results, it is important to recall that people with schizophrenia face many health and service hazards that may increase their risk for cancer (Grinspoon et al, 2005). We therefore suggest that results in this area should not be stopped prematurely, especially since one study (Lichtermann et al, 2001), but not another (Dalton et al, 2004), found a reduced risk of cancer among first-degree relatives of patients with schizophrenia, an indication of a genetic factor (Park et al, 2004).

The study by Goldacre et al (2005) had some limitations, as acknowledged by the authors. We wonder whether a diagnosis of schizophrenia at the time of the first admission may not constitute an additional limitation. Patients admitted with an early diagnosis of schizophrenia but who later received other psychiatric diagnoses might have diluted the risk (Carney et al, 2004), whereas others who did not have a diagnosis of schizophrenia on first admission but did on later contact might have been lost to the enquiry. We also wonder whether the decision to exclude some people from the reference population for selected cancers was sound. Admittedly, dietary factors may be imputed for those conditions selected for elimination as well as for cancer risk. We look forward to a repetition of the analysis after their inclusion.

Authors' reply: We agree that further evidence is needed to gain greater certainty about whether or not cancer risk is altered in people with schizophrenia. By their nature, observational epidemiological studies include more biases and confounding than randomised controlled trials, but the latter are not an option for studying this association.

We included people in the schizophrenia cohort if they had a discharge diagnosis of schizophrenia at any admission and not just at the first admission. We accept that there could be a dilution effect from early misclassification, but it seems unlikely that this would completely reverse any real and substantial inverse association between schizophrenia and cancer.

We excluded people with appendicectomy, haemorrhoids and inguinal hernia from the reference cohort when studying colorectal cancer because we knew, from other work, that they have a significantly increased risk (although fairly small). We therefore felt that, in principle, they were inappropriate for the colorectal cancer analyses. However, this was more a decision on principle than one with much practical effect. Comparing the schizophrenia cohort with the reference cohort, including all people in the reference cohort, the rate ratio for cancer of the rectum fell to 0.55 (95% CI 0.31–0.90), compared with 0.57 (0.33–0.93) reported by us. The rate ratio for cancer of the colon, including all the reference cohort, fell to 0.59 (0.39–0.85) compared with 0.72 (0.50–1.01) reported by us. Thus, a result on the borderline of significance became significant; but we consider that it was right to exclude the three reference groups as in the original analysis. None the less, the case does seem to be building, considering results from other studies as well as ours, that there may be a deficit of colorectal cancer in people with schizophrenia. As suggested by Dr Levav and his colleagues, it is unclear whether this is a result of confounding with dietary factors. Finally, we would like to correct a typographical error in the footnote to our table: 'superficial injury and confusion' should have read 'superficial injury and contusion'.

Schizophrenia, weight gain and atypical antipsychotics

Thakore (2005) highlights the increased prevalence of the metabolic syndrome in schizophrenia. He briefly discusses the relationship between atypical antipsychotic drugs, weight gain and abnormal glucose and lipid metabolism. He rightly concludes that this relationship is poorly understood and that much of the evidence is contradictory or of dubious quality. Unfortunately this narrow focus on the aetiology of the metabolic syndrome risks diverting attention from the urgent need to reduce obesity among people with schizophrenia.

There is good evidence that people with schizophrenia have a high and growing cardiovascular mortality (Osby et al, 2000). Many also have multiple lifestyle-related cardiovascular risk factors such as smoking, lack of exercise and poor diet (Brown et al, 1999), none of which are convincingly susceptible to modification. Schizophrenia may also be associated with intrinsic metabolic disadvantage (Thakore, 2005).
The data on atypical antipsychotic drugs, weight gain and metabolic dysregulation come from a heterogeneous collection of largely uncontrolled studies, but there is no doubt that these drugs induce weight gain and that some are worse than others. ‘First do no harm’. There can be no justification for continuing to prescribe particular atypical antipsychotic drugs which cause serious weight gain to a population who are already at high risk of cardiovascular disease. Equally effective alternatives are readily available and are no more expensive. Obesity increases cardiovascular mortality by 50% (McGee, 2005). We must stop regarding weight gain as an acceptable price to pay for control of psychiatric symptoms.

**Declaration of interest**

S.B. has attended many educational functions supported by pharmaceutical companies but has no other links with the pharmaceutical industry.


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**Akathisia as a risk factor for suicide**

Hawton et al (2005) have produced a comprehensive, systematic review of risk factors for suicide in schizophrenia. The study questions the fundamental procedures that are an integral part of our clinical assessment of this vulnerable group of patients. Suicide is notoriously difficult to predict because of the rarity of the event, the obvious ethical problems of designing informative studies and the uncertainty about risk factors. However, although there is no study of akathisia and suicide that fulfills their strict inclusion criteria, there is more research available than the case reports mentioned (for example, Chow et al, 1997; Hansen, 2001; Hansen et al, 2004). We found no association between akathisia and suicidality in a group of 90 patients with treatment-resistant schizophrenia (Hansen et al, 2004). Akathisia may, however, have a very different impact on patients at different stages of their illness and according to the duration of treatment. Akathisia emerging early in treatment or after increases in dosages may be the more malignant in terms of distress.

Hawton et al also identified agitation (motor restlessness), impulsivity and depression as risk factors but not akathisia. However, akathisia could contribute to or be confused with any of these three identified risk factors.

There is also evidence that akathisia can occur as a consequence of antidepressant treatment, which is common in patients with schizophrenia (Muller-Oerlinghausen & Berghofer, 1999; Hansen & Wilkinson, 2001). Whether there is an additive effect of antipsychotic and antidepressant medication on the intensity and duration of akathisia is not yet known. None the less, in our opinion, it would be premature to exclude akathisia from a role in the complex web of factors that lead to suicide in schizophrenia and perhaps also in other conditions.


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**Brief psychotherapy for Alzheimer’s disease**

I read with interest the paper by Burns et al (2005). This study into an under-researched and important matter is welcome. However, I would like to comment on the conclusions.

The authors quite appropriately comment that the lack of any quantifiable effect of their psychotherapy could result from the small sample size or the relative insensitivity of the outcome measures. They present qualitative data on participants’ experience of the psychotherapy which show the therapy in a positive light. The collection of these data was highly biased, since participants in the ‘standard care’ arm of the trial were not asked about their experience of their treatment. In addition, these patients were not followed-up in the same way as those receiving the therapy. I suspect that if multidisciplinary, holistic care were being provided as it should, these patients would have made equally positive comments about their community psychiatric nurse, social worker, psychiatrist or general practitioner.

The authors of this study have neither devised the adapted therapy (this was described by Brierley et al, 2003), nor have they shown that the therapy works. Hence I disagree with the authors’ main conclusion that ‘this study shows it is possible to adapt a model of psychotherapy for those with Alzheimer’s disease’. They have none the less presented some interesting preliminary data, suggesting a potential benefit of the therapy. I look forward to further research in this area.


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**Author’s reply:** Dr White has raised some important points. The qualitative data on the participants’ experience was only a tiny part of the study and, although agreeing with the points made, I feel they are hardly relevant to the main thrust of the work. Dr White is correct that we did not devise
the adapted therapy, but we did adapt the devised therapy, and superficial scrutiny of the authors on both the papers cited will attest to this. We stand by our conclusion that the model can be adapted and that, at least in part, it works. This is more than anyone else has ever done in this field, and one could only agree with Dr White’s comment about future research in this area.

Call for a European Guidelines Institute

The guideline assessment by Gaebel et al (2005) has been long overdue. Whereas there was originally quite a bit of scepticism about guidelines in psychiatry, there now appears to be a sort of ‘guidelines mania’, as each European national association tries to produce its own guidelines. We have recently completed an assessment of even more (n=61) guidelines, which was not limited to schizophrenia but focused on European psychiatric guidelines. Although our results were similar – the general quality of the guidelines was medium grade, although there were some of outstanding quality – we arrived at somewhat different conclusions.

By amending the AGREE instrument (AGREE Collaboration, 2003) with an additional item, we found that national particularities were very rarely considered by European psychiatric guidelines (18%), and then only very vaguely. This is not surprising, since the evidence available for guidelines is almost always of an international nature. However, in our view not all guidelines share or should share the same evidence. Those developing guidelines may ask different clinical questions and may consider different interventions or outcomes as relevant. This is not a methodological problem of guidelines.

Furthermore, methodologically sound qualitative evidence such as consumer preference studies may be used by guideline developers; this may be restricted to certain regions or nations. It is certainly of concern, however, that the external validity of the available study evidence is rarely evaluated. There are also few efforts to analyse minority ethnic or other subgroups within multicentre studies or to run effectiveness studies in non-Western countries. Of the 5000 randomised controlled trials (RCTs) in the database of the Cochrane Schizophrenia Group, 80% are from Western countries (Moll et al, 2003). The American Psychiatric Association’s 1997 clinical practice guideline for schizophrenia does not provide any information regarding potentially different outcomes for minority ethnic groups included in the RCTs from the USA (U.S. Department of Health & Human Services, 2001).

It is not obvious to us that a European Guidelines Institute would be of great help or would be accepted as a legitimate developer of guidelines. During the development of guidelines many decisions must be made at an early stage by national consensus groups with the contribution of key stakeholders. In our survey most respondents preferred to develop national guidelines with the help of international experts or to share experiences or data with other guideline developers. In our view, evidence concerning efficacy and risk of specific interventions could be reviewed by an international group in order to develop core recommendations that could be adapted by those developing national or local guidelines. For this purpose activities have been started within the World Psychiatric Association (WPA). However, the adaptation of available evidence to local circumstances is an important national or regional duty in guideline development.

Declaration of interest

W.G. is Chairman of the Section on Schizophrenia of the WPA and Chairman of the Section on Guideline Development of the German Society of Psychiatry, Psychotherapy and Nervous Diseases (DGPPN). S.W. is involved in the revision of the schizophrenia practice guideline of the DGPPN.


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One hundred years ago

Scotland. Baldovan Asylum for Imbecile Children

The jubilee report of this asylum, which claims to be the first institution for imbeciles in Scotland, and the second, as regards date of foundation, in the British Empire, contains an interesting narrative, from the pen of the Medical Superintendent, Dr. C. M. Greig, of the rise and progress of the movement for the care and training of mentally-defective children, dating from the middle of the last century. Early in 1853 Sir John and Lady Jane Ogilvie took steps to found “an orphanage or hospital for orphans and imbecile children,” with the idea that when the imbecile children reached a certain stage of cure, their association in play with the orphans would help to raise them towards the normal development, an idea, however, which subsequent experience proved to be erroneous and impracticable. The two institutions, indeed, continued to exist side by side till 1901, when the orphanage was removed to another site, but in the meantime the imbecile institution was considerably enlarged, and from 17 pupils in the school at the close of 1858 the licence was extended to 40 children in 1867, and to 52 in 1870. In 1892 the pupils numbered 72, and in 1902 a licence was granted for 150 children. A new building had previously been erected on a site at Balneydown Farm, and a legacy of £7,000 opportunately becoming available in 1904, the directors increased the accommodation, which at the present time is available for as many as 160 children, with staff.

In the fifty years of its existence 676 children have passed through the institution, and of this number 212 are recorded to have been discharged relieved, 58 not improved, while as many as 256 have died.

This high mortality, equivalent to about 37 per cent., is in marked contrast with the death-rate of the English idiot institutions, in which a mortality of 5 per cent. would be considered large; but it must be borne in mind that the directors of the Baldovan Asylum, unlike those of the English institutions, have resorted to little, if any, selection of cases, throwing open their doors widely to all sorts and conditions of defective children, not excluding epileptics. The rates of board are extremely moderate, £77 per annum for first-class patients, £31 for second class, and £28 for third class, including clothing.

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