Correspondence

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Contents • Devaluation of PTSD • Time for a broad phenotype in schizophrenia? • Cancer and schizophrenia • Schizophrenia, weight gain and atypical antipsychotics • Akathisia as a risk factor for suicide • Brief psychotherapy for Alzheimer's disease • Call for a European Guidelines Institute

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 medicolegal, 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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Tyrer, P. (2005) From the Editor's desk. British Journal of Psychiatry, 186, 552.

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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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Sanjuan, J., Rivero, O., Aguilar, E. J., et al (2005) Serotonin transporter gene polymorphism (5-HTTLPR) and emotional response to auditory hallucinations in schizophrenia. International Journal of Neuropsychopharmacology, **26**, 1–3.

Weiser, M., van Os, J. & Davidson, M. (2005) Time for a shift in focus in schizophrenia: from narrow phenotypes to broad endophenotypes. *British Journal of Psychiatry*, **187**, 203–205.

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