Dissociative disorders have been recognised in both DSM-IV and ICD-10 for some 25 years now. Yet among psychiatrists in particular, they continue to be denied or misdiagnosed, causing serious re-traumatisation for a significant number of patients.

Merskey writes of the absence of 'critical statement[s] by a professional society', but fails to cite the acknowledged leaders in the field, the International Society for the Study of Trauma and Dissociation (ISSTD; www.isst-d.org) and the European Society for Trauma and Dissociation (ESTD; www.estd.org). The ISSTD includes among its members a number of eminent psychiatrists and psychologists and it has produced extensive online guidelines on treatment. The charity First Person Plural, in association with the ESTD and Cheshire & Wirral Partnership NHS Foundation Trust, has produced a training and information DVD.[2]

Furthermore, the National Institute for Health and Clinical Excellence's guidelines accept the existence of dissociative disorders. It has not yet produced a treatment protocol for this condition and recommends that clinicians follow the guidelines of the best informed organisation (www.isst-d.org/education/treatmentguidelines-index.htm).

It should be noted that many psychiatric services and community mental health teams across the country are now implementing treatment protocols for dissociative identity disorder and dissociative disorders that are not only effecting significant changes for patients but are also bringing about cost savings for services.[3]

Declaration of interest

R.A. is President of the European Society for Trauma and Dissociation.

1 Mersky H. Book review: *Attachment, Trauma and Multiplicity: Working with Dissociative Identity Disorder*. *Br J Psychiatry* 2011; **199**: 347–8.

2 First Person Plural. *A Logical Way of Being: The Reality of Dissociative Identity Disorder and Other Complex Dissociative Conditions* (DVD). FPP (http://www.firstpersonplural.org.uk).

3 Lloyd M. How investing in therapeutic services provides a clinical cost saving in the long term. *Health Serv J* 2011; 1 Sept.

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Author's reply: Dissociation begins with hypnotists, was developed by Janet, promoted by Freud and ruined by the absurdities of multiple personality disorder.[1] Consider Janet[2] hypnotising 'Lucie', an alternative personality of this patient producing automatic writing:

Q. 'How are you?'
A. 'I don't know.'
Q. 'There must be someone there who hears me.'
A. 'Yes.'
Q. 'Who is it?'
A. 'Someone other than Lucie.'
Q. 'Ah, Indeed!'
A. 'Another person.'
Q. 'Would you like us to give her a name?'
A. 'No.'
Q. 'Yes it will be more convenient.'
A. 'Alright, Adrienne.'
Q. 'Very well Adrienne. Do you hear me?'
A. 'Yes.'

In 1889 Binet observed that Janet '. . . himself created her by suggestion'.[3]

Hacking[4] showed that the first 19th-century fugue states in young men were in French military conscripts exploiting the novel long-distance continental railways. In older persons fugues are only found with dementia. Experimental attempts by excellent social psychologists over 60 years have completely failed to replicate repression[5] and dissociation. Freud's own accounts of his cases with alleged repression/dissociation were completely unreliable,[6] particularly as shown in the Freud–Fliess correspondence.[7] Further, Pope *et al*[8] have shown that a phenomenon like dissociation (i.e. losing complete trace of some important event and then recovering it through memory) has not been found so far in world literature preceding 1786, and by then Mesmer was actively using hypnotic procedures. If dissociation is a genuine human experience, it is remarkable that it was not known before that time.

There is no case of proven 'dissociation' fulfilling Pope's criteria without organic disorder, although many cases of alleged dissociative memory loss exist, not to mention the generally rejected syndrome of dissociative identity disorder, of which dissociation is the artefactual foundation no matter how much the name or term may be changed.

1 Merskey H. The manufacture of personalities: the production of multiple personality disorder. *Br J Psychiatry* 1992; **160**: 327–40.

2 Janet P. *L'Automatisme psychologique*. Felix Alcan, 1889.

3 Binet A. *Les altérations de la personnalité*. Felix Alcan, 1892. Reprinted as *Alterations of Personality* (ed DN Robinson). Georgetown University Publciations of America, 1977: 146.

4 Hacking I. *Mad Travelers. Reflections on the Reality of Transient Mental Illnesses*. University of Virginia, 1998.

5 Holmes DS. The evidence for repression: an examination of 60 years of research. In *Repression and Dissociation. Implications for Personality Theory, Psychopathology, and Health* (ed JL Singer). University of Chicago Press, 1990: 85–102.

6 Grunbaum A. Made-to-order evidence. *Unauthorized Freud: Doubters Confront a Legend* (ed FC Crews). Viking, 1998: 76–84.

7 Masson JM (trans & ed). *The Complete Letters of Sigmund Freud to Wilhelm Fliess, 1887–1904*. Belknap Press, 1986.

8 Pope Jr HG, Poliakoff MB, Parker MP, Boynes M, Hudson JI. Is dissociative amnesia a culture bound syndrome? Findings from a survey of historical literature. *Psychol Med* 2007; **37**: 225–33.

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Childhood sexual abuse and chronic fatigue syndrome

We have read the important article on the premorbid risk markers for chronic fatigue syndrome in the 1958 British birth cohort[1] with a lot of interest. The authors reported that parental physical abuse, childhood gastrointestinal symptoms and parental reports of many colds were independently associated with self-reported chronic fatigue syndrome (CFS), after adjusting for psychopathology.

Notably, the authors did not comment on the fact that parental physical abuse, but not sexual abuse, was predictive of CFS, even though childhood sexual abuse is a well-documented risk factor for CFS. More precisely, chronic fatigue was significantly predicted by childhood sexual abuse in a population-based study by Taylor & Jason.[2] Also, childhood sexual abuse and emotional abuse were most effective in discriminating CFS cases from control individuals in two population-based studies by Heim *et al* (as well as emotional neglect in one of these studies).[3,4] A possible reason for this inconsistency is the relatively low frequency of sexual abuse in the study by Clark *et al*[1] (6.3 %), compared with its frequency in the others studies (>26%).[2–4]

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Interestingly, there may be a differential clinical effect according to the subtype of childhood trauma. In the study by Taylor & Jason,[2] a history of childhood sexual abuse emerged as a significant predictor of post-traumatic stress disorder. Furthermore, significant correlations between scores on a trauma questionnaire and scores for depression, anxiety and post-traumatic stress were observed by Heim *et al*.[3] These correlations remained unchanged when the analysis was restricted to the subscales sexual abuse and emotional neglect.

Recently, our research group examined the impact of childhood trauma in a well-described tertiary sample of patients with CFS. In accordance with the previously mentioned population-based studies, childhood sexual harassment was the best predictor of psychological symptoms in CFS (unpublished data). Taken together, these data emphasise the importance of childhood sexual abuse as a premorbid risk marker for CFS.

1 Clark C, Goodwin L, Stansfeld SA, Hotopf M, White PD. Premorbid risk markers for chronic fatigue syndrome in the 1958 British birth cohort. *Br J Psychiatry* 2011; **199**: 323–9.

2 Taylor RR, Jason LA. Chronic fatigue, abuse-related traumatization, and psychiatric disorders in a community-based sample. *Soc Sci Med* 2002; **55**: 247–56.

3 Heim C, Wagner D, Maloney E, Papanicolaou DA, Solomon L, Jones JF, et al. Early adverse experience and risk for chronic fatigue syndrome: results from a population-based study. *Arch Gen Psychiatry* 2006; **63**: 1258–66.

4 Heim C, Nater UM, Maloney E, Boneva R, Jones JF, Reeves WC. Childhood trauma and risk for chronic fatigue syndrome: association with neuroendocrine dysfunction. *Arch Gen Psychiatry* 2009; **66**: 72–80.

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Clozapine *v.* chlorpromazine in treatment-naive first-episode schizophrenia

Girgis *et al*[1] present data on the usefulness of clozapine versus chlorpromazine in patients with first-episode schizophrenia. The authors must be complimented for conducting a follow-up study of the same cohort after 9 years and being able to have such a high retention rate. Further, the study provides information with respect to the naturalistic setting, reflecting the true clinical situation, and the authors have taken care of possible confounders with appropriate statistical analysis proper explanation. However, there are certain issues with the study. First, the title of the article is somewhat misleading because the randomisation phase of the study was only for the initial 2 years and after that the patients received treatment at the discretion of the clinicians. The title would have been appropriate if the authors were describing the outcome in terms of efficacy/effectiveness and side-effect profile by using survival analysis focusing on either of the medications. But actually the authors describe the effect of clozapine and chlorpromazine for the initial 1 year and outcome at the 9-year follow-up. Second, we need to understand that there are controversies in relation to the definition of first-episode psychosis and the definition used by the authors may appear to be very broad.[2] Third, the sample size in each treatment group that remained on the same medication (clozapine (n=21) or chlorpromazine (n=8)) at the 9-year follow-up is too small to generalise. Hence, to conclude that there is no difference between clozapine and chlorpromazine with respect to effectiveness would be wrong. Fourth, the authors also conclude that there is no difference in metabolic and other side-effects between the two groups; besides having incomplete baseline data for weight there is no mention of other metabolic variables such as high-density lipoprotein, triglyceride and blood pressure. Fifth, more than half of the study sample (55% of the chlorpromazine group *v.* 73% of the clozapine group) was not on any anti-psychotic medication at 9-year follow-up, but the authors have not elaborated about their clinical status. Last of all, a quarter of participants (24%) were diagnosed with schizophreniform disorder which might have directly affected the outcome as this group of disorders is considered to have better outcome than schizophrenia.

1 Girgis RR, Phillips MR, Li X, Li K, Jiang H, Wu C, et al. Clozapine *v.* chlorpromazine in treatment-naive, first-episode schizophrenia: 9-year outcomes of a randomised clinical trial. *Br J Psychiatry* 2011; **199**: 281–8.

2 Breitborde NK, Srihari VH, Woods SW. Review of the operational definition for first-episode psychosis. *Early Interv Psychiatry* 2009; **3**: 259–65.

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Authors' reply: We appreciate Nebhinani & Grover's interest in our study[1] as well as the opportunity to respond to the six comments. First, our study was analysed using the intent-to-treat principle. Implicit in the intent-to-treat principle is that the outcome is not the effect of treatment *per se*, but rather the effect of initial assignment irrespective of treatment(s) received.[2] Second, we agree that there are controversies as to the definition of first-episode psychosis.[3] As reported by Breitborde *et al*, 'duration of psychosis' possesses the most construct validity, followed by other criteria, such as 'duration of antipsychotic medication use' and 'first treatment contact'.[3] We conservatively identified individuals with first-episode schizophrenia using both duration of psychosis and duration of antipsychotic medication use as two of our criteria. Furthermore, we included a maximum age criterion (i.e. 40 years old at the time when symptoms began) and symptom criteria to further narrow and restrict our study participants to those who are most likely to have first-episode psychosis. Third, our conclusions and main outcomes used the intent-to-treat principle and were based on the entire sample, rather than primarily based on the 29 individuals who remained on their originally assigned medication after 9 years. We described characteristics of this smaller group, without an intent to generalise, owing to the obvious lack of representativeness in this subgroup of patients. Furthermore, it is important to note that the generalisability of a clinical finding is determined by the representativeness of the sample observed, rather than the sample size observed.

Fourth, as described in the article, we did not have any missing baseline data for weight for those participants whose weights were included in our metabolic analyses. In addition, we disagree that we indicated that there were no differences in side-effects between the two groups. Rather, we descriptively reported differences in tardive dyskinesia and agranulocytosis between the two treatment groups. Finally, we did not claim that the metabolic findings in this study are generalisable, but we do agree with Nebhinani & Grover that it would have been valuable to report on additional metabolic indices (e.g. lipids and blood pressure). Unfortunately, these data were not available.