BJPsych | The British Journal of Psychiatry (2022) 220, 98–100.

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Edited by Kiriakos Xenitidis and Colin Campbell

Contents

The shadow costs of dissociative identity disorder

The editorial entitled 'Dissociative identity disorder: out of the shadows at last?'[1] considers that the diagnosis has often been rejected through misleading information, and the prejudices derived therefrom, and through self-protection, a cultural dissociation from the reality of the impact of severe trauma on later clinical presentations. Psychiatrists can then choose to 'dislike' the diagnosis and refuse to use it in a way that would never happen, without severe medico-legal consequences, for schizophrenia or bipolar affective disorder. This occurs despite evidence that: many patients with dissociative identity disorder (DID) are severely ill and functionally impaired, have high rates of severe comorbidities, and are often at risk for non-suicidal self-injury and suicide attempts.[2] However, another reason for mental health services encouraging such dismissive perspectives, and stigmatising/scapegoating those who use the diagnosis, while denying those in need of treatment, is that the treatment is considered prohibitively expensive. Medication is of limited value[3] and specialist psychotherapy for DID not only takes years,[4] but recovery with therapy often has a non-linear course.[5] As psychiatric doctors define their domains by severe and enduring mental illness, with DID omitted, training of psychiatrists remains largely devoid of mention of complex trauma and its sequelae, with DID seen then as the province of others – such as clinical psychology.

DID is usually considered to be at the most severe end of a spectrum of complex trauma disorders, but its treatment requires different skills in the therapist from those required for treating someone with post-traumatic stress disorder (PTSD) not involving structural dissociation.[6] There are many ways to have a diagnosis of PTSD,[7] so the ICD-11 diagnosis of complex PTSD,[8] while welcome, will raise similar questions about the classification of individual patients with complex PTSD and DID, diagnoses which are not synonymous. Also, individuals with DID should not have diagnostic labels of non-dissociative or personality disorders, nor vaguely defined mood, anxiety or psychotic disorders, inappropriately attached to them; nor should clinicians feel the need to eschew the appropriate diagnosis of DID to avoid opprobrium, whether from other clinicians or from management. Any potential gains, service or financial, of not providing comprehensive, continuing, treatment that acknowledges causative factors are short term as there are long-term implications for morbidity and mortality, even across generations (see for example[9]). Pathological dissociation has an impact on the effectiveness, or otherwise, of specialist treatment for adults with histories of early traumatisation so its recognition is vital for treatment planning.[10] Moreover, a specialist online educational programme for patients and clinicians with dissociative disorders has been demonstrated to reduce non-suicidal self-injury in this group.[11] Clinicians should follow the evidence for DID; it has a defined aetiology and pathology, characteristic clinical features for which there are well-established structured interviews – and effective, non-pharmacological, treatments.

The development of the skills for treating DID can improve the ability to treat other disorders in which traumatic experiences have had an aetiological impact and that manifest with some expression of emotion dysregulation but, even with these additional gains, the comprehensive and effective treatment of DID will still have huge service implications. Training of staff to provide clinically relevant diagnostic formulations, and the appropriate treatments, could challenge individual ontological perspectives, and would require significant resources, but would benefit the many individuals who are burdened with the clinical manifestations of these severe post-traumatic states. There is also the distinct possibility that appropriate treatment would not be as economically burdensome as feared when the costs to society of hitherto-unrecognised disorders are compared with the costs to health services from the absence of appropriate treatment.[2]

Declaration of interest

None.

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doi:10.1192/bjp.2021.73

Authors' reply

We welcome the opportunity to respond to Corrigan & Hull's response to our editorial[1] that presented neurobiological evidence for a trauma-related aetiology of dissociative identity disorder (DID). Corrigan & Hull offer an important additional reason to our proposed DID-dismissive perspectives, namely that DID

https://doi.org/10.1192/bjp.2021.74 Published online by Cambridge University Press

treatment is considered prohibitively expensive. Not only do they point out costs because of the length of phase-oriented treatment[2] and its unpredictable non-linear course, they also highlight the costs involved in the training of staff because DID treatment requires specialised skills currently not developed during psychiatrists' training. They conclude that the evidence for DID should be followed by clinicians and that appropriate treatment will cost less overall than leaving disorders involving pathological dissociation untreated.

An important avenue that might reduce treatment length, and therefore treatment costs, is pharmacological intervention. Corrigan & Hull state that medication is of limited value, but to date no double-blind placebo-controlled studies have been performed with the aim to develop evidence-based pharmacotherapy to alleviate pathological dissociative symptoms in DID. However, it has been proposed that kappa-opioid receptor antagonists may be of interest for the selective pharmacological targeting of debilitating dissociative symptoms in post-traumatic stress disorder and trans-diagnostically.[3] Abnormal serotonin neurotransmission in frontal and temporal regions has been found in relation to dissociative amnesia in a positron emission tomography receptor binding study[3] and therefore serotonergic medication might also be of interest to treat pathological dissociative symptoms. In addition, the authors would like to offer the consideration of a glutamate hypothesis for dissociation on the basis of scientific evidence that (a) the glutamatergic agent ketamine induces dissociative symptoms in humans[4] and in animal models,[5] (b) the psychotropic drug lamotrigine can reduce dissociative symptoms induced by ketamine in healthy individuals,[6] (c) glutamatergic hyperactivity could be relevant in the neurobiology of depersonalisation and (d) lamotrigine can be an augmenting treatment to reduce dissociative symptoms in depersonalisation disorder,[6] and (e) anterior cingulate glutamate concentration correlates positively with dissociative symptoms in individuals with borderline personality.[3] Glutamate concentrations in the brain of individuals with pathological dissociation can relatively easily be measured using magnetic resonance spectroscopy, which may provide information on whether glutamate is a neurochemical biomarker of dissociation.

Although more has become known about what happens in the dissociated brain and functional neurocorrelates of pathological dissociation[1,3] are being unravelled, it remains largely unknown how dissociative symptoms are mediated in the brain at a neurotransmitter level. Neurobiological research into the neurochemical biomarkers of pathological dissociation could possibly lead to the development of pharmacological agents that facilitate more rapid symptom alleviation. Although the development of such pharmacological interventions offers a challenge for the scientific community, they are expected to reduce the treatment costs of individuals with DID.

Declaration of interest

None.

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doi:10.1192/bjp.2021.74

Letter to the editor about 'Context and outcome of first-episode psychosis in India and Canada'

The study by Malla et al[1] explored the differences in the 2-year outcome of first-episode psychosis at two sites, one in Montreal, Canada, and the other in Chennai, India. The study concluded a better outcome for negative symptoms in low- and middle-income contexts compared with a high-income context, concurring somewhat with the long-held notion of a better outcome in psychosis, particularly schizophrenia.[2] More family support partly explained this outcome. Evidence against this axiom has also been published[3] in light of methodological limitations of studies supporting this hypothesis, human rights abuses in people with mental illness prevalent in low- and middle-income contexts, and sociocultural transformations occurring in this part of the world. Notwithstanding these debates, we wish to point out a few issues with the present study.[1]

Primarily the way family support was evaluated and used as a statistical metric. The two items (support and family relationship) from the Wisconsin Quality of Life Index – Provider Version were scored on a Likert-type scale; support on a scale of 1–3, and family relationship on a scale of 0–5. For a single-weighted score of family support, both the scores were multiplied, thus ending up with zero total scores occasionally if the latter was scored zero despite a variable score on the former item. Its significance is related to the variation in environmental support and family relationship in the two sites.

Another essential variable of interest missing from the study is the aspect of income (or family income adjusted to the gross domestic product per capita) and controlling for it for site difference other than family support at month 3.

For the examination of predictors of negative symptoms, remission and remission status at month 24, the adherence to medication variable was dropped. We do not find any reason for doing so.

The high-income context site had one-third of participants with affective psychosis versus 10% in the low- and middle-income context site. Patients with affective psychosis are more prone to extrapyramidal symptoms from antipsychotics than those with non-affective psychosis.[4] The higher chances of categorising depressive and extrapyramidal symptoms as negative symptoms without an evaluation of side-effects results in the possibility of inflating the findings.

Finally, concerning individuals who were non-completers of the study, first, mortality in four participants (three because of suicide) in the India site, to us, needs greater emphasis (and may be interpreted as a unique aspect in outcomes research for psychiatric disorders). Second, the disproportionately small number of participants lost to follow-up in the India site is not well explained. The latter could probably be as a result of a combination of family

https://doi.org/10.1192/bjp.2021.74 Published online by Cambridge University Press